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Pedicon 2011 would be a desirous and rare opportunity to interact with the best experts in the field of Pediatrics and to benefit from their knowledge and experience.

"Atithi Devo Bhava" (A guest is like God), "Padharo Mahare Desh" (Welcome to Our Place)

We firmly believe in these mottos, and it shall be our dedicated and sincere effort to ensure that every delegate enjoys the academic feast along with the cultural fiestas and resplendent tourist venues that our splendid city offers.

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**Sun, moon and the stars** denote the vast enriching horizons of knowledge.

**18 Stars** convey the newborn-to-18 years-age-span of pediatric care.

**VIBGYOR** stars depict the wide spectrum of essential care and cure.

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**APPROACH TO THE CONVULSING CHILD**

* Indumathy Santhanam

Abstract: Overt generalized status epilepticus if left untreated will evolve into non-convulsive status epilepticus (NCSE). Failure to recognize and treat NCSE, will lead to electrical status epilepticus. Failure to manage the airway and breathing during seizure management could result in profound hypoxia and shock, which if not identified and corrected early in the management of seizure could result in deleterious effects on the myocardium, brain and metabolic profile. Further, seizures become more difficult to control. Hence failure to manage the co-existing hypoxia, shock, myocardial dysfunction, hypoglycemia and status epilepticus simultaneously could result not only in mortality but also have an impact on the final neurological outcome. This article outlines the current concepts in the management of status epilepticus in the emergency department.

**Keywords:** Status epilepticus, Emergency department, Hypoxia, Shock, Myocardial dysfunction, Drug protocol.

**Definition**

Status epilepticus (SE) is defined as seizures persisting for more than 5 minutes in children above 5 years of age or two or more seizures occurring consecutively without an intervening period of full recovery of consciousness. More recently, as an improvement of this time based definition, SE in children has been divided into an early stage of SE (5-30 minutes), and an established stage of SE (30-60 minutes).

Seizures that do not cease in 5-10 minutes are less likely to terminate without intervention. Hence, a child who is convulsing on arrival in to the emergency department (ED) is more likely to continue to convulse unless actively treated.

**Progression of SE**

Overt generalized status epilepticus if left untreated will evolve in to non-convulsive status epilepticus (NCSE). Failure to recognize and treat NCSE, will lead to electrical status epilepticus. Periodic epileptiform discharges on EEG monitoring is diagnostic.

The deleterious cerebral, metabolic, and physiological changes that occur as the duration of seizures increases is shown in Table.1.

The effect of drugs in the management of SE appears to be mediated by the neuropeptide activity in the brain. Most seizures terminate spontaneously within 2 minutes, due to γ-aminobutyric acid (GABA) mediated inhibition that occurs in response to seizures. However, ongoing seizure activity results in loss of the protective effect mediated by GABA as the GABA receptors are either destroyed or recycled in to the cellular membrane. At the same time, continued seizure activity results in mobilization of excitatory N-methyl-d-aspartate receptors.
### Table 1. Medical complications of status epilepticus

<table>
<thead>
<tr>
<th>Complication</th>
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<tr>
<td><strong>Interictal coma</strong></td>
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<tr>
<td><strong>Cumulative anoxia</strong></td>
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<tr>
<td>Cerebral and systemic</td>
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<tr>
<td><strong>Cardiovascular complications</strong></td>
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<tr>
<td>Tachycardia, bradycardia</td>
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<tr>
<td>Cardiac arrest</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>Cardiac failure, hypotension</td>
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<tr>
<td>Cardiogenic shock</td>
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<tr>
<td><strong>Respiratory system failure</strong></td>
</tr>
<tr>
<td>Apnea</td>
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<tr>
<td>Cheyne-Stokes breathing</td>
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<tr>
<td>Tachypnea</td>
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<tr>
<td>Neurogenic pulmonary edema</td>
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<tr>
<td>Aspiration, pneumonia</td>
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<tr>
<td>Respiratory acidosis</td>
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<tr>
<td>Cyanosis</td>
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<tr>
<td><strong>Renal failure</strong></td>
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<tr>
<td>Oliguria, uremia</td>
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<tr>
<td>Acute tubular necrosis</td>
</tr>
<tr>
<td>Rhabdomyolysis</td>
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<tr>
<td>Lower nephron necrosis</td>
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<tr>
<td><strong>Autonomic system disturbance</strong></td>
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<tr>
<td>Hyperpyrexia</td>
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<tr>
<td>Excessive sweating, vomiting</td>
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<tr>
<td>Hypersecretion (salivary, tracheobronchial)</td>
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<tr>
<td>Airway obstruction</td>
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<tr>
<td><strong>Metabolic and biochemical abnormalities</strong></td>
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<tr>
<td>Acidosis (metabolic, lactate)</td>
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<tr>
<td>Hypernatremia, hyponatremia</td>
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<tr>
<td>Hypoglycemia</td>
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<tr>
<td>Hepatic failure</td>
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<tr>
<td>Dehydration</td>
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<tr>
<td>Acute pancreatitis</td>
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<td><strong>Infection</strong></td>
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<tr>
<td>Pulmonary</td>
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<tr>
<td>Bladder</td>
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<td>Skin</td>
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<tr>
<td><strong>Other</strong></td>
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<tr>
<td>Altered autoregulation and CBE</td>
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<tr>
<td>Increased cerebral metabolic rate for oxygen (CMRO₂)</td>
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<tr>
<td>Disseminated intravascular coagulation</td>
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<tr>
<td>Multiple organ system dysfunction,</td>
</tr>
<tr>
<td>Fractures, thrombophlebitis</td>
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</table>

The reduced activity of GABA (seizure control neuropeptides) and increased activity of N-methyl-d-aspartate receptors (seizure provoking neuropeptides) results in a decreased inhibitory control and increased excitation that may lead to continuing status epilepticus. Benzodiazepines, which work via γ-aminobutyric acid mechanisms, are less effective as seizure durations increase.

Studies in children have revealed a high morbidity and mortality associated with refractory status epilepticus. In addition, serum levels of neuron-specific enolase, a marker of neuronal injury, were elevated in children with continuous electrographic discharges, even without clinical seizures. These data underscore the importance of early and aggressive management of SE in the ED.
Airway

Open the airway using the head tilt and chin lift maneuver. Cervical spine precautions are taken if trauma is suspected by using the jaw thrust maneuver. With the help of a large bore rigid suction catheter, gentle suction is applied to clear oropharyngeal secretions. The stomach is rapidly decompressed with a nasogastric tube to prevent vomiting and pulmonary aspiration. An appropriate sized oropharyngeal airway is introduced if feasible, taking care to avoid forcible opening of clenched jaws during a convulsive episode. One nurse should be delegated exclusively for assisting the airway physician.

Rationale: Airway compromise in status epilepticus occurs as a result of various factors.

- Unresponsiveness due to seizure activity results in the falling back of tongue
- Loss of airway protective reflexes results in failure to handle the increased tracheo-bronchial secretions
- Glottic spasm obstructs the airway due to seizure activity of the voluntary muscles of the larynx

Simultaneous correction of hypoxia during the management of status epilepticus is mandatory for intact neurological survival.

Breathing

Children who are convulsing despite appropriate pre-hospital care on arrival into referral emergency departments invariably require respiratory support. While the airway is being cleared by the airway nurse, an appropriate sized bag valve mask device (BVM) is used to provide ventilation. Effective respiratory support using the BVM device by a trained health care provider can reduce the deleterious effects of hypoxia in SE. In upto 50% of children presenting with SE, effective bag valve mask technique could reduce the need to intubate and ventilate. Oxygen saturation has to be monitored using pulse oximeter.

Rationale: Ineffective respiration occurs as a result of convulsive activity of the muscles of respiration.

- Seizure activity of inter-costals: Apnea
- Contraction of involuntary muscles of diaphragm: Jerky respirations
- Drugs used in the management of status epilepticus can also lead to respiratory failure

Indications for intubation in SE

- Failure to maintain optimal saturation despite effective bag valve mask technique
- Status epilepticus refractory to phenytoin
- Cardiogenic shock
- Hypotensive shock with SE
- Head trauma- Controlled ventilation
- Raised ICP

Failure to provide effective ventilatory support during the management of status epilepticus is perhaps one of the most frequent causes of morbidity and mortality from this life threatening emergency in our country. On the contrary, effective respiratory support along with appropriate anti-convulsants often establishes the airway and breathing along with seizure control in the ED.

Glucose

Hypoglycemia can cause severe disruption of autoregulation of cerebral blood flow leading to adverse neurological outcomes. Hence, dextrostix should be used to measure sugar levels early in the management of SE. Documented
hypoglycemia is corrected with an intra-venous bolus of 2 ml/kg of 25% dextrose solution. In settings without access to immediate dextrostix, to avoid the dangerous effects of un-recognized hypoglycemia 25% dextrose may be administered empirically in the same dose.

**Circulation**

Secure intra venous access on arrival and provide non glucose containing isotonic fluids. Atleast 2 nurses may be needed to secure intra-venous access. If IV access is difficult, intra-osseous access must be secured. If shock is identified the first bolus of isotonic fluid 20ml/kg is administered. If the child’s circulatory status is euvoeomic, restrict fluids to 2-3ml/kg/hour. Shock due to idiopathic SE will usually get corrected with 20-30 ml/kg of fluids. SE complicated by diarrhea, sepsis would require larger volumes of fluids to attain therapeutic goals. During the correction of shock in SE, features of myocardial dysfuntion and pulmonary edema may be unmasked. The latter, warrants interruption of fluids, initiation of an inotrope and early intubation. Further fluids are administered only if shock persists and the etiology is sepsis or hypovolemia.

**Rationale:** Shock could occur due to a wide variety of causes in convulsing children.

- Neurogenic: Distributive shock
- Hypoxia: Distributive shock with or without myocardial dysfuntion
- Co-existing sepsis
- Co-existing hypovolemia

During SE, cerebrovascular resistance falls due to hypoxia, resulting in severe derangement of cerebral auto-regulation. Cerebral perfusion becomes directly dependent on systemic blood pressure. Within the first ½ hour of SE, blood pressure rises. Later blood pressure either becomes normal or hypotensive. Shock which occurs in convulsing children secondary to both seizure activity and other causes complicating SE (as mentioned above) severely deranges cerebral physiology.

Aggressive management of shock based on etiology is mandatory for intact neurological survival.

Resuscitation of SE requires team coordination and effort in a time sensitive manner. Whilst one emergency physician manages the airway and breathing on arrival, the second physician performs the rapid cardiorespiratory assessment and documents the clinical findings. One nurse assists the airway manager while 2 nurses are needed to secure IV access, start fluids, dextrose and administer the first anti-convulsant. As effective ventilation is being provided the airway manager should also attempt to obtain a targeted history, confirm eye signs, ensure that a thermometer and pulse oximeter are placed to monitor the child.

**Drug therapy**

The goal of drug therapy is the rapid control of convulsions. The longer the duration of convolution, the greater the risk of complications. Hence, it seems mandatory to follow a clear drug protocol which is understood by all personnel. Continuous monitoring and skilled care is essential during administration of drugs due to the grave risk of hypoventilation and hypotension during resuscitation.

**Benzodiazipines**, are the most potent and effective first line drugs in the management of SE. Presence of apnea, is not a contraindication to the administration of benzodiazipines. The rapid onset of action of benzodiazipines is often useful in resolving seizure induced apnea.
Lorazepam controls seizures within 3 minutes in 89% of patients. Despite being comparable in potency and efficacy to diazepam, lorazepam has a longer duration of anti-seizure effect (12-24 hours). Less risk of recurrence with use of lorazepam has made this drug the preferred first line benzodiazepine in the treatment of SE. Besides, there is a suggestion that lorazepam has less respiratory depression than diazepam. However, lorazepam needs to be refrigerated and should be diluted and administered as a bolus over one minute.

Diazepam controls seizure within 1 minute in 80% of patients. Due to the risk of respiratory depression, ability to support ventilation is a prerequisite during administration of any of the benzodiazepines. The duration of anticonvulsant effect of diazepam is only for 30 minutes resulting in a high risk of recurrence of SE if this drug is used alone. It is therefore recommended that, even if diazepam has stopped a convulsive SE, phenytoin should be given to prevent recurrence of seizures. Even if the fit appears to have been controlled during administration of benzodiazepines the full dose should be given in order to avoid converting a convulsive SE to a non-convulsive SE. Midazolam has no advantage over diazepam or lorazepam. It is extremely efficacious as an intramuscular anti-convulsant when other routes are not available. It has a rapid onset of action and controls seizures in 90% of patients. Its shorter half life and resultant increased risk of recurrence makes it a less preferred drug to lorazepam in the initial management of CSE in the ED. Midazolam also has an added risk of hypotension, a dreaded complication in SE resuscitation.

A second dose of lorazepam or diazepam maybe repeated in 5 minutes if seizures are not controlled with the first dose.

Phenytoin is the second line drug in patients not responding to the initial two doses of benzodiazepines. It is poorly soluble in water and precipitates in dextrose containing solutions. It is infused in normal saline in the dose of 15-20 mg/ kg at the rate of 1 mg/kg/min with maximum rate of 50 mg/min. Anti-seizure threshold in the brain is reached within 10-30 minutes after infusion. If convulsions are not controlled, 5 mg/kg increments can be administered up to a maximum loading dose of 30 mg/kg. This drug, however, does not cause respiratory depression, thus enabling resuscitation of SE without need for intubation. However, phenytoin is cardiotoxic.

Phenytoin administration often unmasks underlying cardiogenic or non-cardiogenic pulmonary edema. Pink frothy secretions, gallop, muffling of heart sound, bradycardia, hypotension, widening of pulse pressure with low mean arterial pressures, increasing liver span or drop in oxygen saturations herald the onset of this lethal complication. SE with co-existing septic shock (CNS infection) is the commonest cause of this complication being unmasked during phenytoin administration. Myocardial dysfunction which occurs due to prolonged SE or underlying congenital heart disease are other less common causes of deterioration. Indeed, it may be safer to avoid phenytoin in SE complicated by septic shock or congenital heart disease. To avoid this lethal side effect, the rapid cardiopulmonary assessment must be repeated during phenytoin administration. Fosphenytoin, a water soluble phosphoester of phenytoin has less cardiotoxic effects than phenytoin and may be safer. This drug can be administered through the intramuscular route if IV access is not available.

The APLS recommends that, the loading dose of phenytoin should be avoided for children taking chronic phenytoin therapy. Unfortunately, avoidance of Phenytoin in patients with seizure disorder with or without developmental delay, increases the need for early intubation.
In addition, non-compliance, failure to monitor drug levels and failure to titrate drug doses in growing children may prevent maintenance of anti-seizure threshold levels of phenytoin. Hence, in our Pediatric Emergency Department, loading doses of phenytoin are administered even in chronic phenytoin therapy.

In children less than 18 months of age, pyridoxine may be administered if seizures are refractory to phenytoin infusion. A stat dose of 50-100 mg may be administered. Apnea is a complication during administration.

**Alternative routes of drug administration:**
When intravenous access is not available, midazolam (0.2 mg/kg) can be given intramuscularly, rectally or into the buccal space. All anti-convulsant drugs can be administered via the intraosseous route. The doses of these drugs are the same as for the IV route.

**Pre-hospital therapy**
A convulsing child should be placed in the recovery position to enable drainage of secretions. Suctioning to clear the airway and provision of oxygen improves outcome. Midazolam is the drug of choice in the prehospital setting. 0.05-0.2 mg/kg is administered intramuscularly, sublingually or rectally. Fever a common cause of seizures in children should be controlled rapidly by tepid sponging and placing a rectal suppository of paracetamol.

**Non-convulsive Status Epilepticus (NCSE)**
Evidence of conjugate deviation of eyes, lid twitch, nystagmus or unilateral clonus in an unresponsive child helps to recognize this condition. Anticipate NCSE, in children who developed sudden unresponsiveness or who had seizures and had not regained base line consciousness. More commonly, convulsive status epilepticus evolves into NCSE during resuscitation. Active convulsions disappear, but the child remains apneic or tachypneic, shocky and unresponsive. The eye signs indicate persistence of ongoing seizure activity. Often mistaken for a post ictal state, a high index of suspicion is needed to identify NCSE. Continuation of the aggressive management of the airway, breathing and circulation is recommended with the same drug protocol as for CSE until all therapeutic goals are achieved.

**Refractory status epilepticus (RSE)**
Definitions of RSE vary. Since, majority of children have been convulsing for greater than one hour and have not received pre-hospital respiratory support during the management of seizures, we define this entity as, SE not controlled with the initial 2 adequate doses of benzodiazepines and phenytoin.

Midazolam, a water soluble benzodiazepine rapidly penetrates the blood-brain barrier and exerts a short duration of action. It suppresses neuronal excitability by modulating the γ-aminobutyric acid receptors. Midazolam is hydroxylated in the liver and the metabolite is excreted by the kidneys. Hence midazolam should be used with caution in children with underlying hepatic or renal dysfunction, presenting with refractory convulsions to the ED.

Midazolam, a good choice for the initial treatment of RSE, is given as 0.1-0.5 mg/kg IV bolus. To avoid hypotension, midazolam is given slowly over 1 to 3 minutes. A continuous infusion of 1 μg/kg/min of midazolam with increments of 1 μg/kg/min every 15 minutes is recommended until the seizures are controlled. The maximum rate of infusion is 30 μg/kg/min. Though higher boluses and more rapid escalation may be associated with more prompt seizure control, hypotension following use of midazolam remains a serious concern.
Since, midazolam infusion requires the use of infusion pumps for precise titration, phenobarbitone (PB) appears to be the drug of choice if seizures are refractory to Phenytoin. PB infusion, can be manually administered, unlike midazolam infusion. An additional 10 % of SE is controlled with the use of PB without resorting to midazolam infusions.

**Phenobarbital** is useful in RSE and neonatal SE. It depresses neuronal excitability by enhancing the γ-aminobutyric acid receptor response. Depression of mental status and respiratory failure are common. Intubation is mandatory if PB is administered after benzodizepines. Besides, the drug must be administered slowly to avoid hypotension. The recommended dose is 20 mg/kg upto a maximum of 30 mg/kg and the rate of infusion is 1-2 mg/kg/min. The anti-seizure effect occurs within 10-20mins. Unlike phenytoin, PB can be safely used in patients already maintained on this drug.

**Valproic acid**, a broad-spectrum anticonvulsant which acts by modulating sodium and calcium channels, as well as inhibitory γ-aminobutyric acid transmission, is being advocated for RSE. Status epilepticus not responding to midazolam, may be treated with sodium valproate, 25 mg/kg bolus (max 40 mg/kg), followed by an infusion of 5 mg/kg/hr. The advantages of this drug are that it has less sedation than other drugs, good cardiovascular profile and enables quick recovery. The lower risk of respiratory failure compared to other anti-convulsants, makes it a good drug in situations where intubation needs to be withheld. However, it should be used with caution in children with risk of metabolic disease and hepatotoxicity.

**Levetiracetam**, is an anticonvulsant, which may act through calcium channels, glutamate receptors, and γ-aminobutyric acid modulation.

Intravenous levetiracetam, is not metabolized by the liver, has low protein binding, is renally excreted, and has minimal drug-drug interactions. When, RSE is complicated by coagulopathy, liver failure or hypotension, traditional anticonvulsants are contra-indicated. In these situations levetiracetam may be a good alternative anti-seizure drug. It is administered as 20-30mg/kg IV at 5mg/kg/min (maximum, 3g).

A loading dose of thiopentone sodium 3-5 mg/kg followed by an infusion has also been given for refractory status epilepticus. But severe hypotension requiring vasopressor therapy and prolonged post-infusion weakness delaying weaning make it a less commonly used drug in RSE.

**Emergency investigations**

Blood is collected for glucose, renal and liver function, calcium, magnesium, CBC, cultures, PT, PTT and blood gases. If available, blood and urine should be collected for anti-convulsant levels, toxicological and metabolic screening. If SE is focal or coexistent with an acute illness cranial CT scan is necessary. LP is performed when CNS infection is suspected. X-rays are also taken when needed.

**Treatment of specific causes of SE**

CNS infections, head trauma, cerebral edema, space occupying lesion, hemorrhage, poisons, hypoglycemia, hypoxia, hypertensive encephalopathy, electrolyte imbalances and drug toxicity can all produce seizures that are difficult to control. Assessment of patient for possible etiology, is performed after controlling the seizures.

Medical complications in patients being treated for status epilepticus are mentioned in Table.1.
Points to Remember

- Always differentiate convulsion from hypoxic posturing.
- Failure to differentiate post ictal state and persistence of altered level of consciousness secondary to ongoing NCSE or hemodynamic compromise can be disastrous.
- Initiation of bag valve mask and correction of shock during management of status epilepticus management is crucial to successful outcomes.
- Administration of IM diazepam will worsen sedation and respiratory failure but will not resolve seizure activity.
- Phenytoin administration could be dangerous in children who develop signs of pulmonary edema and myocardial dysfunction during management of status epilepticus.
- Rapid intra-venous administration of midazolam could result in hypotension.

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Sri Ramachandra Intensive Pediatric Postgraduate Exam-review - RIPE 2010

Organized by the Department of pediatrics, SRMC, Porur for everyone who intends to take pediatric post graduate exam DCH/MD/DNB

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INTERPRETATION OF CSF ANALYSIS

* Viswanathan V

Abstract: Cerebrospinal fluid analysis is commonly performed in most centers routinely but sometimes interpretation of the results causes a lot of difficulties. One of the key features in clinical practice however is that (clinical) judgement based on history and clinical features is the most important step and the lab results are used as materials to add weight to the clinical diagnosis. The results of a test per se should never be taken in isolation. Some of the common findings in CSF are discussed in the article.

Keywords: Cerebrospinal fluid, Meningitis, Demyelinating disorders, Benign intracranial hypertension, Protein, Cell count, Lactate.

Cerebrospinal fluid (CSF) analysis is a set of laboratory tests that examine a sample of the fluid surrounding the brain and spinal cord. This fluid is an ultrafiltrate of plasma. It is clear and colourless. It contains glucose, electrolytes, amino acids, and other small molecules found in plasma, but has very little protein and few cells. CSF protects the central nervous system from injury, cushions it from the surrounding bone structure, provides it with nutrients and removes waste products by returning them to the blood.

Properly interpreted tests can make cerebrospinal fluid (CSF) a key tool in the diagnosis of a variety of diseases.

Proper evaluation of CSF depends on knowing which tests to order, normal ranges for the patient’s age, and the test’s limitations.

Table 1. Indications for CSF analysis

<table>
<thead>
<tr>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Suspicion of meningitis</td>
</tr>
<tr>
<td>• Suspicion of subarachnoid hemorrhage</td>
</tr>
<tr>
<td>• Suspicion of central nervous system diseases</td>
</tr>
<tr>
<td>such as multiple sclerosis, acute disseminated</td>
</tr>
<tr>
<td>encephalomyelitis, Guillain-Barré syndrome.</td>
</tr>
<tr>
<td>• Therapeutic relief of benign intracranial</td>
</tr>
<tr>
<td>hypertension.</td>
</tr>
</tbody>
</table>

Indications for CSF analysis are given in Table 1 and contra-indications to CSF analysis are given in Table 2, while indications to imaging of brain by computerised tomography (CT) before lumbar puncture (LP) are given in Table 3. The characteristics of a normal CSF given in Table 4.

Risks

The most common side effect after the removal of CSF is headache. This occurs in 10-30% of adult patients and in up to 40% of children. It is caused by a decreased CSF pressure related to a small leak of CSF through the puncture site. These headaches usually are a dull pain, although some people report a throbbing sensation. A stiff neck and nausea may accompany the headache. Lumbar puncture headaches typically begin within two days after the procedure and persist for a few days to several weeks or months.

* Consultant Pediatric Neurologist, Kanchi Kamakoti CHILDs Trust Hospital, Chennai.
Table 2. Contra-indications

<table>
<thead>
<tr>
<th>Absolute contra-indications to lumbar puncture</th>
<th>Relative contra-indications to lumbar puncture</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Unequal pressures between the supratentorial and infratentorial compartments, usually inferred by characteristic findings on the brain CT scan:</td>
<td>• Increased intracranial pressure (ICP)</td>
</tr>
<tr>
<td>       • Midline shift</td>
<td>• Coagulopathy</td>
</tr>
<tr>
<td>       • Loss of suprachiasmatic and basilar cisterns</td>
<td>• Brain abscess</td>
</tr>
<tr>
<td>       • Posterior fossa mass</td>
<td></td>
</tr>
<tr>
<td>       • Loss of the superior cerebellar cistern</td>
<td></td>
</tr>
<tr>
<td>       • Loss of the quadrigeminal plate cistern</td>
<td></td>
</tr>
<tr>
<td>• Infected skin over the needle entry site</td>
<td></td>
</tr>
</tbody>
</table>

Table 3. Indications for CT scan brain prior to lumbar puncture

- Patients who are immunocompromised
- Patients with known CNS lesions
- Patients who have had a seizure within 1 week of presentation
- Patients with abnormal level of consciousness
- Patients with focal findings on neurological examination
- Patients with papilledema seen on physical examination with clinical suspicion of elevated ICP

Table 4. Normal CSF characteristics

- Gross appearance: Normal CSF is clear and colourless.
- CSF opening pressure: 50–175 mm H₂O.
- Specific gravity: 1.006–1.009.
- Glucose: 40–80 mg/dL.
- Total protein: 15–45 mg/dL.
- Lactate: less than 35 mg/dL.
- Leukocytes (white blood cells): 0–5/mm³ (adults and children); up to 30/mm³ (newborns).
- Differential: 60–80% lymphocytes; up to 30% monocytes and macrophages; other cells 2% or less. Monocytes and macrophages are somewhat higher in neonates.
- Gram stain: negative and culture sterile.
- Red blood cell count: Normally, there are no red blood cells in the CSF unless the needle passes through a blood vessel on route to the CSF.
Opening pressure

To measure CSF opening pressure, the patient must be in the lateral decubitus position with the legs and neck in a neutral position. The patient should be advised not to strain, because straining can increase the opening pressure, and cautioned not to hyperventilate, because hyperventilating will lower the opening pressure.

Normal opening pressure ranges from 10 to 100 mm H2O in young children, 60 to 200 mm H2O after eight years of age, and up to 250 mm H2O in obese patients. Intracranial hypotension is defined as an opening pressure of less than 60 mm H2O. This finding is rare except in patients with a history of trauma causing a CSF leak, or whenever the patient has had a previous lumbar puncture.

Opening pressures above 250 mm H2O are diagnostic of intracranial hypertension. Elevated intracranial pressure is present in many pathologic states, including meningitis, intracranial hemorrhage and tumors. Benign intracranial hypertension is a condition seen in children usually as a result of hyper vitaminosis A or due to drugs such as nalidixic acid, nitrofurantoin, cortico steroid withdrawal, hypothyroidism, hyperthyroidism, hypoparathyroidism etc. When an elevated opening pressure is discovered, CSF should be removed slowly and the pressure monitored during the procedure. No additional CSF should be removed once the pressure reaches 50 percent of the opening pressure.

Supernatant fluid colour

Normal CSF is crystal clear. However, as few as 200 white blood cells (WBCs) per mm³ or 400 red blood cells (RBCs) per mm³ will cause CSF to appear turbid. Xanthochromia is a yellow, orange, or pink discoloration of the CSF, most often caused by the lysis of RBCs resulting in hemoglobin breakdown to oxyhemoglobin, methemoglobin and bilirubin (Table 5). Discoloration begins after RBCs have been in spinal fluid for about two hours, and remains for two to four weeks. Xanthochromia is present in more than 90 percent of patients within 12 hours of subarachnoid hemorrhage onset and in patients with serum bilirubin levels between 10 to 15 mg per dL. CSF protein levels of at least 150 mg per dL, as seen in many infectious and inflammatory conditions, or as a result of a traumatic tap that contains more than 100,000 RBCs per mm³ - also will result in xanthochromia. Newborn CSF is often xanthochromic because of the frequent elevation of bilirubin and protein levels in this age group.

Table 5. Cerebrospinal fluid supernatant colors and associated conditions or causes

<table>
<thead>
<tr>
<th>Color of supernatant CSF</th>
<th>Conditions or causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yellow</td>
<td>Blood breakdown products</td>
</tr>
<tr>
<td></td>
<td>Hyperbilirubinemia</td>
</tr>
<tr>
<td></td>
<td>CSF protein &gt;150 mg per dL (1.5 g per L) &gt;100,000 red</td>
</tr>
<tr>
<td></td>
<td>blood cells per mm³</td>
</tr>
<tr>
<td>Orange</td>
<td>Blood breakdown products</td>
</tr>
<tr>
<td></td>
<td>High carotenoid ingestion</td>
</tr>
<tr>
<td>Pink</td>
<td>Blood breakdown products</td>
</tr>
<tr>
<td>Green</td>
<td>Hyperbilirubinemia</td>
</tr>
<tr>
<td>Brown</td>
<td>Meningeal melanomatosis</td>
</tr>
</tbody>
</table>

CSF = Cerebrospinal fluid
in newborns. Normal adult CSF will contain approximately 70% lymphocytes and 30% monocytes. Occasionally a single eosinophil or polymorph will be seen in normal CSF. In neonates it is not unusual to find several polymorphs in the CSF.  

In bacterial meningitis CSF usually shows predominance of polymorphonuclear cells. However more than 10% of the bacterial meningitis cases will show a lymphocytic predominance especially early in the course of the disease. Lymphocytosis is usually seen in viral, fungal and tuberculous infections of the CNS, although a predominance of polymorphonuclear cells may be present early in the course of these infections. Eosinophils are usually predominant in the CSF in parasitic infestations like neurocysticercosis.

**Glucose**

CSF glucose level normally approximates 60% of the peripheral blood glucose level at the time of the tap. A simultaneous measurement of blood glucose (especially if the CSF glucose level is likely to be low) is recommended. Low CSF glucose level usually is associated with bacterial infection (probably due to enzymatic inhibition rather than actual bacterial consumption of the glucose) (Table 6). It is also seen in tumor infiltration and may be one of the hallmarks of meningeal carcinomatosis, even with negative cytologic findings. High CSF glucose level has no specific diagnostic significance and is most often spillover from elevated blood glucose level.

**Protein**

CSF protein is elevated in most conditions where there is a breakdown / leak in the blood brain barrier.

Assessment of CSF protein level, while nonspecific, can be a clue to otherwise unsuspected neurologic disease. The high protein levels in demyelinating polyneuropathies or postinfectious states, can be informative. High CSF protein levels may be seen in infections/inflammatory conditions like meningitis, irritation to the meninges as in carcinomatous infiltration or leukaemic infiltration, degenerative conditions involving the white matter in the brain like leukodystrophies. A traumatic tap can introduce protein into the CSF. An approximation of 1 mg of protein per 750 RBCs may be considered, but a repeat tap is preferable.

**Lactate**

Estimation of CSF lactate is helpful in children as it is elevated early in infections and also in certain metabolic / degenerative disorders involving the CNS. Any estimation of CSF lactate should preferably be combined with a serum lactate estimation for comparison. Both CSF and serum lactates should ideally be performed with fasting for about 4 hours at least. In conditions like Leigh’s disease the CSF lactate is abnormally elevated in comparison to the serum lactate and the CSF lactate to pyruvate ratio is abnormal.

**CSF Gram stain, culture and PCR**

It is very important to get a good Gram stain in the CSF soon after the sample is removed as this will help early and appropriate diagnosis and treatment if positive. CSF cultures typically take a few days to do and for the results to arrive and so most of us would treat any suspected infection while we await the results. CSF polymerase chain reaction for both TB and viral studies like HSV again take a few days to come and should be treated if there is clinical suspicion while awaiting results.

Peripheral blood in the CSF after a “traumatic tap” will result in an artificial increase in WBCs by one WBC for every 500 to 1,000 RBCs in the CSF. This correction factor is accurate as long as the peripheral WBC count is not extremely high or low.
A traumatic tap occurs in approximately 20 percent of lumbar punctures. Common practice is to measure cell counts in three consecutive tubes of CSF. If the number of RBCs is relatively constant, then it is assumed that the blood is caused by an intracranial hemorrhage. A falling count is attributed to a traumatic tap. The three-tube method, however, is not always reliable.

Xanthochromia is a more reliable predictor of hemorrhage. If a traumatic tap occurs within 12 hours of a suspected subarachnoid hemorrhage, it is reasonable to repeat the lumbar puncture one interspace up to try and obtain clear CSF.

### Table 6. Cerebrospinal fluid findings in various types of meningitis

<table>
<thead>
<tr>
<th>Test</th>
<th>Bacterial</th>
<th>Viral</th>
<th>Fungal</th>
<th>Tubercular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening pressure</td>
<td>Elevated</td>
<td>Usually normal</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>White blood cell count</td>
<td>&gt;1,000 per mm³</td>
<td>&lt;100 per mm³</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>Cell differential</td>
<td>Predominance of PMNs*</td>
<td>Predominance of lymphocytes†</td>
<td>Predominance of lymphocytes</td>
<td>Predominance of lymphocytes</td>
</tr>
<tr>
<td>Protein</td>
<td>Mild to marked elevation</td>
<td>Normal to elevated</td>
<td>Elevated</td>
<td>Elevated</td>
</tr>
<tr>
<td>CSF-to-serum glucose ratio</td>
<td>Normal to marked decrease</td>
<td>Usually normal</td>
<td>Low</td>
<td>Low</td>
</tr>
</tbody>
</table>

CSF = cerebrospinal fluid; PMNs = polymorphonuclear leucocytes.

*—Lymphocytosis present 10 percent of the time.
†—PMNs may predominate early in the course.

Usefulness / Limitations of rapid antibody tests and PCR

Rapid antibody tests are useful in conditions like tuberculous meningitis where in the results of the culture takes a few weeks and this period can be detrimental for the child if not treated. But the problem with rapid tests like the Erizyme linked immunesorbent asyay (ELISA) is that of limited sensitivity and a negative test does not exclude the diagnosis of tuberculous meningitis.
Ploymerase chain reaction for the diagnosis of tuberculous meningitis has been shown to be useful and accurate method for early diagnosis, the sensitivity of PCR for diagnosis of tuberculous meningitis is around 63-100% and the specificity is 88-100%.

In a healthy person, there should be little or no antibodies in the CSF. Where there is a viral meningitis or encephalitis, antibodies may be produced against the virus by lymphocytes in the CSF. The finding of antibodies in the CSF is said to be significant when the titre of antibody in the serum and that in the CSF is less than 100. But this does depend on an intact blood-brain barrier. The problem is that in many cases of meningitis and encephalitis, the blood-brain barrier is damaged, so that antibodies in the serum can
actually leak across into the CSF. This also happens where the lumbar puncture was traumatic in which case the spinal fluid would be bloodstained.\textsuperscript{13}

**CSF findings in children with other disorders**

**a) Complex febrile seizures**

Children with complex febrile seizures had a median CSF white blood cell count of 1 cell/mm\(^3\) (range 0-19 cells/mm\(^3\)) and a median CSF polymorphonuclear (PMN) cell count of 0 cells/mm\(^3\) (range 0-8 cells/mm\(^3\)). The CSF white blood cell (WBC) count was elevated above the upper limit of normal of 5 cells/mm\(^3\) in 9.8\% and the absolute number of polymorphonuclear cells was more than 0 cells/mm\(^3\) in 26.2\% of the complex febrile seizure subjects. They conclude that complex febrile, idiopathic nonfebrile convulsions or status epilepticus may affect CSF findings in children: CSF with > 20 WBC/mm\(^3\) or > 10 PMN/mm\(^3\) should not be attributed to seizures.\textsuperscript{14}

**b) Demyelinating disorders**

Patients with Guillain Barre syndrome will show 10 or fewer monocytes per mm\(^3\) and a minority of patients may have 11 to 50 monocytes per mm\(^3\). Patients with acute disseminated encephalomyelitis or multiple sclerosis will usually show up to 50 monocytes per mm\(^3\). Elevated CSF protein and albumin cytological dissociation may be a diagnostic clue in demyelinating disorders.

**Points to Remember**

- **Lumbar puncture should be performed with caution after ensuring that you are not dealing with a child with raised intracranial pressure** – if necessary consider neuroimaging prior to lumbar puncture
- **Whenever you collect the CSF always check the opening pressure and document in the notes** – very simple and useful procedure.

  - Do ensure that the CSF is sent immediately after collection for the various tests and if in doubt it may be useful to store one extra sample in the fridge for a few hours before making a decision about what other tests need to be sent
  - Correct interpretation of the results depends on the history, examination findings and the clinical progress in the child and a CSF results alone should never be taken in isolation.

**References**

9. Zunt JR, Marra CM. Cerebrospinal fluid testing for the diagnosis of central nervous...


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**CLIPPINGS**

**Deis JN, et al. Parental Knowledge and Use of Preventive Asthma Care Measures in Two Pediatric Emergency Departments. Journal of Asthma, 08/06/2010**

Parents of children with persistent asthma presenting to urban tertiary care PEDs with asthma exacerbations frequently have inadequate understanding of appropriate ICS use. Parents with less than a high school education, in particular, may benefit from focused educational interventions that address the importance of daily ICS use in asthma control. Parents who receive a written action plan are more confident in their ability to provide care for their child during an asthma exacerbation.

**Al–Ansari K et al. Nebulized Hypertonic 5%, 3%, and 0.9% Saline for Treating Acute Bronchiolitis in Infants The Journal of Pediatrics, 07/12/2010**

The efficacy and safety of 5%, 3%, and 0.9% saline solution for treating acute bronchiolitis in the prehospital setting was studied. The study concluded that nebulization with 5% hypertonic saline is safe, can be widely generalizable, and may be superior to current treatment for early outpatient treatment of bronchiolitis.

**Patrick W. Brady,Patrick H. Conway,Anthony Goudie, Length of Intravenous Antibiotic Therapy and Treatment Failure in Infants With Urinary Tract Infections. Pediatrics, 08/05/2010**

Treatment failure for generally healthy young infants hospitalized with UTIs is uncommon and is not associated with the duration of intravenous antibiotic treatment. Treating more infants with short courses of intravenous antibiotic therapy might decrease resource use without affecting readmission rates.
CT/MRI-CHOICE OF INVESTIGATION

* Kumaresan G

Abstract: Advances in neuro-imaging have greatly influenced our approach in the management of neurological illnesses. It has replaced invasive investigations. The choice between Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) is influenced by many factors. While undoubtedly MRI is the ideal choice in view of its superiority in diagnosis, CT may be a life saving investigation in deciding management of a sick child in the emergency room. Easy availability and less cost are important points to consider. The risk of radiation to children with long life span and sensitivity to damage are important points to be kept in mind. Adherence to protocols for indications, expertise in pediatric settings and adjustment of parameters as per brain size of the child will reduce the risks.

Keywords: Computed tomography, Magnetic resonance imaging.

Roentgen discovered X-rays in 1895 and was awarded the Nobel prize. The next major advance in the diagnostic field was the invention of Computed Tomography (CT) scan. For this, the inventors Godfrey Hounsfield and Allen Mcleod Cormack were awarded Nobel prize in 1979.

CT scan utilizes ionizing radiation as source for imaging, while in magnetic resonance imaging (MRI), magnetic current is utilized.

The advent of these two investigations has greatly influenced the management of neurological illnesses. However, CT scan can never replace conventional neurological examination. Like in all other investigations clinical correlation is needed. Normal physiological findings like Virchow-Robin space should not be mistaken for abnormalities. Non-specific changes seen in MRI white matter like “unidentified bright objects” have to be recognized and not mistaken for pathological lesions.

Computed tomography (CT)

CT scan brain has the following advantages:

1. It is more easily available than MRI scan. CT scan is the most often used investigation in developing countries for this reason.

2. It is less expensive. CT brain costs roughly one third the cost of conventional MRI.

3. It is the best investigation in emergency setting and the information obtained will influence management strategies. It is a life saving investigation in these circumstances.

4. CT brain delineates bony abnormalities better. Cranial sutures are better visualized. Calcifications are picked up better in CT.

The disadvantages of CT are:

1. It does not show changes in posterior fossa or spinal cord.
2. Artefacts are caused by aneurysmal clips, metallic foreign bodies or dental fittings.

3. There is a lot of disturbing reports about its safety, especially to sensitive brain of children. Children, in view of the long life span have greater cumulative risk. United Nation International Atomic Energy Agency (IAEA) in April 2010 has warned to be cautious about usage of CT in children.

However in many situations as a life saving investigation “benefits outweigh the risks”.

The following guidelines are useful.

1. Is it the best investigation? Are there alternative investigations? Standard indications for CT has to be laid down.

2. Can the dose be adjusted to suit the brain size?

Radiation dose to be based on child’s brain size and adult dose to be avoided.

Region to be scanned to be kept minimum.

Keep the resolution parameters minimum as low as reasonably achievable (Table.1).1

3. Is the team familiar with pediatric CT?

Magnetic Resonance Imaging (MRI): The advent of MRI followed CT scan and has a distinctive advantage over CT scan, especially because of non-usage of ionizing radiation. Various improvements have led to its increasing use. Its other advantages are

1. Better grey-white matter differentiation.

2. Better visualization of posterior fossa structures and spinal cord.

3. Delineates white matter better. The various types of white matter abnormalities like hypomyelination, delayed myelination, demyelination or dysmyelination are well brought out by MRI.

Brain myelination is a dynamic process that begins during the fifth fetal month and continues in a highly structured order (Table.2).

4. Vascular abnormalities, especially venous occlusions seen clearly.

5. Diffusion-weighted images detect ischemia very early and this is vital for early therapy within three hours window period in strokes.

6. Various metabolic studies are useful in the diagnosis of conditions like Leigh’s disease,

<table>
<thead>
<tr>
<th>Table 1. Risk of radiation with CT²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Examination type</td>
</tr>
<tr>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Head (unadjusted) (200mAs)</td>
</tr>
<tr>
<td>Head (adjusted) (100mAs)</td>
</tr>
<tr>
<td>X-ray chest AP</td>
</tr>
<tr>
<td>X-ray chest –Lateral</td>
</tr>
</tbody>
</table>
Krabbe’s. Functional MRI is useful in the differential diagnosis of tumors versus inflammatory lesions. Characteristic lipid peak helps to differentiate tuberculosis from cysticercosis. Functional MRI also helps localisation in epilepsy when other modalities are non-informative and in identifying eloquent areas during surgery. The technique of fibre tractoscopy helps in following white matter fibre tracts like pyramidal fibres as seen in anatomical dissections.

However MRI also has some disadvantages:

1. It is an expensive equipment for installation and maintenance, and is not easily available.

2. It is expensive. The cost ranges from Rs.3,000-Rs.16,000 per patient depending on the strength of magnetic field (1.5-3 Tesla) open or closed unit, other techniques utilized like vascular, metabolic studies or contrast flair studies, diffusion weighted studies, etc. The need for anesthesia and contrast studies will increase the cost. In very sick children equipments compatible with MRI like monitors, ventilators will increase the cost further.

3. Interpretation of pediatric MRI especially in children below two years of age needs a lot of experience. Myelination proceeds in a particular order as per age. Familiarity with this is needed in interpretation at various ages.

4. MRI is very sensitive to movement than CT scan. Hence it is very difficult to perform in very sick children.

5. MRI is not compatible with pace makers, cochlear implants and vagal nerve stimulators.

**How to choose between CT vs MRI?**

**Development disorders:** When CT was introduced, it was not in favour of routine use. The indications were macrocephaly, presence of focal signs, uncontrolled seizures or when the

<table>
<thead>
<tr>
<th>Table 2. Process of myelination of various regions of brain$^3$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Corpus callosum</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Corticospinal tract</strong></td>
</tr>
<tr>
<td><strong>Optic nerve</strong></td>
</tr>
<tr>
<td><strong>Brain stem</strong></td>
</tr>
</tbody>
</table>
diagnosis of static encephalopathy is not definite or when intrauterine infection is suspected (intra cranial calcification). MRI has changed this attitude and has become the investigation of choice in static encephalopathies.

MRI clearly gives useful information about the timing and origin of insult very well. Intrauterine insults before 20-22 weeks of gestation causes hydranencephaly or porencephaly, while insults beyond 30 weeks of gestation cause multi-cystic changes.

The MRI also gives useful information about pathological process. Changes of acute insults-near total versus partial prolonged changes are different.

Acute ischemia causes changes in cortical structures, especially in the basal ganglion, thalamus and brainstem. At the acute stage, cerebral ischemia is picked up very early by diffusion weighted scan whereas conventional MRI or CT picks it by 3-4 days.

Partial prolonged ischemia affects mainly watershed zones ie., parasagittal areas. When prolonged, changes spread beyond watershed zones to involve the rest of the cortex, causing laminar necrosis. This is shown by poor grey-white differentiation.

Chronic insults result in calcification, scar, cystic changes or ulegyria. These changes are best seen in intra uterine strokes as opposed to intrapartum asphyxia.

White matter changes are seen in preterm children. MRI brings this out much better than ultra sound or CT. MRI picks up non cystic component of peri ventricular leukomalacia (PVLM) better than other modalities. Venous occlusions are brought out well by Magnetic Resonance Venography (MRV) studies. Thus MRI has become an important investigation in the evaluation of neo-natal encephalopathy.

Cowen, et al4 found children evaluated for neo-natal encephalopathy to have acute lesions consistent with acute ischemia, 4% had features suggestive of antenatal injury. “MRI is the diagnostic modality of choice in the immediate neo-natal period in infants with hypoxic-ischemic injury”.5

Fetal MRI can be done after first trimester and may show one of the following: 1. Hydrocephalus, 2. Holoprosencephaly, 3. Destruction of brain tissue.

**Intracranial infections**

CT brain will be useful in deducting hydrocephalus, sub dural fluid collections, calcification and basal exudates. MRI will be informative in picking up lesions of white matter like ADEM or multiple sclerosis and venous occlusions.

**Epilepsy**

Calcification and solitary granulomas are picked up by CT brain. On evaluation of uncontrolled seizures especially with normal CT, MRI gives valuable information leading to advances in surgery for epilepsy. Neuronal migration disorders and mesial temporal sclerosis are well picked up in MRI. Functional MRI studies are useful in epileptic surgery.

**Neuro-metabolic/Degenerative diseases**

CT/MRI can give diagnostic clues in many conditions like Wilsons disease, Leigh’s disease, glutaric aciduria or Hallevordan disease. The pattern of involvement of white matter lesions as from central to peripheral or anterior to posterior gives valuable clues. Diagnostic patterns are seen in conditions like adrenoleucodystrophies and Pelzius-Merzbackers disease. In all these conditions MRI is more informative than CT.
MR Spectroscopy helps in confirming the diagnosis in many metabolic diseases.

Thus both CT and MRI have made great impact in modern neurology. Selection between them is based mainly by constraints of availability and affordability. MRI scores over CT as a primary modality of screening of neurologically ill children.

**Points to Remember**

- **MRI is a better mode of investigations as it shows the timing of insult and also changes earlier than CT scan.**

- **CT scan is more easily available and less sensitive than MRI and hence is the choice in emergency department. It delineates bony abnormalities better than MRI and also calcification better.**

- **There is a small risk of radiation in CT scan. However in acute situations the benefits outweigh this.**

**Before ordering a CT scan, careful consideration of the indications and expertise of the person in minimising the radiation dose has to be analysed.**

**References**


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**CLIPPINGS**

**Sun Y et al. Prenatal exposure to elevated maternal body temperature and risk of epilepsy in childhood: a population-based pregnancy cohort study Paediatric and Perinatal Epidemiology, 08/06/2010**

Elevated maternal body temperature during pregnancy is of clinical concern as side effects have been reported. The association between maternal fever and sauna bathing during pregnancy and risk of epilepsy in the offspring was studied. Maternal sauna bathing during pregnancy was not associated with an increased risk of epilepsy. Maternal fever during pregnancy in general was not associated with an increased risk of epilepsy in the offspring and no dose-response pattern was found according to number, level and duration of fever. However they found an increased risk of epilepsy among children exposed to at least 3 fever episodes, maternal fever with symptoms in the urinary system and one-day maternal fever of 39.0–39.4°C. Though their findings do not support a strong association between hyperthermia and epilepsy but the associations between underlying causes of fever, especially prenatal infections, needs further research.
CROUP

* Shanthi S

Abstract: Croup or laryngotraceho bronchitis is an important cause of upper airway obstruction (UAWO) in children aged between 6 months-3 years. It is characterized by sudden onset of barking cough, inspiratory stridor and respiratory distress. It often occurs secondary to a viral infection. The disease is usually mild. However rarely it can present as severe upper airway obstruction (UAWO) necessitating emergent airway management. Glucocorticoids are proven to be the drug of choice for croup. Nebulised adrenaline is administered for moderate and severe croup along with glucocorticoids. Humidified mist has been found to be ineffective in croup.

Keywords: Croup, Upper airway obstruction, Stridor.

Croup is one of the most frequent causes of acute respiratory distress in young children. The term croup refers to a heterogenous group of mainly acute and infectious processes that are characterized by a bark like or brassy cough and may be associated with hoarseness, inspiratory stridor and respiratory distress. Croup also known as acute laryngotracehoobronchitis typically affects the larynx, trachea and bronchi. However in most cases of croup, the laryngeal symptoms dominate the clinical picture over the tracheal and bronchial signs as subglottic edema is the most prominent finding.

Etiology and epidemiology

Croup is often secondary to a viral infection. Parainfluenza viruses (types 1, 2, 3) influenza A and B, adenovirus, respiratory syncitial virus and measles are the common viruses which cause croup. Parainfluenza 1 and 3 and influenza A cause moderate to severe croup. Mycoplasma pneumonia has been associated with mild croup. Laryngeal diphtheria is a well known historical cause for croup in yester years.

Croup is a seasonal disease. The disease mainly affects children between 6 months and 3 years old with peak in the second year. However babies as young as 3 months to adolescents can be affected.. Males are more affected. There is a strong family history of croup in approximately 15% of patients.

Clinical features

The typical patient has a prodrome of upper respiratory infection a combination of rhinorrhea, pharyngitis, mild cough and fever for 1-3 days before the classical features of barking cough, hoarseness and inspiratory stridor develop. The onset is usually gradual and the symptoms typically worsen in the night and aggravated by agitation and crying. The child may have mild respiratory distress. Fever is usually low grade but may be as high as 39°C-40°C. Croup symptoms are generally short lived with about 60% of children showing resolution of their
barky cough within 48 hours and most within one week. Rarely the upper airway obstruction (UAWO) progresses and is accompanied by severe respiratory distress with nasal flaring, suprasternal, sternal and intercostal retractions and continuous stridor. These children may develop hypoxemia and respiratory failure necessitating urgent airway management. Signs of impending respiratory failure include hypotonia, noticeable retractions, decreased or absent inspiratory breath sounds, depressed level of consciousness, tachycardia out of proportion to fever and cyanosis.³

**Complications**

Occur in about 15% of patients with viral croup. The children with croup may develop otitis media and pneumonia. Bacterial tracheitis is an important complication of viral croup.

**Recurrent croup**

Children presenting with recurrent episodes of croup may have any of the following cause: Underlying subglottic stenosis, cysts, hemangioma or laryngeal cysts.

**Differential diagnosis**

In a child presenting with classic symptoms and signs of croup alternate diagnosis is uncommon. Table 1 shows the differential diagnosis in children with croup presenting with moderate to severe UAWO.

Table 1. Differential diagnosis for croup

| 1. Epiglottitis |
| 2. Bacterial tracheitis |
| 3. Foreign body aspiration- Tracheal, esophageal |
| 4. Retropharyngeal abscess |
| 5. Peritonsillar abscess |
| 6. Angioneurotic edema |
| 7. Laryngeal diphtheria |

This tends to run in families. Recurrences are common. Most children respond to nebulised epinephrine.

**Diagnosis**

Croup is a clinical diagnosis. Laboratory tests are not needed to confirm the diagnosis. Radiological studies are not recommended in a child who has a typical history of croup. Radiographs should be considered only after airway stabilization in children who have an atypical presentation or clinical course. An anterior posterior and lateral neck radiographs may show the ballooning of the hypopharynx, subglottic narrowing or ‘steeple sign’ of croup. However this sign may be absent in patients with croup or may be present as a normal variant. Avoid blood tests as the child may get agitated.

**Assessment of severity**

Determination of disease severity depends on clinical assessment. Various scoring systems have been proposed. However such scores are useful for research studies and none has been shown to enhance routine clinical care.

Children with croup are classified as mild, moderate or severe based on the following clinical features.²,⁶
### Table 2. The differences between croup, epiglottitis and bacterial tracheitis.5

<table>
<thead>
<tr>
<th></th>
<th>Subglottic (Viral Croup)</th>
<th>Supraglottic (epiglottitis)</th>
<th>Purulent tracheobronchitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Appearance</strong></td>
<td>Usually not seriously ill</td>
<td>Seriously ill, drooling</td>
<td>variable</td>
</tr>
<tr>
<td><strong>Preferred position</strong></td>
<td>Lying</td>
<td>Sitting</td>
<td>Variable</td>
</tr>
<tr>
<td><strong>Barking cough</strong></td>
<td>Typical</td>
<td>Rare</td>
<td>Possible</td>
</tr>
<tr>
<td><strong>Hoarseness or aphonia</strong></td>
<td>Variable</td>
<td>Typical</td>
<td>Uncommon</td>
</tr>
<tr>
<td><strong>Red or edematous epiglottis</strong></td>
<td>Absent</td>
<td>Typical</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Inspiratory stridor or retraction</strong></td>
<td>Present</td>
<td>Present</td>
<td>Variable</td>
</tr>
<tr>
<td><strong>Expiratory wheezes</strong></td>
<td>Absent</td>
<td>Absent</td>
<td>Often present</td>
</tr>
<tr>
<td><strong>Fever</strong></td>
<td>Variable</td>
<td>Usually high</td>
<td>Moderately high</td>
</tr>
<tr>
<td><strong>Purulent secretions in trachea</strong></td>
<td>Absent</td>
<td>Rare</td>
<td>Typical</td>
</tr>
<tr>
<td><strong>Course</strong></td>
<td>Gradual-days</td>
<td>Rapid-hours</td>
<td>Rapid . Airway needed</td>
</tr>
</tbody>
</table>

Mild - Patients without stridor or significant chest-wall in drawing at rest.

Moderate - Stridor and chest wall indrawing at rest without agitation.

Severe - Stridor and indrawing of the sternum associated with agitations or lethargy.

**Treatment**

Treatment of viral croup is based on the symptoms. Most children with croup can be managed at home. However airway management is the priority in a small number of children who may present with impending respiratory failure.

**General principles:** The children with croup should be kept as comfortable as possible. They should not be frightened or upset because agitation causes worsening of the symptoms. It is preferable to examine the child over the mothers’ lap. Oxygen if needed should be given in a non-threatening manner through oxygen tube with opening held within a few centimeters of the nose and mouth referred to as “blow by” oxygen.

**Humidified air:** is not effective in the management of croup.7,8 On the contrary, potential difficulties with administration of humidified air have been identified. Hot humidified air can cause scald injuries; mist tents are cold and wet and separate the child from the parent which causes the children to be agitated.

**Corticosteroids:** is the drug of choice in the management of croup. Studies have clearly
shown their effectiveness even in mild croup. They decrease the edema in the laryngeal mucosa through their anti-inflammatory action. Oral steroids use in mild croup reduced the need for hospitalization, the duration of hospitalization and the need for epinephrine administration. In children with severe croup and impending respiratory failure steroids reduced the rate of intubation, duration of ventilation and the need for reintubation. Onset of action of nebulised steroids occurs within 2 hours.

**Route of administration and dosage:** Conventionally, oral dexamethasone in a single dose of 0.6 mg/kg is used widely and found to be effective. A lower dose of 0.15 mg/kg may be just as effective. Intramuscular dexamethasone (0.6mg/kg), nebulised budesonide (2mg) and oral dexamethasone all have an equivalent clinical effect. Oral prednisolone (1mg/kg) has also been used in croup. In a child with vomiting, nebulized budesonide or intramuscular dexamethasone is preferable. In a child with hypoxia nebulized steroid may be superior to intramuscular or oral steroid in view of decreased gut and local tissue perfusion.

**Risks of corticosteroids:** Steroid treatment of children with croup is generally safe. Steroids should not be given to children with varicella or tuberculosis (unless the patient is receiving appropriate antituberculosis therapy) because they worsen outcome.

**Epinephrine:** Is indicated in moderate to severe croup. The mechanism of action is probably due to constriction of the precapillary arterioles through the β adrenergic receptors, causing fluid resorption from the mucosal edema. The onset is rapid (10-30 min) but the duration of effect is short (less than 2 hrs). Hence these children should be observed for at least 2 hours as symptoms of croup may reappear to their baseline severity. The administration of epinephrine in children with severe croup has been reported to have reduced the number needing intubation or tracheotomy.

**Dose:** Traditionally racemic epinephrine has been used in a dose of 0.25 – 0.75 ml in 3ml of normal saline. However studies have clearly shown that L- epinephrine is equally effective and safe. The dose of L- epinephrine is 0.5 ml/kg body weight nebulized up to a maximum of 5 ml (10kg or greater). Tachycardia and pallor are relatively common. More serious side effects are rare. Nebulized L- epinephrine should still be used cautiously in patients with tachycardia, heart conditions such as tetralogy of Fallot or ventricular outlet obstruction because of possible side effects.

**Antibiotics:** Are not indicated in croup.

**Heliox:** (70/30 helium/oxygen mixture) has been tried in patients with severe croup who may need intubation. However at present there is no evidence to recommend its routine use in severe croup.

**Management of croup based on severity**

**Mild croup:** Children with mild croup require supportive therapy with oral hydration, antipyretics for fever and minimal handling. Give a single dose of 0.6mg/kg of oral dexamethasone. Parents should be educated about the anticipated course of illness, signs of respiratory distress and when to seek medical attention.

**Moderate croup:** Children with moderate croup require active intervention. Allow the child to assume position of comfort. Administer a dose of 0.6mg/kg of oral dexamethasone and nebulised epinephrine. If symptoms improve within 3 hours patients can be discharged. However,
if recurrent nebulised treatment are required or respiratory distress persists, patients require hospitalization.

**Severe croup**: These children may need airway management. The child should be allowed to assume the position of comfort, oxygen should be given by blow-by-method. Interventions should be minimized including venipuncture. The proper size bag, face mask, intubating equipment should be kept ready. An otolaryngologist and an anesthesiologist should be available to manage the difficult airway and or tracheotomy. A dose of intramuscular dexamethasone or nebulised budesonide (2mg) is given along with epinephrine and the child is monitored for improvement or worsening. If child deteriorates bag and mask ventilation is initiated followed by intubation by the most experienced airway manager. A smaller size (0.5-1mm lesser) endotracheal tube is generally preferred. Shock if present should be treated. Further treatment in the intensive care unit includes nebulised epinephrine with IPPV, dexamethasone and ventilation. These children may develop pulmonary edema once the UAWO is relieved. Extubation should be usually accomplished within 2-3 days.

It is important to remember that the need for intubation in croup is very rare. Only 1-3% of children who are admitted with croup require intubation. Hypoxia in croup is usually secondary to severe UAWO and signals the need for airway intervention rather than administering oxygen.

**Points to Remember**

- **Croup presents with barking cough, inspiratory stridor and respiratory distress.**
- **A single dose of oral dexamethasone 0.6 mg/kg is a very effective treatment.**
- **Nebulised L-epinephrine is as effective as racemic epinephrine.**

- **Epiglottitis and bacterial tracheitis should be considered as differential diagnosis if the child presents with signs of severe UAWO.**
- **In the rare event of respiratory failure, bag mask ventilate the child till expert help is available for intubation.**

**References**


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**Friedman D et al. Pediatric Mock Code Curriculum: Improving Resident Resuscitations. Pediatric Emergency Care, 07/15/2010**

Resuscitation of the acutely ill child is a necessary skill for pediatric residents. The effects of a hospital–wide mock code program on involvement, anxiety and leadership have not been studied. The authors hypothesized that after 1 year of mock codes, pediatric residents would report (1) increased participation, (2) decreased anxiety and increased comfort with knowledge and (3) increased likelihood of leading and feeling capable of running a code. One year after starting a mock code program, residents attended more mock codes and reported more comfort with knowledge in codes. A continued monthly mock code program will provide residents with critical skills training and experience and may translate into active participation, increased leadership and decreased anxiety in actual codes.
INFLUENZA VACCINES

* Senthur Nambi P  
* Suresh Kumar D  
** Ram Gopalakrishnan

Abstract: Pandemics of influenza which occur occasionally get global attention, of which the recent novel H1NI influenza was of no exception. Influenza outbreaks which occur every year resulting in significant mortality and morbidity are easily forgotten. Immunization is the major means of influenza prevention. Two types of influenza vaccines are available: a) inactivated vaccine which is administered intramuscularly and b) live attenuated vaccine which is administered intranasally. Children aged six months through eighteen years and children with comorbidities are a priority for immunization. In addition to the novel H1NI vaccine we should employ annual influenza immunization as a routine in the future.

Keywords: Influenza, Vaccine, H1NI influenza.

Influenza is an acute respiratory illness caused by influenza A or B viruses, which occurs in outbreaks worldwide every year, during the winter seasons in temperate regions and throughout the year in tropical regions. Influenza causes an appreciable disease burden (eg. school and work absence, increased frequency of outpatient medical visits), and children are important vectors for the spread of disease.

Inactivated and live attenuated vaccines against influenza are available and their use represents the major public health measure for prevention of influenza. Since children are an important reservoir of influenza infection, increasing the numbers of immunized children may reduce influenza among unimmunized contacts within the household and community (“herd immunity”). This may be particularly helpful in preventing influenza infection among infants younger than six months and high-risk individuals who did not receive the vaccine.

Two types of influenza vaccines are available. a) Trivalent inactivated influenza vaccine (TIV) administered intramuscularly and b) Trivalent live-attenuated, cold-adapted influenza vaccine (LAIV) administered intranasally.

The vast majority of currently used vaccines are inactivated (“killed”) preparations derived from influenza A and B viruses that circulated during the previous influenza season. The effectiveness during any given season depends upon the match between the circulating and vaccine strains and the activity and severity of circulating strains. If the vaccine virus and the currently circulating viruses are closely related, 50–80% protection against influenza would be expected from inactivated vaccines. The U.S. Public Health Service recommends the administration of inactivated influenza vaccine to individuals who, because of age or underlying disease, are at increased risk for complications of influenza and to the contacts of these individuals. Inactivated vaccines may be

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administered safely to immunocompromised patients (Table 1).

**Table 1. Persons for whom annual influenza vaccination is recommended**

- Children 6 – 59 months old - Children and adolescents (6 months to 18 years old) who are receiving long-term aspirin therapy and therefore may be at risk for developing Reye’s syndrome after influenza
- Children who have chronic disorders of the pulmonary or cardiovascular systems, including asthma
- Children who have required regular medical follow-up or hospitalization during the preceding year because of chronic metabolic diseases (including diabetes mellitus), renal dysfunction, hemoglobinopathies, or immunodeficiency (including immunodeficiency caused by medications or by HIV)
- Children who have any condition that can compromise respiratory function or the handling of respiratory secretions or can increase the risk of aspiration. (eg. cognitive dysfunction, spinal cord injuries, seizure disorders or other neuromuscular disorders)

Vaccine should be administered early in the autumn before influenza outbreaks occur and should then be given annually to maintain immunity against the most current influenza virus strains. Families of infants and children susceptible to severe disease from influenza should be reminded that these patients may contract acute influenza infection despite immunization.

Annual immunization is necessary because immunity declines in the year after vaccination. Two doses of influenza vaccine are necessary for optimal protection in children younger than nine years of age. Protection is increased when both doses are administered during the same influenza season.

- Children >9 years should receive one dose of influenza vaccine.
- Children six months through eight years should receive two doses of influenza vaccine in the first year that they are vaccinated; the two doses should be separated by at least four weeks. The two doses may consist of two doses of TIV, two doses of LAIV (if the child is >2 years of age), or a combination of TIV and LAIV (if the child is >2 years of age).
- Children who are between six months and nine years of age and received only one dose (either TIV or LAIV) in their first season of vaccination should receive two doses of influenza vaccine during the second season. However, if it is the third (or later) season and the child received one dose in the first season, even if no influenza vaccine was administered in the intervening year(s), only one dose is required.
- TIV dose — The dose of TIV varies depending upon the age of the child.
  - 6 to 35 months — 0.25 ml intramuscularly (IM), >36 months — 0.5 ml (IM)

The available inactivated vaccines have been highly purified and are associated with few reactions. Up to 5% of individuals experience low-grade fever and mild systemic symptoms 8-24 hours after vaccination and up to one-third develop mild redness or tenderness at the vaccination site. Since the vaccine is produced from eggs, individuals with true hypersensitivity to egg products either should be desensitized or
should not be vaccinated. Although the 1976 swine influenza vaccine appears to have been associated with an increased frequency of Guillain-Barre syndrome, influenza vaccines administered since 1976 generally have not been. Possible exceptions were noted during the 1992–1993 and 1993–1994 influenza seasons, when there may have been an excess risk of Guillain-Barre syndrome of slightly more than 1 case per million vaccine recipients. However, the overall health risk following influenza outweighs the potential risk associated with vaccination.

The live attenuated influenza vaccine that is administered by intranasal spray is generated by reassortment between currently circulating strains of influenza A and B virus and a cold-adapted, attenuated master strain. The cold-adapted vaccine is well tolerated and has an efficacy of 72 to 82 percent in meta analysis in young children. In one study, it provided protection against a circulating influenza virus that had drifted antigenically away from the vaccine strain. LAIV may be more efficacious than TIV, may provide greater immunity against mismatched strains and may provide immediate protection during an outbreak. Although LAIV is more expensive than TIV, its relative increased efficacy may result in decreased healthcare and societal costs for children 24 to 59 months of age.

Contraindications to LAIV include:

- Age <2 years
- History of anaphylactic reaction to egg or chicken protein, gentamicin, gelatin, or arginine.
- Long-term aspirin or salicylate therapy
- Known or suspected immunodeficiency
- History of Guillain-Barré syndrome
- Asthma
- Recurrent wheezing in children younger than five years
- Other conditions considered to be risk factors for severe influenza infection or complications of influenza infection, including:
  - Chronic pulmonary disorders (including asthma), Cardiac disorders
  - Pregnancy
  - Chronic metabolic disease, Renal dysfunction
  - Hemoglobinopathies
  - Immunodeficiency or immunosuppressive therapy

TIV is preferred to LAIV for contacts of severely immunocompromised individuals (eg, hematopoietic stem cell transplant recipients). Contacts of individuals with lesser degrees of immunocompromise (eg, persons with diabetes, asthma or HIV infection, those who are receiving corticosteroids) may receive either TIV or LAIV vaccine. Healthcare workers who receive LAIV should refrain from contact with severely immunocompromised individuals for seven days. LAIV can be administered to children with minor acute illnesses, with or without fever. Clinical judgment should be used before administering the vaccine to children with nasal congestion of such severity that it may impede delivery of the vaccine to the nasopharyngeal mucosa. Administration of the vaccine may be deferred until resolution of illness unless deferral of vaccination may result in the child not being vaccinated. If the child sneezes after LAIV is administered the dose should not be repeated.

LAIV dose — The dose for LAIV is 0.2 ml administered intranasally (0.1 ml to each nostril). LAIV can be administered at the same time as
other live and inactivated vaccines. However, if it is not administered on the same day as other live vaccines (eg, measles-mumps-rubella, varicella zoster), it should be administered at least four weeks later. The safety of LAIV has been evaluated in a number of trials including more than 18,000 children younger than five years. Few adverse events occurred in controlled trials in healthy children 12 months to 18 years of age. Nasal congestion/runny nose and fever $>100^\circ\text{F}$ ($37.8^\circ\text{C}$) occurred in at least 5 percent more vaccine than placebo recipients after the first dose (58 versus 50 percent and 16 versus 11 percent for congestion and fever, respectively).

**Pandemic H1N1 influenza vaccine**

In terms of susceptibility, the rate of H1N1 infection globally has been highest among children and young adults. The CDC recommends that H1N1 influenza vaccine be given to all persons six months of age and older. Though non-adjuvanted and adjuvanted formulations of the pandemic H1N1 influenza vaccine are being evaluated, only non-adjuvanted vaccines have been approved for use in the United States. Vaccination against pandemic H1N1 influenza A will not provide protection against seasonal strains of influenza and vaccination against seasonal influenza is not expected to provide protection against pandemic H1N1 influenza A infection as both strains are antigenically different. Since the pandemic H1N1 influenza A vaccine is similar in design to the seasonal influenza vaccine, adverse effects are expected to be similar to those seen following seasonal influenza vaccination. To date, the pandemic H1N1 influenza vaccine appears to be safe and well tolerated.

The Advisory Committee on Immunization Practices recommends that infants and children aged 9 years or younger receive two doses of H1N1 influenza vaccine at least 21 days apart, based on existing experience with seasonal trivalent influenza vaccines in this age group. Individuals $>10$ years of age should receive one dose. The World Health Organization (WHO) recommends a single dose of vaccine not only for individuals $>10$ years of age, but also for younger children in order to provide some protection to as many children as possible.

**Dosage**

- Inactivated vaccine: Children 6-35 months: 0.25 ml/dose (7.5 mcg); administer 2 doses, IM
- Children $>36$ months: 0.5 ml/dose (15 mcg); administer 2 doses, IM
- The dose for LAIV: is 0.2 ml administered intranasally (0.1 ml to each nostril).

Regarding coadministration of pandemic H1N1 and seasonal influenza vaccines, several studies suggest that immunogenicity is retained if pandemic and seasonal influenza vaccines are coadministered. Inactivated vaccines against seasonal and pandemic H1N1 influenza viruses may be administered simultaneously if different anatomic sites are used. In contrast to recommendations for the administration of inactivated vaccines, simultaneous administration of live attenuated intranasal vaccines against seasonal and pandemic H1N1 influenza viruses is not recommended. For individuals with indications for both of the live attenuated influenza vaccines (against seasonal and pandemic H1N1 influenza viruses), the minimum recommended interval between the vaccines is 14 days, and ideally at least 28 days.

**Conclusion**

Paediatric population should be vaccinated against pandemic H1N1 influenza and annual seasonal influenza vaccination should be strongly considered (Table 2).
Table 2. Risk categories and type of influenza vaccine to be given

<table>
<thead>
<tr>
<th>Risk category</th>
<th>Type of influenza vaccine</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 through 4 years (59 months)</td>
<td>Vaccine not licensed for this age group; household contacts and out-of-home caregivers should receive TIV or LAIV</td>
</tr>
<tr>
<td>0 to 6 months (High risk for 2009-2010 influenza season)</td>
<td>TIV</td>
</tr>
<tr>
<td>&gt;24 months (Not considered high risk for 2009-2010 influenza season)</td>
<td>TIV or LAIV</td>
</tr>
<tr>
<td>6 to 24 months (High risk for 2009-2010 influenza season)</td>
<td>TIV</td>
</tr>
<tr>
<td>Chronic disorders: (High risk for 2009-2010 influenza season)</td>
<td>TIV</td>
</tr>
<tr>
<td>Pulmonary (including asthma in children of all ages and history of wheezing in</td>
<td>Renal</td>
</tr>
<tr>
<td>the previous 12 months for children aged 2 through 4 years)</td>
<td>Cardiovascular disease (excluding hypertension)</td>
</tr>
<tr>
<td>Congenital heart disease with functional abnormalities</td>
<td>Chronic metabolic disease (including diabetes mellitus), renal dysfunction, hemoglobinopathy, or immunosuppression (</td>
</tr>
<tr>
<td>Renal</td>
<td>including immunosuppression secondary to medications)</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Long-term aspirin or salicylate therapy</td>
</tr>
<tr>
<td>Metabolic (including diabetes mellitus)</td>
<td>Residents of chronic-care facilities</td>
</tr>
<tr>
<td>Disorders that compromise respiratory function, or the handling of secretions,</td>
<td>TIV</td>
</tr>
<tr>
<td>or that increase the risk of aspiration (eg, cognitive dysfunction, spinal cord</td>
<td>TIV</td>
</tr>
<tr>
<td>injury, seizure disorder, neuromuscular disorder)</td>
<td>TIV</td>
</tr>
<tr>
<td>Chronic metabolic disease (including diabetes mellitus), renal dysfunction,</td>
<td>TIV</td>
</tr>
<tr>
<td>hemoglobinopathy, or immunosuppression (including immunosuppression secondary</td>
<td>Long-term aspirin or salicylate therapy</td>
</tr>
<tr>
<td>to medications)</td>
<td>Residents of chronic-care facilities</td>
</tr>
<tr>
<td>TIV: Trivalent inactivated influenza vaccine; LAIV: Live attenuated influenza</td>
<td>TIV</td>
</tr>
<tr>
<td>vaccine.</td>
<td></td>
</tr>
</tbody>
</table>

Points to Remember

- **Immunization is the major means of influenza prevention. Two types of influenza vaccines are available: inactivated vaccine (TIV), which is administered intramuscularly, and live-attenuated, cold-adapted influenza vaccine (LAIV), which is administered intranasally.**

- **LAIV is not licensed for use in children younger than two years, nor in children with asthma or risk factors for serious influenza disease. TIV should be used for children 6 to 24 months of age and young children with a history of wheezing.**

- **Annual immunization against seasonal strains of influenza is necessary because**
immunity declines during the year following vaccination.

• Pandemic H1N1 influenza A vaccine will not provide protection against seasonal strains of influenza, and vaccination against seasonal influenza is not expected to provide protection against pandemic H1N1 influenza A infection.

• Immunogenicity is retained if pandemic and seasonal influenza vaccines are coadministered.

Acknowledgement: V. Ramasubramanian, Senior consultant, Infectious diseases, Apollo Hospitals, Chennai Abdul K. Ghafur, Consultant, Infectious diseases, Apollo Hospitals, Chennai

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COMMON OPHTHALMIC PROBLEMS IN CHILDREN

* Harsha G Mangalgi
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Abstract: Childhood cataracts may have associated systemic diseases. Cataracts have to be detected and operated early. Strabismus or squints have to be diagnosed and treated appropriately to restore binocular vision. Children may have a decreased vision in one eye due to amblyopia. Routine screening can detect this and offer early treatment. Nasolacrimal duct block, causing tearing have to be differentiated from the most feared congenital glaucoma that also cause tearing. Children who are premature, low birth weight or have had a stormy perinatal course, needs an early eye examination. Cortical visual impairment (CVI) is a common but under diagnosed cause of visual loss in children.

Keywords: Congenital cataract, ROP, Strabismus, CVI.

Cataracts

Cataracts in the pediatric age group require special attention and these patients cannot be compared to those with adult senile cataract. Cataract in children poses much greater problems in comparison with its adult counterpart in terms of serious and disabling complications such as amblyopia.

These children also need to be evaluated thoroughly as the cataract maybe just one feature of a more widespread systemic affliction.

Congenital cataracts may be isolated, inherited as an autosomal dominant condition or part of a systemic syndrome like congenital rubella. Timing is of the essence in the detection and management of congenital cataract. It is imperative that all children are screened for the presence of red reflex soon after birth using an ophthalmoscope by the pediatrician. This is best followed by one more screening at the first month at the time of the vaccination.

Bilateral congenital cataracts are the most common cause of treatable blindness in children and account for about 5% – 20% of blindness in children worldwide.1,2

Cataract surgery in infants has advanced and gives excellent results. It is best undertaken by a pediatric ophthalmologist. Children have the best potential for vision if operated around one month of age. Numerous studies have shown that a clear focused image is necessary for good visual development and this must occur within the first 2 months of life.3,4

Good vision and even stereoacuity can be obtained in dense unilateral cataracts operated on by 6 weeks of age.5,6

Rodgers, et al showed that visually significant bilateral congenital cataracts cause
irreversible amblyopia and sensory nystagmus unless the retinal images are cleared before 2 months of age.

Bilateral complete congenital cataracts should be removed within the first few months of life, first in the eye with the more opaque lens opacity and approximately within a week later in the other eye. This interval should be brief (5-10 days).

**Retinopathy of prematurity (ROP)**

Retinopathy of prematurity is a disease that affects immature vasculature in the eyes of premature babies. Retinopathy of prematurity may occur not only in premature children with low birth weight but even normal birth weight who have had a stormy perinatal course. All children who are premature need an eye exam within 4 weeks of birth. Screening and laser photocoagulation has clearly shown to reduce the incidence of blindness from ROP.

It can be mild with no visual defects or it may become aggressive with new blood vessel formation (neovascularization) and progress to retinal detachment and blindness.

As the survival of smaller and preterm babies is improved, the incidence of ROP has increased

- ROP in premature babies is inversely proportional to their birth weight (16% to 56%).
- Fielder studied infants weighing less than 1700 g and noted development of ROP in 51%.
- All babies less than 1500 g birth weight or younger than 32 weeks’ gestational age (GA) at birth are at risk.

Most infants who develop retinopathy of prematurity undergo regression of their disease, and for those who do, retinopathy of prematurity lasts approximately 15 weeks.

**ROP screening protocol:** As more preterm and smaller infants are surviving, the screening protocols are changing to include earlier GA. In any neonatal intensive care unit (NICU), the timing of the first evaluation must be based on the GA at birth.

Screen all eligible babies at

- 31 weeks post conceptional age or 3-4 weeks after birth whichever is earlier
- Infants weighing less than 1200 grams at birth and those born at 24-30 weeks are usually screened earlier, usually not later than 2-3 weeks after birth
- No examination needed in the first 2-3 weeks of life
- Next screening decided by ophthalmologist based on findings in the first screening
- Complete one screening session before day 30 of the infant’s life

The development of the visual system may be affected in many ways, even after the ROP has resolved.

There may be macular dragging, glaucoma, strabismus, refractive errors and amblyopia that may, even after aggressive treatment, lead to visual loss. Hence regular follow up of these children is very essential.

**Congenital nasolacrimal duct obstruction**

Congenital block of the nasolacrimal duct is the commonest developmental anomaly of the lacrimal apparatus. It is a very frequent anomaly in a child and commonest anomaly of the eye and adnexa. It is found in about 6% of new borns. It is more common in premature children.
It may be unilateral or bilateral; both the sex are equally involved and there may be a positive family history. Commonest site of the obstruction is at the inferior osteum of the duct. A typical presentation is watery discharge in a congested eye with out photophobia. After few days, the conjunctiva gets inflamed and there is mucopurulent discharge. At this stage pressure over the sac may result in positive regurgitation.

Management of the condition is simple provided the treatment is initiated before twelve weeks. The treatment consists of opening of the block by external pressure over the sac and treatment of associated infection. Relief of the obstruction is achieved by application of gentle and firm pressure over the sac to force the fluid down the nasolacrimal duct, which dislodges the epithelial debris.

The application of pressure is generally called massaging of the sac. This gives relief in 95-98% of cases. The mother is taught this simple method by repeated demonstration and is asked to perform the same in presence of the doctor. She is instructed to trim finger nails and keep the hands clean. The sac is pressed with the pulp of the index finger. After the sac has been pressed the mucopurulent discharge should be removed by wet cotton swab and a broad spectrum antibiotic drop is instilled.

The process of cleaning the hands, pressing the sac, removing the discharge, instillation of antibiotic should be under gone for five to six times a day for six weeks. However, all watering in an infant need not be due to nasolacrimal duct obstruction. If there is associated photophobia, cornea is cloudy or cornea looks large it may be the more vision threatening congenital glaucoma. This needs early referral to a tertiary care eye centre.

**Strabismus and amblyopia**

Strabismus is treated in a timely fashion to restore binocularity. Strabismus can be physiological for first three months of life. Any squint persisting beyond 3 to 4 months of age needs to be examined. It may be associated with amblyopia or reduced vision in one eye which can be detected only in a routine eye exam. Tests to differentiate squint from pseudosquint are known as cover test, corneal reflex test which are usually performed by ophthalmologist. It is treatable by simple methods. Strabismus may be corrected with glasses, exercise, prisms or surgery.

<table>
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<tr>
<th>Table.1. Ophthalmic examination at various ages</th>
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<tr>
<td><strong>Steps</strong></td>
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<td>Inspection</td>
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Cortical visual impairment

Children who have a stormy perinatal course with seizures, hypoglycemia etc. may develop cortical visual impairment. If such a child shows delay in visual development, a pediatric ophthalmologist can help diagnose cortical visual impairment and suggest appropriate rehabilitation. Clinically the eye examination may be normal cortical visual impairment except the finding of poor vision. There may be existing optic atrophy. Child would have other neurological problems due to perinatal hypoxia.

Visual development is most active and vulnerable during the first 3 months of life, which is termed the critical period of visual development. Visual acuity development continues up to 7 to 8 years of age, but development is slower and plasticity is progressively less in later childhood. Abnormal visual stimulation by a blurred retinal image or strabismus during early visual development (e.g., congenital cataract, strabismus) can result in permanent damage to visual centers in the brain.

Early treatment of pediatric eye disorders is important to promote normal visual development. Vision screening examinations should start at birth and continue as part of routine checkups for primary care physicians.

The acronym I-ARM (inspection—acuity, red reflex and motility) can be a helpful guideline (Table.1).

Points to Remember

- **It is important to test the red reflex in the eyes of a child soon after birth and at the first vaccination visit with an ophthalmoscope to detect developmental cataracts.**

- **Beware of congenital glaucoma as a cause of tearing in a child. It is usually associated with photophobia.**

- **Even children with normal birthweight and almost term can develop retinopathy of prematurity if they have had a stormy perinatal course. Such children need an eye examination within 4 weeks.**

- **All children with squints need early examination to detect amblyopia and correction of squint to restore binocularity.**

- **Children with birth asphyxia, neonatal hypoglycemia or seizures may develop cortical visual impairment.**

References


CHOICE OF ANTIBIOTICS IN OFFICE PRACTICE

* Palani Raman R

Abstract: Antibiotic is the most common drug prescribed by a pediatrician in office practice next only to paracetamol. To write a rational prescription he should be knowledgeable about when to use, when not to use and also the hazards of irrational use of antibiotics. It is a fact that simple, safe, first line narrow spectrum antibiotics are more than enough to treat common community acquired infections. Antibiotic resistance is not a very common phenomenon with respect to common community acquired infections with the exception of typhoid.

Keywords: Antibiotics, Office practice, Rational, Resistance.

Antibiotic prescription is an every day affair in office practice. Antibiotics are the most frequently used and arguably, the most frequently abused agents in the pharmacopoeia. One has to develop the art of rational prescription of antibiotics particularly when, what, and how to use over the years.

A rational prescription should give answers to the following three questions,

1. When to use?
2. When not to use? and
3. What are the hazards of irrational use?

When to use?

One must be well acquainted with the common conditions which are bacterial in origin. In office practice, these constitute just 10% of our patients. In short, the need to use antibiotics only in 1 out of 10 patients. We should have a thorough knowledge with these handful of conditions, organisms and drugs.

Golden rules of rational antibiotic usage

- We must have a provisional diagnosis in the first place.
- Then an educated guess about the most probable organisms involved. (Age and site of infection plays a major role in this guess).
- Must also have knowledge about the prevalence of sensitivity pattern in the community.
- Then choose the narrowest spectrum antibiotic which hits the organism hard, as well as the best antibiotic with less side effects, with a low cost.

Common bacterial infections in the order of prevalence in office practice are as follows:

1. Skin and soft tissue infections
2. Acute suppurative otitis media
3. Acute dysentery
4. Acute lower respiratory infections
5. Urinary tract infection
6. Acute tonsillopharyngitis
7. Acute lymphadenitis
8. Sinusitis
9. Enteric fever
10. Bites, etc.

For understanding, we can consider the above infections under following groups:

**Group A**

Skin and soft tissue infections, acute lymphadenitis, bites.

**Organisms:** Streptococcus, Staphylococcus and anaerobes for bites

**Drugs of choice:** First generation cephalosporins

Cephalexin - 50mg/kg/day, Cefadroxil – 30 mg/kg/day, Cloxacillin- 50mg/kg/day, Co-Amoxyclov - 40mg/kg of amox.component.

For bites (animal/human), co-amoxyclov is the drug of choice.

**Group B**

Pneumonia, acute otitis media and acute sinusitis.

**Common pathogens:** Strep.pneumoniae, H.influenza, Moraxella, atypical organisms i.e mycoplasma and chlamydia (Pneumonia <1 month and >5years)

**Drugs of choice:** 1st line antibiotic is amoxycillin – 40mg/kg/Day.

2nd line antibiotics is amoxycillin-clavulanate – 40mg/kg of amoxycillin component

2nd and 3rd generation oral cephalosporins – cefuroxime – 30mg/kg, cefdinir – 15 mg/kg, cefpodoxime – 10mg/kg

For outpatient management of pneumonia

Dosage of amoxycillin to be doubled - 80-100mg/kg

Suspected atypical pneumonia/pertussis: Azithromycin - 10mg/kg for 5 days.

Acute pharyngotonsillitis– Streptococcus pyogenes

**Drugs of choice:** Penicillin – 250mg BD for children <30Kg, 500mg BD if >30Kg. for 10 days (or) amoxycillin– 40mg/kg for 10 days (or) first generation cephalosporins. macrolides in case of β-lactam allergy (azithromycin – 12mg/kg for 5 days),

**Group C**

Acute dysentery : Shigella, enteroinvasive E.Coli

UTI : E.Coli

Enteric fever – Salmonella typhi and paratyphi

**Drugs of choice**

Cefixime – 10mg/kg (dysentery and UTI),

20mg/kg (typhoid)

Ofloxacin – 10 - 20mg/kg (enteric fever)

Duration differs for each condition: For dysentery – 5 days, UTI and enteric fever – 14 days.

In sick children where oral intake is not possible, Inj. Ceftriaxone is the drug of choice. Aminoglycosides are useful only for UTI, whereas Shigella and Salmonella are intracellular pathogens where aminoglycosides do not act.

**Antibiotics of choice for prophylaxis**

Antibiotics are to be given as chemoprophylaxis only in select definitive situations. The common infections in office practice where prophylactic antibiotics to be used are,

a) UTI : Co-trimoxazole (after 2 months of age)

2mg/kg of trimethoprim or cephalexin - 10mg/kg as a night time dose.

b) Rheumatic fever : PenicillinV-250mg twice daily.
c) Pertussis contacts: Azithromycin – 10mg/kg for 5 days.

d) Tuberculosis: Baby of AFB positive mother – INH(10mg/kg) for 6-9 months. Six months of INH chemoprophylaxis is recommended for all children under 6 years of age with contact of an infectious case.

e) For patients with heart disease, before orodental and other invasive procedures use amoxicillin (50mg/kg) one hour before the procedure.

**Antibiotic resistance in office practice**

It is not a major issue with respect to community acquired pathogens except for enteric fever where multi-drug resistance is a common phenomenon. Nowadays we have in small percentage of community acquired infection, have methicillin resistant staphylococci (C-MRSA), where clindamycin and co-trimoxazole are the drugs of choice.

**When not to use antibiotic?**

As already explained, except for the 10% of conditions covered, the remaining 90% are viral in origin where antibiotics are misused. Common conditions like fever, cold, cough, diarrhoea, etc are mostly viral and one should be knowledgable enough to differentiate this from bacterial etiology. The reasons for misuse are:

a) ignorance of the above facts, b) fear of secondary infection, c) false sense of security, d) fear of losing the patient and e) parental anxiety and pressure.

**Hazards of irrational use**

1. Increases the emergence of antibiotic resistant organisms in the community.

2. Increase the conditions like brain abcess, empyema and chronic renal failure secondary to partial treatment and starting antibiotics without proper diagnosis.

3. Increasing the cost of treatment and overall increase of the economic burden to the country.

**Summary**

Antibiotic usage in office practice is both art and science. Let us practice this exercise everyday in a rational way by updating ourselves and by following self-discipline in prescription. Over the years it will be a pleasant exercise leading to a drastic reduction in irrational usage of antibiotics.

**Points to Remember**

- **Only 10% or less of our patients need antibiotics (benign bacterial infections like AOM, dysentery).**

- **Let us be familiar with these 10% of conditions and think twice before we write antibiotics. (Avoid using antibiotics for the remaining 90% which are mostly viral in origin).**

- **In short, amoxycillin is the drug of choice for respiratory conditions, cefixime, the drug of choice for GI and GU conditions and cephalosporins (1st generation) the choice for skin and soft tissue infections.**

- **Antibiotic resistance is not a common phenomenon with respect to common community acquired infections except for typhoid and TB.**

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*Why are they having infant colic? A nested case–control study Paediatric and Perinatal Epidemiology, 08/18/2010*

The study aimed to analyse infant (birth characteristics, feeding type, faecal enzyme activities) and environmental (maternal smoking, nutrition and psychological status, mother–child bonding, family structure, support for the mother, familial atopy) risk factors for infant colic and to follow infants with respect to physical growth, sleeping status up to 8 months of age in a nested case–control study. When the infants were 7–8 months old, another interview was done. The colic group had higher proportions of less-educated (<8 years) and smoking mothers, extended family and families with domestic violence than the non-colic group. The colic group of mothers had significantly higher rates of ‘impaired bonding’ in the Postpartum Bonding Questionnaire, higher scores on the Edinburgh Postnatal Depression Scale, higher scores for hostility subscales of the Brief Symptom Inventory and a more irregular sleep pattern than the non-colic group. No differences were revealed for faecal enzyme activities. At 7–8 months, the colic group was shorter than the non-colic group. Colic was associated with various perinatal factors (maternal education, smoking habits, cheese consumption, hostility scores and domestic violence) and having colic in infancy negatively affected the sleeping pattern and the height of the infant.

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The article documents the change in anterio–posterior pelvic diameter (APPD), cortical thickness (CT) and pelvis/cortex ratio (P/C ratio) following pyeloplasty, and determine the usefulness of each of these parameters in assessing postoperative outcome. P/C ratio could be used as an early marker of success following pyeloplasty. The authors recommend improvement in other sonographic criteria or on radionuclide scans which may be useful in the long term.
MEDICAL DOCUMENTATION

* Ramachandran B

Abstract: Medical documentation is extremely important, both for purposes of patient care and for medico legal reasons. However, this skill rarely taught in medical college or during postgraduate training. This article reviews the principles of writing accurate and concise notes. A structured format, known as the SOAP note, will also be discussed.

Keywords: Documentation, Notes, Medical.

Writing good notes is an art. Unfortunately, notes are often written in a hurry and may be incomplete or unclear. Complete and accurate documentation is essential to ensure continuity of care. In the court of law, the best defense is a well-written case sheet. Though documentation is so important, it is unfortunately not taught in medical school or even during post-graduate training.

We will review the fundamentals of writing good notes, including admission, progress, procedure, transfer notes, discharge summaries and prescriptions. Using a structured framework has been shown to take less time and be more complete than writing notes from scratch.

General points

Each note should have a proper heading. Both the date and time should be written and the writing should be legible to others. Erasures or correction fluid should not be used – instead, mistakes should be stricken through and the word “error” written on top. The note should end with the signature and printed name of the writer, along with his or her designation. Each page of a multi page notes should be signed and carry the date and time.

Always be truthful – if you did not examine a system, skip it rather than making something up. Never add to, or delete from, an existing note or chart – instead, make a new, corrected entry. Always document what the family has been told. Avoid wordiness and jargon – medical notes are not the place to exhibit your command of the language.

Admission notes

A clear and complete admission note is essential. It need not be long and a pre-printed format is acceptable. The note should document the presenting complaint, history of the presenting complaint, family history, physical examination, investigations, assessment, plan and the presence or absence of known allergies.

Admission orders

This should have the destination ward, admitting consultant, diagnosis, activity, diet, allergies (presence or absence), weight, i.v. fluids, medications, investigations, special monitoring and when the physician should be informed.

Progress notes

The requirements for a progress note include being readable, easily understood, complete,
accurate and concise. These pre-requisites can be easily satisfied by using a structured approach to writing notes. One such technique is called the S.O.A.P. format.¹

S Subjective – what the patient says

O Objective – what you find on examination, including vital signs and lab reports

A Assessment

P Plan – can be problem oriented for complex patients

**Subjective** : In this section, mention why the patient has come. Describe his problems concisely and include only information pertinent to his complaints. State what the patient (or parent) says. If it is a new patient, document the past medical history, medications, allergies, family history and any relevant social history. This section applies equally to both out patients and in patients.

**Objective** : What do you see? This is the physical examination, which should be focused on the systems related to the patient’s complaints. Vital signs are mandatory. Include the maximum temperature in the past 24 hours. Write the examination in order from head to toe, even if you did not perform it that way.

In addition, state what lab tests have already been performed – list these and the results after the physical examination. Don’t list investigations that are going to be performed – they will come later, under the plan.

**Assessment** : In this section, state what you think is going on with the patient. List the diagnosis, if you are sure, or the possible diagnoses, if you are not. Imagine that you will not be there tomorrow and someone else has to figure out why you chose the tests or treatment that you did by reading your assessment.

**Plan** : State what you are going to do. What lab tests are planned and when. What medicines are being started? If an out patient, what should the patient watch out for and when should he call the physician immediately. What are the plans for follow up?

**Procedure note**

It is essential to document every major procedure performed on a critically ill patient – the note should include the indication, sedation/analgesia used, the brief details of the procedure, whether the procedure was successful and the presence or absence of any complications. The note should also state whether separate informed consent was obtained. Procedures that should have a procedure note include lumbar puncture, central venous catheterization, swan ganz catheterization, arterial catheter insertion, chest tube insertion and bone marrow aspiration (or intraosseous needle insertion).

**Prescription writing**

It is extremely important to write all prescriptions clearly – misinterpreted prescriptions cause thousands of iatrogenic problems in hospitals.

- Each prescription should have the date and time, and the weight of the patient (on the initial prescription). Indicate the drug name, dose, strength, route and frequency for every order.
- For single doses, write “x 1” or “once”. For drugs to be given as needed, write “prn” (not “sos”) and the parameters for giving a dose.
- To discontinue an order, write a DC order – do not just cross out the existing order.
- To correct an incorrect order strike through and rewrite.
• Do not add to an order after the nurse has signed it off.

• For drugs where serum levels correlate with therapeutic response (antibiotics, CVS drugs, theophylline etc.), write dosing intervals (q 8 hrs, q 12 hrs, etc.), rather than t.i.d. or b.i.d.

• For whole numbers, do not add a decimal and 0 (e.g. write 10 mg, not 10.0 mg – this can be misread as 100 mg!). For fractions less than 1, always add 0 plus a decimal point (e.g. 0.5 mg, not .5 mg).

• Do not order in ampoules, mL or tablets – drugs come in various preparations and incorrect dosing can occur. For example, phenytoin comes as both 50 mg and 100 mg tablets and suspensions of 30mg/5 mL (Eptoin) and 125 mg/5 mL (Dilantin).

• Always discuss the order with the nurse to make sure there is no misunderstanding.

• Most importantly, write and sign legibly

Transfer note

A transfer note is needed whenever a patient is transferred from the ICU to either the ward or to another hospital. The note should include the presentation, relevant investigations performed, course in the ICU, procedures performed, complications if any, the current status, ongoing treatment required and a list of investigations for which results are pending.

A good transfer note need not be long, but it will go a long way in ensuring that proper care is continued, since the receiving physician need not go through several pages of ICU notes to try and decipher the course in the ICU.

Discharge summary

The discharge summary is an extremely important document. This statement will be seen by other physicians and you will be judged by the quality of the discharge summary. Assume that this patient has been referred to you and write the discharge summary so that it gives all the information that you require, in an easily readable format.

The summary should contain a brief clinical summary, including the presentation, relevant investigations, diagnosis, treatment, hospital course, procedures, complications, outcome, discharge medications and advice and what follow up is required. Use complete sentences and avoid abbreviations that persons not from your specialty will not understand. Pay particular attention to spelling and grammar and above all, avoid unnecessary detail.

Points to Remember

• Good notes are essential for both patient care and medico-legal purposes

• Notes should be concise and legible

• Date, time and proper signature are a must

• Progress notes can be written in a SOAP format

• Prescription errors can and do kill patients

Reference

DIFFICULT ADOLESCENT

*Yamuna S

Abstract: Adolescents who do not conform to the expectations of parents and other significant adults are perceived as “Difficult” adolescents. They present either with externalizing or internalizing behaviors and reach clinicians when their social functioning is also affected. They are usually unhappy and the reasons for their behavior have to be explored with one to one interaction with the adolescent and the parents. Depression and associated psychiatric morbidity deserves the attention of the mental health professionals. Positive parenting tips help parents in bringing back family connectedness.

Keywords: Difficult Adolescent, Parenting.

An adolescent who is perceived as non cooperative or not fitting into the expected frame of behavior is considered a ‘Difficult’ adolescent. Such adolescents are usually brought to the attention of the clinician when the social functioning is also affected.

Animals have species typical behaviors. Humans are flexible and the behaviors are influenced by environment, desire and motivation. Human behavior is the collection of behaviors exhibited that are influenced by culture, attitudes, emotions, values, ethics, authority and the genetic make up.

When an adolescent behaves in a manner that is different from the pattern of behavior that is expected for the culture, parents and other adults make an earnest attempt to rectify the same. When the adolescent is reluctant to modify the behavior he or she is considered as “Difficult” adolescent.

Behaviors that label an adolescent as “Difficult” can be grouped as follows based on the perceptions of the adults around the adolescent.

Physiological behaviors

Behaviors not accepted by parents who belong to a certain culture with certain habits, eg. sleeping late, studying late in the night

Behaviors not understood by parents as they have not seen such patterns of behavior before. eg. listening to music for prolonged periods of time, sleeping in a separate bedroom

Behaviors not accepted by the microcosm in which the parents lived eg. walking with members of opposite gender from school, sleep overs, etc

Behaviors perceived as different because the parents did not have them at a similar age eg. Crushes at 12 and proposing to each other around 14 years!

The above behaviors are physiological but may be anxiety provoking in a parent who is used to collectivistic, conservative, culture abiding way of life. Delayed phase shift in the sleep rhythm, self entertainment and individualization,
healthy cross gender friendships, and attractions to members of opposite gender are part of normal adolescent processes and need to be considered as normal milestones in the development of an adolescent.

**Externalizing behaviors**

These are behavior manifestations of disturbances in an adolescent directed outwards or at others.

Aggressiveness, argumentativeness, disobedience / non cooperation, disruptive and damaging to property, use of obscene language, use of violence and indulgence in objectionable activities like lying, stealing, bullying, eve teasing, etc are some of the externalizing manifestations.

**Internalizing behaviors**

These are behavior manifestations of disturbances in an adolescent that are directed inwards and at self.

Socially isolated, withdrawn adolescents are easy targets for bullying and teasing. They are usually neglected by peer group members, and remain isolated and withdrawn in social settings. These adolescents are either shy or scared to meet and interact with others, and are often in a depressed state of mind with frequent crying and tearful states. Such adolescents express death wish, suicidal ideation and thoughts about running away from parental home or dwelling places like hostel, etc. They may also be anxious and restless and deprive themselves of normal self care. These are the internalizing adolescents who may harbour suicidal ideation.

**Clinical presentation of a “Difficult” adolescent**

Adolescents who are perceived as “Difficult” are usually brought by parents when the normal functioning is affected. Sometimes these adolescents are referred to clinicians by school authorities. Few adolescents are brought to clinical care when the peer group members see a change in the disposition of the index adolescent.

**Presenting features**

Drop in grades and poor scholastic performance, diminished drive to strive towards academic excellence in an adolescent who was a topper, school refusal, truancy when school attendance is insisted by parents, non cooperation, being violent and aggressive with parents and siblings, attempt to run away from home, attempt at self harm by wrist slashing, head banging, starving, etc., frequent change in friendships and relationships, reckless driving, addictive behaviors including texting, chatting, social networking, prolonged television watching, playing web based games for long hours, pornography, substance use, sexual promiscuity, etc., social withdrawal by locking self within the bedroom or sometimes hiding in the loft, are some of the commonly reported features by parents when they bring “Difficult” adolescents for consultation.

Breaking school disciplinary ground rules like not being punctual, not adhering to uniform requirements with respect to grooming, breaking or damage to school property like sinks in the toilets, window panes, etc. as a reaction to being reprimanded at school, not being regular at school assignments, being irritable and violent at the slightest provocation, use of obscene abusive language at rival groups, usage of substances within school premises, spending school hours in a video game parlor, are some of the reasons that make school authorities refer adolescents for clinical care.

Frequent crying, attempts at self harm and self injury, withdrawn behavior make peer group of a “Difficult” adolescent reach clinical setting.
Clinical examination

It is recommended that a separate session with the parents/ accompanying adults of the adolescent is conducted prior to seeing the “Difficult” adolescent. This exclusive session with the parents is usually helpful in understanding the “Difficult” behaviors of the adolescent as perceived by parents and others and in understanding the sociocultural expectations of the parents from their “Difficult” adolescent.

During clinical examination it is essential to create rapport with the adolescent and the family for better inputs. Adolescent is our client and he or she should be treated with respect and unconditional positive regard.

Clinical evaluation of the adolescent

It is recommended that the interaction begins with the parents and the adolescents seated together with frequent positive eye contact with the “Difficult” adolescent. These few minutes help the adolescent to assess the clinician’s attitude and get convinced about the professional capacity of the clinician to seek further help in care and counseling. Then the parents are made to be seated outside the consultation chamber when the adolescent is interacted to, with the maintenance of audiovisual privacy and assurance of confidentiality.

This is an exclusive interaction that is aimed at creating a therapeutic alliance, assessing the mental status of the adolescent and screening the adolescent using the HEADSSS tool. Home environment, Eating habits and Education, Activities the adolescent indulges on a routine day, Drug usage, Suicide ideas with or without Depression, Sexual life and Sexuality and Safety of the environment in which the adolescent lives have to be elaborated when using the screening tool. This is done using counseling microskills like usage of open ended questions and free flow communication. The adolescent is also examined for features suggestive of psychiatric morbidity like major depression, bipolar disorders, oppositional defiant disorders, conduct disorders or co morbid conditions like attention deficit hyperkinetic disorder, specific learning disorders, etc.

A clinical probe into the presence of alteration in appetite, alteration in sleep rhythm, recent disinterest in regular activities, disinterest in long term friendships, avoidance of interactions with parents and others, craving for ecstasy like high speed driving, drugs, sex, motor ecstasy like playing table tennis for eight hours a day on a given school day helps the clinician in understanding the status of current functioning of the adolescent.

The outcome of the one to one interaction with the adolescent should be aimed at ruling out

a) Risk to Life: Whether the candidate is planning self harm to the extent of taking away his or her life.

b) Risk to Health: Whether the adolescent is indulging in high health risk activities like using substances, reckless driving that could make him or her accident prone, sexual experimentation with no protection making the adolescent prone to exposure to sexually transmitted diseases, eating behavior abnormalities that could make the adolescent either lose on essential nutrients or make the candidate obese with consumption of extra calories with associated proneness for early onset of life style diseases, etc.

Clinician should approach the adolescent with the aim to rule out clinical depression that could be addressed with appropriate referral to mental health professionals for medication and counseling. Almost all “Difficult” adolescents are
“Unhappy” adolescents unless proved otherwise. The adolescent usually expresses the self directed anger as non cooperation and this deserves compassionate attention from the health care professionals.

During the evaluation process the clinician could assess the coping mechanisms used by the adolescent to handle negative moods as this would reveal the adolescent’s support system and defense mechanisms. It is also essential to find out about the presence of a trust worthy adult who believes in the capacity of the adolescent as this is a major protective factor. Sense of purpose for future, family connectedness, school connectedness and religious beliefs are the other protective factors that help an adolescent to look forward to challenges with ease.

The interaction with the adolescent is usually wound up with the highlighting of assets in the adolescent in a positive tone with reassurance that confidentiality would be maintained and permission is sought to interact with parents in the absence of the adolescent.

**Evaluation with the parents**

The following have to be addressed in addition to listening to the free flow communication by parents.

Family history to rule out the differential diagnosis of conduct Disorders, oppositional defiant disorders, and other associated psychiatric morbidities should be done.

The type of attachment that was prevalent during infancy should be explored as it determines the adolescent’s feelings of security and the strength of the relationships they cherish. A securely attached infant enjoys better relationships for life.

Style of parenting during early childhood and adolescence establishes the communication pattern between children and parents and also frames the personality of the adolescent. An adolescent who is used to “Do as I say, and not do as I do” type of parenting also referred to as authoritarian type of parenting either becomes a submissive person or turns into an aggressive rebellious teenager. A child who is exposed to permissive parenting style reaches adolescence with no sense of direction and drifts away following the tune of the external influences. The recommended “authoritative” parenting style where explanation and reasoning is offered in association with warmth and love, ensures the growth of a self confident, self reliant independent adolescent who becomes an asset not only to the family but to all concerned.

Family environment determines the emotional happiness of the adolescent. Marital disharmony, domestic violence, substance use by adults, use of weapons, use of obscene language by adults are some of the harsh influences on the children and adolescents that could lead to difficult behavioral disposition in the offspring.

Disciplining methods practiced by the family members determine the reaction of the children and adolescents. Use of corporal punishment by parents signifies that aggression could be used in the event of anger and provocation and this principle is imbibed by the adolescent and used when distressed.

Parents’ other activities decide the time spent on monitoring and spying on the activities of adolescent. Parents with more unused time at hand make the adolescent the centre of attention and this is not liked by the adolescent as the need for privacy and independence is very high during middle adolescence. Rebelliousness ensues.

**Assessment**

To know the primary reason we would need a psychologist’s assessment of the intellectual
functioning and educational capacity. Depression, specific learning disorders, ADHD, conduct disorder and oppositional defiant disorder can also be detected with psychologist’s assessment. Psychiatrist’s clinical evaluation is mandatory to identify the exact morbidity.

Screening for thyroid abnormalities and anemia could be done with the help of lab investigations.

**Intervention**

Though the treatment begins from the time the interaction begins with the client and the family, it is necessary that normal adolescent processes and parenting techniques are clarified for the parents to internalize.

It is necessary that a clear distinction has to be made between the perceived abnormalities in the behaviour of the adolescent and the problem behaviors. It is necessary adolescents should not be labeled as “Difficult” adolescents as they are usually not difficult to handle but are unhappy within and are longing for a person who could understand them.

Parents should be encouraged to practice positive parenting techniques in the context of unconditional love. Parents should be encouraged to seek the help of mental health professionals both for themselves and for the adolescents.

Co morbid conditions deserve intervention with remedial education and therapy. Parents should be helped to reestablish family connectedness with love based authoritative parenting style.

**Points to Remember**

- “**Difficult**” adolescents are usually “**Unhappy**” adolescents.
- Parenting style practiced has to be explored.

- **Clinical interview should be done individually with the adolescent to understand the client.**
- **Parents should be given guidelines on positive parenting techniques for better family connectedness.**
- **Help of mental health professionals have to be sought at an appropriate time and the therapy advised should be followed for a good outcome.**

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ACUTE SEVERE ASTHMA

* Gowrishankar NC

Abstract: Acute severe asthma is one of the commonest causes for emergency room visits. Every child presenting with acute severe asthma will need continuous assessment during treatment. Inhaled short acting $\beta_2$ agonists by holding chambers or nebulisers is the drug of choice for bronchodilatation. Corticosteroids reduce the duration, severity and hospitalization rates in acute asthma. This article gives an account of managing acute severe asthma.

Keywords: Acute asthma, Inhaled $\beta_2$ agonists, Corticosteroids, IV magnesium sulphate, Treatment.

Hospitalization rates in acute asthma have remained relatively stable over the last decade, with lower rates in some age groups but higher rates among young children 0–4 years of age. There is some indication that improved recognition of asthma among young children contributes to these rates. (National asthma education and prevention program). But mortality from asthma in children is rare and declining. In preschool children, exacerbations of asthma are far more common in boys than in girls.

With increasing age, this pattern is reversed.

Pathophysiology

In children with asthma, airway mucosa is chronically inflamed due to a complex interaction between T-lymphocytes, neutrophils, eosinophils, epithelial cells and mast cells. The hyper reactivity as a result of this chronic inflammation primes the airways for further airway obstruction for a range of triggers leading to asthma exacerbations clinically. In young children, infection of viral aetiology with mucosal edema predominates and muscular bronchoconstriction is less important; while in older children, and in attacks triggered by allergens, acute bronchospasm due to bronchial smooth muscle contraction is the most important factor. Asthma exacerbations mainly involving inflammatory processes require time to develop and to resolve and symptoms therefore tend to increase and improve relatively slowly. In these children, airway narrowing is mainly due to inflammatory changes along with an associated down-regulation of $\beta$-receptors. Hence the response to $\beta_2$-agonists may be less optimal. In
contrast, allergen induced attacks may develop very rapidly with bronchoconstriction as the dominating pathophysiology, and hence also responding quickly to bronchodilator treatment.

During an acute exacerbation, the chronic inflammation is aggravated by mediators (histamine, leukotrienes and other mediators) released by mast cell degranulation. Vasodilatation and edema in the mucosa, increased mucous secretion and smooth muscle contraction, particularly in the medium sized and small airways occurs decreasing the airway luminal diameter which leads to increased resistance to air flow, particularly towards the end of expiration at low lung volumes. As the airflow limitation becomes severe there is premature airway closure resulting in lung hyperinflation and air trapping.³

**Pulmonary mechanics and gas exchange abnormalities:** Easier air entry during inspiration and airflow obstruction during expiration causes air trapping with each breath leading to lung hyperinflation. Higher end expiratory lung volumes, coupled with bronchospasm lead to increased airway resistance and reduced expiratory flow making expiration an active rather than a passive process which results in higher energy expenditure and substantial increased work of breathing (WOB). Diaphragmatic flattening from hyperinflation causes additional mechanical disadvantages for the muscles of expiration and additional energy expenditure. Both forced expiratory volume and forced vital capacity are decreased as a result of high airway resistance. Total lung volumes are increased because of increased functional residual capacity.

Hyperinflated lungs stretch the pulmonary vasculature, thereby increasing pulmonary vascular resistance. Hypoxemia also induces pulmonary vasoconstriction which is further aggravated by acidosis and lung hyperinflation. These factors increase right ventricular afterload which may compromise right ventricular function. Fluctuations in pleural pressures also produce significant effects on the intrathoracic vessels and right atrial venous return. During the large negative intrathoracic pressure observed during inspiration, left ventricular afterload is increased and systolic blood pressure is decreased. This exaggerated variation in systolic blood pressure (> 10 mm Hg) associated with intrathoracic pressure variation during inspiration is pulsus paradoxus. These changes may result in decreased cardiac output, further increasing both hypoxemia and acidosis.³ During treatment fluid overload caused either by over-hydration or fluid retention due to inappropriate secretion of anti-diuretic hormone, may put the patient further at risk of pulmonary edema.

V/Q mismatch occurs due to increased dead space (airway overdistension) and increased intrapulmonary shunt (atelectasis) which initially manifest as hypoxia and hypocarbia. Atelectasis from small airway obstruction due to mucus plugs causes areas of decreased ventilation but adequate pulmonary blood flow, and the resultant shunt leads to arterial hypoxemia. As the disease severity worsens greater distal airway obstruction causes alveolar distention and increased pulmonary dead space. To compensate for this V/Q mismatch tachypnoea occurs but hypocarbia persists. As the intercostals and diaphragmatic muscles fatigue, the increased minute volume is unable to compensate leading onto progressive hypercarbia, hypoxemia and the end result is respiratory failure.

**Metabolic acidosis:** Airway obstruction, hyperinflation and air trapping also leads to ventilation/perfusion mismatch and hypoxemia. Hypoxemia and the increased work of breathing may result in anaerobic muscle work and accumulation of lactate. The metabolic acidosis
may be further aggravated by dehydration from poor fluid intake. During an asthma attack, metabolic acidosis may initially be compensated for by hyperventilation and respiratory alkalosis, but as respiratory failure develops, increasing arterial CO$_2$ will result in a respiratory acidosis and a further decrease in arterial pH.$^3$

**Clinical features**

The symptoms of acute asthma include worsening cough and wheeze while the signs include supraclavicular, substernal recesions, accessory respiratory muscles use, prolonged expiration in auscultation and cyanosis with worsening of asthma. Severe/rapid developing attack (near fatal and fatal asthma) presents with altered level of consciousness, hypercapnia, acidemia, development of cardiorespiratory arrest. Table.1 shows the ‘Red flag signs’ of asthma.

**Table. 1. Red flag signs**

- Unable to talk or cry
- Cyanosis
- Feeble chest movements
- Absent breath sounds
- Fatigue or exhaustion
- Agitated
- Altered sensorium
- Oxygen saturation < 92%

The ‘pulmonary scoring system’ given by IAP can be used not only to assess the severity of the acute episode but also to evaluate the response to treatment which is very useful practically everywhere (Table.2).

**Management**

Though investigations are not commonly done in acute asthma, xray chest and arterial blood gas are done when the child does not show adequate response to therapy. Xray chest is done when foreign body or pneumonia is suspected or when the child is intubated and ventilated. Arterial blood gas when done should be interpreted only in conjunction with the clinical condition of the child as it keeps changing during progression of acute severe asthma. It can be staged as follows:

- **Stage 1**: Low PCO$_2$ and normal PO$_2$ (Non hypoxemic, hyperventilating child)
- **Stage 2**: Low PCO$_2$ and low PO$_2$ (Hyperventilating and hypoxemic child)
- **Stage 3**: Normal PCO$_2$ and low PO$_2$ (Serious sign of respiratory muscle fatigue)
- **Stage 4**: High PCO$_2$ and low PO$_2$ (Respiratory failure)

The main aims of treatment are reversal of bronchoconstriction, correction of hypoxemia, treatment of airway inflammation with close monitoring for any complications arising due to acute asthma or due to treatment.$^7$ The cornerstones of the management of acute asthma in children are rapid administration of oxygen, inhalations with bronchodilators and systemic corticosteroids. The treatment strategies can be further simplified to

1. **First level** - Oxygen, inhaled $\beta_2$ agonists, corticosteroids, anticholinergics
2. **Next level** - IV Magnesium sulphate, IV $\beta_2$ agonists, aminophylline
3. **Advanced** - Heliox, mechanical ventilation.

**Oxygen**: The first drug to be used in ASA is oxygen. It should be given by face mask or preferably by non-rebreathing mask to maintain a saturation above 95%. The oxygen flow has to
Table 2. Pulmonary score

<table>
<thead>
<tr>
<th>Score</th>
<th>Respiratory rate</th>
<th>Wheezing*</th>
<th>Accessory muscle Sternomastoid activity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;6 yrs</td>
<td>&gt;6 yrs</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>&lt; 30</td>
<td>&lt; 20</td>
<td>None</td>
</tr>
<tr>
<td>1</td>
<td>31–45</td>
<td>21–35</td>
<td>Terminal expiration with stethoscope</td>
</tr>
<tr>
<td>2</td>
<td>46–60</td>
<td>36–50</td>
<td>Entire expiration with stethoscope</td>
</tr>
<tr>
<td>3</td>
<td>&gt; 60</td>
<td>&gt; 50</td>
<td>During inspiration &amp; expiration (without stethoscope)</td>
</tr>
</tbody>
</table>

*If no wheezing due to minimal air exchange, score >3

Score 0–3: mild, 4–6: Moderate, >6: Severe; **Score > 6 should be admitted to a pediatric ICU**

be adjusted based on pulse oximetry. Oxygen decreases the pulmonary vasoconstriction caused by hypoxia but it does not suppress the respiratory drive. An important point to note is that for delivering drugs through nebulisation oxygen has to be used at the rate of 8-10 litres per minute.7

**Short acting β₂ agonists (SABA):** They are the drug of choice in ASA. It has got a rapid onset of action (within minutes) reversing the bronchoconstriction and thereby the airflow obstruction. It produces greater bronchodilatation when compared to methylxanthines and anticholinergics but the duration of action is less. SABA has the advantage that it can be given by inhalation- delivery to the site of action and hence minimal systemic side effects. Under ideal conditions less than 10 % of nebulised drugs reaches the lung. Oxygen at the correct flow rate of 8-10 litres per minute is crucial for getting correct particle size of 0.8 - 3 μm for deposition in the alveoli. Drug delivery can be affected by breathing pattern, tidal volume, nebulizer type and gas flow. SABA is initially given as intermittent therapy. When the child does not respond to the initial therapy of three doses at 20 minutes interval it is given as hourly nebulisation. Continuous nebulisation is found to have a better and prolonged bronchodilation when compared to intermittent therapy as the rebound bronchoconstriction which happens with intermittent therapy is not seen with continuous therapy. The dose of salbutamol to be used for intermittent nebulisation is as follows Salbutamol nebuliser solution (5 mg/ml)- 0.25 ml (< 1 year), 0.5 ml (1-5 year) and 1 ml (>5 year) while the dose for continuous nebulisation it is: 10 mg/hour (5-10 kg) - 15 mg/hour (10-20 kg) - 20 mg/hour (>20 kg). Always give correct dose of salbutamol as errors can occur because nebuliser solution has 5 mg/ml of salbutamol while respules have only 1mg/ml. It is essential that proper dose has to be given while nebulising for the desired effect to be achieved. The side effects of SABA always has to be watched for which includes tremors and tachyarrhythmias. Hypokalemia has to be watched for while nebulising SABA continuously. SABA can be given either by pressurized metered dose inhaler(p MDI) and spacer or nebuliser. The pMDI is good for mild and moderate attacks but for severe attacks nebuliser is the desired apparatus. While using
pMDI dose for a mild attack is 4 puffs every 20-30 minutes and for moderate attacks it is 6-10 puffs (each puff giving 100μg of salbutamol). In gradually developing inflammation there is a possibility of a poor response to SABA due to downregulation of β-receptors and in these conditions adrenaline and ipratropium bromide are beneficial.

**Adrenaline:** Used in condition where other treatment options are unavailable within reasonable time, administered as subcutaneous injection in the dose of 0.01 ml/kg of 1 in 1000 solution. When nebulised adrenaline is used in ASA there was no statistically significant benefit over salbutamol or terbutaline in moderate-severe acute asthma. The use of more than 2 mg of adrenaline per dose was equivalent to 5 mg of salbutamol per dose. While 2 mg or less of adrenaline per dose was inferior to 2.5 or 5 mg of salbutamol per dose. In addition, there were no differences in heart rate and PaO2 between treatments.

**Inhaled anticholinergics:** It is an add on therapy to SABA in acute asthma. It has got a slow onset of action and a lesser degree of bronchodilation but the duration of action is much longer when compared to SABA. When combined with SABA it is found to improve lung function and reduce the hospital admission rates. The dose is 250 μg/dose for less than 20 kg and 500 μg/dose for more than 20 kg. One of the main advantage is that it can be mixed with salbutamol and nebulised.

**Systemic corticosteroids:** Early institution of corticosteroids have been found to reduce the duration, severity and hospitalization in acute asthma. There is no advantage of giving steroids by intravenous route as steroids given by both oral and parenteral route takes 6-12 hours to reach maximum benefit. The dose of oral prednisolone is 1-2 mg/kg while for methylprednisolone it is 1 mg/kg and hydrocortisone 4 mg/kg as IV. There is no evidence to suggest that inhaled steroids are as effective as systemic steroids. No benefit has been found when inhaled steroids are added when systemic steroids are already in use in a particular patient.

**Intravenous fluids:** Used with caution as most often those children with ASA are dehydrated. Care must be taken not to overhydrate them. Overhydration may also lead to pulmonary oedema.

**IV Magnesium sulphate:** Lot of interest is now shown towards magnesium sulphate because of the fact that SABA takes minutes to act while steroids take hours to have its anti-inflammatory effect. So, there is a role for magnesium sulphate in the intervening period between SABA and steroids. Pharmacological action of magnesium is based upon its ability to inhibit the release of calcium from vesicles in the sarcoplasmic reticulum, resulting in bronchial smooth muscle relaxation. The dosage is 25-75 mg/kg given as infusion over 20-30 minutes with normal saline. Even though inhaled magnesium sulphate has a quick onset of action in acute asthma, the desired effect is not achieved because less drug is delivered to the site of action and also some respiratory effort is needed to maximize its effect. It need not be used for mild exacerbations.

**Intravenous β2 agonist (terbutaline):** IV terbutaline is given only if the child is still not improving or showing signs of improvement after inhaled SABA and IV Magnesium sulphate. It is given as a bolus dose 10 μg/kg over 10 minutes followed by maintenance of 0.3-0.5 μg/kg per minute and increased by 0.5 μg/kg/min to a maximum of 5 μg/kg/min. Side effects include tachycardia, agitation, tremors, hypokalemia. When it is given after aminophylline one has to watch for cardiotoxicity.
**IV aminophylline:** The role of IV aminophylline is still changing because of the narrow therapeutic index with a high risk of serious side effects. Its exact mechanism of action in acute asthma is still unclear. The dose is 5 mg/kg in 50 ml of NS followed by 1mg/kg/hour as continuous infusion. It is found to improve lung function (in the first 24 hours) in those already receiving systemic corticosteroids and inhaled SABA. It is used in acute asthma in PICU only if the child is not improving after inhaled SABA, IV Magnesium sulphate, IV SABA (terbutaline). But the child has to be carefully monitored.

**Heliox:** It is a mixture of Helium-oxygen in the ratio of 80:20. Helium lowers gas density and facilitates exhalation thereby decreasing air trapping and reducing work of breathing. This reduces the turbulent airflow and resistance thus improving oxygen and aerosol delivery to the distal lung. But the effects of heliox are transient.

**BIPAP/ Ventilation**

Deterioration to impending respiratory failure - decreasing breath sound, quiet chest, worsening mental status and vitals, inability to speak or cry, arterial pCO2 > 7.5 – 8 kPa are the indications for respiratory support. Ketamine and midazolam / fentanyl intravenous infusion are used for intubation and mechanical ventilation. Paralysis with vecuronium, is done if required but ventilation is the last resort.

**Treatment - simplified**

Mild exacerbation -

- Inhaled SABA - nebulizer or MDI-Spacer.
- If improvement is not satisfactory repeat doses - given every 20–30 minutes - three doses.
- Systemic steroids –if no improvement after one inhalation therapy.

Moderate exacerbation

- Inhaled SABA with ipratropium bromide - three doses - 20–30 minutes.
- O2 supplement if Sa O2 < 92% - room air.
- Systemic steroids if not given earlier.
- Children requiring additional SABA therapy (after 3 inhalations) can be treated intermittently every hourly or switched to continuous therapy.
- IV Mg SO4 if child deteriorates despite treatment with SABA, ipratropium bromide and corticosteroids.

Severe exacerbation

- In addition to the above, intravenous terbutaline infusion or aminophylline infusion.
- If condition worsens despite this - mechanical ventilation.

For stepping down treatment always the principle of “last in first out” is followed with tapering of aminophylline drip/ terbutaline in 24- 48 hours followed by stopping of ipratropium nebulisation in 24-48 hours and then finally reducing salbutamol nebulisation to every 2-4 hourly and then 4-6 hourly as the child shows improvement following treatment. Also steroids should be changed over from parenteral to oral as the child improves. When the child’s pulmonary score comes down to 3 or less, maintains normal saturation in room air, able to eat and sleep well without any difficulty child can be discharged from the hospital.

While managing children with ASA, air leaks - pneumothorax, pneumomediastinum, subcutaneous emphysema, atelectasis are the complications that has to be watched for. Mimics of ASA include central foreign body, vocal cord dysfunction and psychogenic hyperventilation.
Every child who receives treatment for ASA and improves should be counselled about the need to avoid triggers, proper treatment plan for acute asthma at home and the need for regular controller medications. Management of acute asthma will not be complete unless proper guidance is given with regard to long term control of asthma.

**Points to Remember**

- **Extent of wheeze does not always reflect extent of obstruction.**
- **Quiet chest with increasing respiratory efforts denotes imminent respiratory failure.**
- **Regular assessment during treatment is a must.**
- **Give proper dosages of SABA (avoid confusion in dosage between respules and nebuliser solution).**
- **Oxygen should be used only like a drug.**
- **Follow uniform protocol for reducing treatment failures.**

**References**

APPROACH TO
BLEEDING NEONATES

* Durai Arasan G

Abstract: Bleeding problems are often encountered during the neonatal period particularly in intensive care unit. Thrombocytopenia is probably the most common cause but coagulation defects are also observed, and the two problems often co-exist. Although most coagulation problems are acquired, a number of inherited conditions can also present during this time. Diagnosis and management of these conditions is highly dependent on prompt recognition of the bleeding and the initiation of appropriate investigations. While acquired disorders are often present in sick term or preterm infants, many inherited disorders manifest in otherwise healthy infants.

Recognition of the clinical setting in which bleeding occurs is therefore an important clue to the underlying diagnosis. Interpretation of investigation requires careful observation of age dependent features, which are especially important during the early weeks of life.

Keywords: Bleeding, Neonates, PIVKA.

Neonatal hemostatic system

The neonatal hemostatic system is profoundly influenced by age, and concentrations of many haemostatic proteins are dependent on both the gestational and postnatal age of the infant. At birth, concentrations of the vitamin K dependent (FII, FVII, FIX, FX) and contact factors (FXI, FXII) are reduced to about 50% of normal adult values and are further lower in preterm infants. Similarly, concentrations of the naturally occurring anticoagulants, antithrombin, protein C and protein S are low at birth, and as a consequence, both thrombin generation and thrombin inhibition are reduced in the newborn period. Plasminogen is the major protein involved in fibrinolysis, and again this is reduced during the neonatal period, resulting in a relatively hypofibrinolytic state. Despite this apparent functional immaturity of the neonatal hemostatic system, there seems to be relatively lower bleeding problems in a healthy term infant. The hemostatic system matures during the early weeks and months of life, and the concentrations of most haemostatic proteins, both in term and preterm infants, are very close to adult values by 6 months of age.

Normal coagulation mechanisms

It is a process characterised by simultaneous/sequential interactions between blood vessels, platelets and coagulation factors resulting in a stable clot. This is in balance with the natural inhibitors of coagulation factors present in blood like anti-thrombin III, protein C and protein S. Hemostasis can be considered in two phases - primary and secondary.

Primary hemostasis: It is characterised by vessel wall contractions and platelet plug formation in smaller vessels. Following vascular
endothelial disruption, platelets adhere to the exposed collagen with the help of Von Willebrand factor and fibronectin. Following the platelet adhesion substances like ADP, Thromboxane A2 and platelet factor III are released. This leads to primary platelet aggregation which attracts more and more platelets to aggregate and to release more and more ADP and Thromboxane A2 from its dense granules, ultimately expanding the hemostatic plug.

**Secondary hemostasis**: It involves sequential activation of circulating coagulation factors by intrinsic and extrinsic pathways ultimately to form a secondary stable fibrin clot. This controls hemostasis in large vessels.

**Role of Vitamin ‘K’ in neonatal hemostasis**: Vitamin ‘K’ plays a crucial role in neonatal hemostasis. Vitamin K is not required for synthesis of factors but is required for gamma carboxylation of glutamic acid residues of the protein precursors of the factors II, VII, IX and X which are synthesised in liver. These protein precursors are termed as PIVKA (proteins induced in vitamin K absence). It is only after gamma carboxylation that these proteins acquire the ability to chelate calcium and to be subsequently activated during coagulation. This action of vitamin ‘K’ is limited in preterm, as precursor proteins themselves are deficient, often below 30% of adult value as immature liver is incapable of optimal synthesis of many of precursor proteins.

**Evaluation**

A thorough medical history and physical examination should enable the clinician to choose those patients warranting further evaluation. First, it is necessary to confirm whether it is bleeding disorder or not.

**Non pathological conditions**

1. Subconjunctival and retinal hemorrhages are frequent, and petechiae of the skin of the head and neck are common during passage through the birth canal or may be due to venous obstruction.
2. Swallowed maternal blood by newborn
3. Withdrawal bleeding due to hormonal changes
4. Bleeding from an umbilical granuloma
5. Urate crystals which stain the nappy red

These are some of the conditions which manifest with bleeding or mimic bleeding but are not due to bleeding disorder.

**Approach to a bleeding neonate**

In the diagnosis and management it is necessary to take detailed history - antenatal, perinatal, postnatal, family history and to do thorough physical examination along with selected laboratory investigations which play a major role.

**1) History**

a) Maternal history: Presence of underlying maternal systemic diseases like pre-eclampsia, cardiovascular diseases, viral infection, recent drugs taken like aspirin, anticonvulsants like phenobarbitone and phenytoin, anticoagulants should be elicited. History of collagen disorder, past history of ITP in mother should be enquired.

b) Birth history: Detailed birth history, type of delivery, birth asphyxia, trauma, gestational age should be noted. Preterms are more vulnerable for bleeding diathesis compared to term infants. Big cephalhematoma following normal delivery (without prolonged or difficult labour) should lead to suspicion of inherited bleeding disorders.

c) History of administration of vitamin K at birth, use of antibiotics and whether infant is receiving only the breast feeding should be noted to rule out hemorrhagic disease of newborn.
d) Family history: History should include family history of excessive bleeding, spontaneous or after injury should be noted. Proper pedigree charting including both living and dead members will help to know the type of inheritance of the disorder. Enquire history of consanguinity.

X-linked inheritance: Factor VIII, IX deficiency - enquire similar history of bleeding episodes in male siblings, maternal cousins, maternal uncles etc.

Autosomal dominant: Von-Willebrand’s disease, dysfibrinogenemia, hemorrhagic telangiectasia etc.

Autosomal recessive: Other factor deficiencies.

e) What is the time of onset of bleeding?:
Onset of bleeding between day 2-6 is classical of hemorrhagic disease of newborn. Immune mediated thrombocytopenia usually manifests within first 48 hours of age. Early onset bleeding is associated with intrapartum events and maternal status. Late onset manifestations are usually secondary to infections.

2) Physical examination

A rapid assessment is made to evaluate vital functions – heart rate, respiration, blood pressure in addition to colour and general appearance to define whether the newborn is “well” or “sick”. Physical examination should focus upon the site of bleeding – localised or generalised, superficial or deep to identify the type of bleeding – petechiae, purpura, ecchymoses, pallor, oozing from multiple sites, hematemesis, hematochezia, hematuria or pulmonary hemorrhage. Systemic examination is performed to look for congenital anomaly, enlarged liver and spleen, jaundice and stigmata for intrauterine infection. These observations are correlated with history to define the cause of bleeding.

a) Note whether child is sick or well as the causes differ greatly in these two clinical circumstances. Sick babies include those with sepsis, asphyxia, RDS, hypothermia, apneic spells, acidosis, hypoglycemia, seizures, prematurity, hypovolemia, shock, etc. In such babies bleeding is likely to be secondary phenomenon such as DIC, consumption coagulopathy, liver dysfunction, etc.

b) A ‘Healthy’ baby with bleeding indicates hemorrhagic disease of newborn, inherited coagulation factor deficiency, isoimmune thrombocytopenia, platelet function disorders, vascular causes, slipped ligature, etc.

c) If associated hepatosplenomegaly jaundice or choreo-retinitis are present, it may suggest congenital / acquired infections, leukemia, erythroblastosisis fetalis etc.

d) Associated congenital anomalies often give clues to the diagnosis eg. TAR syndrome - absent radius with thrombocytopenia; large hemangioma with DIC suggest Kasabach - Merritt syndrome; presence of eczema with recurrent infections and thrombocytopenia - Wiscott - Aldrich syndrome; Syndactyly with bleeding - Factor V deficiency and Ecchymosis, bruises, purpura with hyperelastic skin - Ehler Danlos syndrome, etc.

e) Site of bleeding

i) Bleeding from umbilicus in a healthy child without any evidence of umbilical sepsis or slipped ligature suspect factor XIII deficiency or hypodysfibrinogenemia.

ii) Bleeding from circumcision or hematoma at injection site in a healthy child-suspect factor deficiency or hemorrhagic disease of newborn.

iii) Bleeding from G.I.T. is probably due to swallowed maternal blood or vit. K deficiency.

iv) In skin bleeds like purpura or petechiae in a healthy child, suspect immune thrombocytopenia and in a sick child-suspect DIC.
**Laboratory tests**

Initial screening tests are

- CBC, differential count, peripheral blood smear and platelet count
- Prothrombin time (PT)
- Partial thromboplastin time (PTT)
- Thrombin time (TT)

Nearly all significant bleeding disorders in neonates can be identified using simple screening tests (Table.1). Notable exceptions are factor XIII deficiency (which requires a specific assay) and platelet function disorders (which require specialized tests and are usually performed after the neonatal period, because of the requirement of more sample volume and data interpretation in neonates). Where an inherited defect is suspected or when coagulation assay derangement is severe, it is extremely important to test both parents for coagulation abnormalities.

Following precautions must be taken while collecting and interpreting blood for coagulation studies.\(^{13}\)

i) One must ensure that there is proper dilution of the infant’s blood with anticoagulant. The standard ratio of 1 part 3.8% trisodium citrate to 9 parts of blood without taking into consideration of the hematocrit (Hct) level would result in excess of citrate. When the Hct is > 55% the anticoagulant must be reduced i.e. the anticoagulant ratio must be based on plasma volume rather than the volume of whole blood. Lab errors in coagulation screening tests are mentioned in Table.2.

ii) Normal values vary from one laboratory to another. The PT is often reported as an international normalised ratio, a standard allowing for the comparison of results between different laboratories.

The complete blood count offers at least two important pieces of information. It allows for rapid determination of the platelet count, either confirming or rejecting a suspected thrombocytopenia. In addition, it may offer clues as to the severity and duration of the patient’s bleeding.

**Subsequent tests**

Based on clinical picture and results of initial screening tests other lab tests may be ordered to reach a conclusive diagnosis. Individual factor assays must be interpreted using appropriate age adjusted ranges. For confirmation of DIC, fibrin degradation products and D dimer assay is performed. In certain circumstances, more specialised techniques may be required to investigate for less common defects including abnormalities of platelet function. A useful screening test for factor XIII deficiency is by demonstrating the infant’s fibrin clot is soluble in 5 M urea. Apt test is used to distinguish between swallowed maternal blood and neonatal gastrointestinal bleed.

**Management**

**Principles of therapy**\(^{13}\)

- Goal should be the well being of infant rather than correcting the laboratory abnormalities.

- Therapy should be focused on treating the underlying disease such as septicemia, infection, shock, hypoxia and acidosis in addition to the supportive therapy and replacing the appropriate blood components.

- Use blood components rather than whole blood wherever possible.

- Use blood products only when they are absolutely necessary.
Table 1. Results of laboratory screening tests in differential diagnosis of the bleeding neonate

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<thead>
<tr>
<th>Platelets</th>
<th>PT</th>
<th>PTT</th>
<th>Likely diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Sick” neonates</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased</td>
<td>Increased</td>
<td>Increased</td>
<td>DIC</td>
</tr>
<tr>
<td>Decreased</td>
<td>Normal</td>
<td>Normal</td>
<td>Platelet consumption (infection, necrotizing enterocolitis, renal vein thrombosis)</td>
</tr>
<tr>
<td>Normal</td>
<td>Increased</td>
<td>Increased</td>
<td>Liver disease</td>
</tr>
<tr>
<td>Normal</td>
<td>Normal/I</td>
<td>Normal</td>
<td>Compromised vascular integrity (associated with hypoxia, prematurity, acidosis, hyperosmolality)</td>
</tr>
<tr>
<td>“Healthy” neonates</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased</td>
<td>Normal</td>
<td>Normal</td>
<td>Immune thrombocytopenia; occult infection or thrombosis</td>
</tr>
<tr>
<td>Normal</td>
<td>Increased</td>
<td>Increased</td>
<td>Hemorrhagic disease of newborn (vitamin K deficiency) or common pathway defect.</td>
</tr>
<tr>
<td>Normal</td>
<td>Normal</td>
<td>Increased</td>
<td>Hereditary intrinsic clotting factor deficiencies.</td>
</tr>
<tr>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>a) Bleeding due to local factors (trauma, anatomic abnormalities)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>b) Qualitative platelet abnormalities (rare)</td>
</tr>
</tbody>
</table>

Table 2. Lab errors in coagulation screening tests 13

<table>
<thead>
<tr>
<th>Error</th>
<th>Cause</th>
</tr>
</thead>
</table>
| Platelet count falsely low | • Platelets adhere to heel after needle prick  
• Errors in dilution (manual technique)  
• Adherance to tube  
• Dilution with EDTA |
| PT & PTT falsely high | • Decreased plasma / citrate ratio (due to either too small a sample or hematocrit > 65 %)  
• Contamination with heparin from indwelling lines  
• Improper storage and transport; PT & PTT falsely low  
• Sample contaminated with tissue thromboplastin from difficult veinpuncture. |
1. Emergency management of bleeding newborns

Infants with bleeding which poses immediate threat to life should receive fresh frozen plasma, vitamin K and PRBC, as needed, as soon as possible after blood has been collected for coagulation studies.

2. Supportive care

Nurse the infant in thermoneutral environment. Ensure oxygenation, perfusion and euglycemia throughout. Correct hypoxia, acidosis, dyselectrolytemia, hypotension and shock. Monitor the vital signs continuously if the infant is sick.

Management of different clinical scenarios in bleeding newborn:

**Vitamin K deficiency bleeding**

Vitamin K deficiency bleeding (VKDB) refers to bleeding that occurs as a consequence of vitamin K deficiency during the first six months of life. Previously known as hemorrhagic disease of the newborn, it was renamed to emphasise that bleeding problems during the neonatal period are not confined to those arising from vitamin K deficiency and that bleeding secondary to vitamin K deficiency may occur beyond the first month of life.¹⁵

VKDB has traditionally been classified as early, classical, and late depending on the timing of the presentation (Table.3).

The diagnosis of vitamin K deficiency may be suspected from the results of coagulation screening where initially there is isolated prolongation of the prothrombin time, followed by prolongation of the APTT, in association with a normal fibrinogen concentration and a normal platelet count.

In an attempt to reduce VKDB, it is recommended that all neonates receive postnatal vitamin K prophylaxis Inj Vit K 1 mg IM. This has become the standard of care since the American Academy of Pediatrics recommended it in 1961.¹⁶

**Management of vitamin K deficiency bleeding**

Any infant suspected of VKDB should receive immediate intravenous vitamin K 1 mg. Intravenous vitamin K can be associated with anaphylactoid reactions and should be administered by slow intravenous injection; if venous access cannot be established it can be given subcutaneously, the intramuscular route being avoided in the presence of a coagulopathy. A single dose of parenteral vitamin K is usually

**Table.3. VKDB has traditionally been classified as early, classical, and late depending on the timing of the presentation**

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Age</th>
<th>Cause</th>
<th>Bleeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early HDN (5% at risk mothers)</td>
<td>1 day</td>
<td>Anticonvulsants, ATT, long term antibiotics</td>
<td>Cephalhæmatoma, ICH, umbilical, GI, etc.,</td>
</tr>
<tr>
<td>Classic HDN (0.01 – 1%)</td>
<td>1-7 days</td>
<td>Idiopathic, maternal drugs</td>
<td>ICH, umbilical ,GI, ENT</td>
</tr>
<tr>
<td>Late HDN(0.02%)</td>
<td>2-24 weeks</td>
<td>Idiopathic, malabsorption, liver disease, breastfeeding</td>
<td>ICH,GI,ENT, skin</td>
</tr>
</tbody>
</table>
sufficient. A rise in coagulation factor levels and function occurs within 2 hours of therapy, with complete correction within 24 hours. In infants who have moderate to severe bleeding, fresh-frozen plasma (FFP) 10–15 ml/kg should be administered in addition to vitamin K.\textsuperscript{17}

\section*{Inherited coagulation deficiencies}

The hemophilias are the commonest inherited bleeding disorders to present in the neonatal period. At least a third of all haemophilia cases occur in the absence of a positive family history and are therefore unsuspected at birth.\textsuperscript{1} Recent cohort studies suggest that 15–33\% of cases may present with bleeding manifestations during the first month of life.\textsuperscript{18}

Umbilical bleeding is relatively uncommon in hemophilia and is typically associated with severe hypofibrinogenaemia and homozygous factor XIII deficiency. Intracranial hemorrhage (ICH) is also seen infrequently in hemophiliacs but is a significant cause of morbidity and mortality in the severe forms of factor VII, factor X and factor XIII deficiency.

\section*{Diagnosis}

Factor VIII levels are within the normal adult range in both term and preterm infants and it is therefore possible to confirm a diagnosis of hemophilia A in the neonatal period regardless of gestational age and severity. The diagnosis of severe and moderate hemophilia B can also be confirmed in the neonatal period. Type 3 von Willebrand disease can be diagnosed in neonates who have essentially a total deficiency of von Willebrand factor. Homozygous deficiencies of factors II, VII, X and XI can be diagnosed in the neonatal period, whereas levels in heterozygotes may overlap with the normal range. Exclusion of Factor XIII deficiency should be carried out in neonates having characteristic bleeding patterns accompanied by normal coagulation screening tests. Factor XII is a special case where there is prolonged aPTT but never cause clinical bleeding.\textsuperscript{17}

\section*{Management}

Where there is clinically significant ongoing haemorrhage and a congenital factor deficiency is suspected but not confirmed, fresh-frozen plasma (10–15 ml/kg) may be administered while the results of laboratory investigations are awaited. Recombinant factor VIII and recombinant factor IX concentrates carry the lowest risk of transmitting viral infection and should therefore be given to neonates with haemophilia A or B who require factor replacement. If recombinant products are not available, a high purity, virus inactivated plasma-derived concentrate should be used. Due to the risks of hyponatremia and water intoxication, desmopressin (DDAVP) should not be used in the treatment of neonatal VWD. A viricidally treated intermediate purity factor VIII concentrate containing the highmolecular-weight multimers of von Willebrand factor remains the treatment of choice.

The treatment of bleeding secondary to other inherited deficiency disorders should be with specific high purity factor concentrates where these products exist (fibrinogen, factor VII, factor XI, factor XIII).\textsuperscript{17}

\section*{Neonatal thrombocytopenia}

Thrombocytopenia (platelets <150 X 10\(^9\)/L) is common in neonates. Several studies report a prevalence of thrombocytopenia of 1-5\%\textsuperscript{10-21} of all newborns and 22-35\% of neonatal intensive care unit admissions and in up to 50\% of neonates who are preterm and sick.\textsuperscript{22,23}

\section*{Causes of neonatal thrombocytopenia}

Conventional lists of the causes of thrombocytopenia include a large number of possible diagnoses, most of which are very rare.
However, in routine clinical practice, it is more useful to be aware of the common causes and patterns of thrombocytopenia. Thrombocytopenia usually presents in one of two clinical patterns which reflect the most common causes: early thrombocytopenia (within 72 hours of birth) and late thrombocytopenia (after 72 hours of life). The principal causes of early and late thrombocytopenia are shown in Table.4.

**Early thrombocytopenia**

The most frequent causes of early thrombocytopenia in preterm infants are conditions resulting in fetal hypoxia. This thrombocytopenia is self-limiting, usually resolving within 10 days. It is seen in infants of mothers with pre-eclampsia, pregnancy-induced hypertension or diabetes; and in IUGR babies. Thrombocytopenia is rarely severe (the platelet count usually remains above 50 X 10^9/L) except in neonates with severe IUGR. It is caused by reduced platelet production secondary to reduced megakaryocytopoiesis. The most important cause of severe early neonatal thrombocytopenia is neonatal alloimmune thrombocytopenia (NAITP).

**Late thrombocytopenia**

The most common and clinically important causes of late thrombocytopenia are sepsis and NEC, which together account for >80% of cases. This form of thrombocytopenia usually develops very rapidly over 1-2 days, is often very severe (platelets <30 X 10^9/L) and takes 1-2 weeks to recover. Sick babies frequently require platelet transfusion. The mechanism is likely to be a combination of increased platelet consumption, often but not always with evidence of DIC, and reduced platelet production.

**Immune neonatal thrombocytopenias**

**Neonatal alloimmune thrombocytopenia (NAITP)**

This is the platelet equivalent of HDN and NAITP affects around 1:1000 pregnancies.

**Table.4. Causes of early and late thrombocytopenia**

<table>
<thead>
<tr>
<th>Early (&lt;72 hours)</th>
<th>Placental insufficiency (PET, IUGR, diabetes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Neonatal alloimmune thrombocytopenia (NAITP)</td>
</tr>
<tr>
<td></td>
<td>Birth asphyxia</td>
</tr>
<tr>
<td></td>
<td>Perinatal infection (group B streptococcus, E. coli)</td>
</tr>
<tr>
<td></td>
<td>Congenital infection (CMV, toxoplasmosis, rubella)</td>
</tr>
<tr>
<td></td>
<td>Maternal autoimmune (ITP, SLE)</td>
</tr>
<tr>
<td></td>
<td>Severe Rh iso immunisation</td>
</tr>
<tr>
<td></td>
<td>Thrombosis (renal vein, aortic)</td>
</tr>
<tr>
<td></td>
<td>Aneuploidy (trisomy - 21, 18, 13)</td>
</tr>
<tr>
<td></td>
<td>Congenital/inherited (TAR, Wiskott Aldrich)</td>
</tr>
<tr>
<td>Late thrombocytopenia</td>
<td>Late onset sepsis and necrotizing enterocolitis</td>
</tr>
<tr>
<td></td>
<td>Congenital infection (CMV, toxoplasmosis, rubella)</td>
</tr>
<tr>
<td></td>
<td>Maternal autoimmune (ITP, SLE)</td>
</tr>
<tr>
<td></td>
<td>Congenital/inherited (TAR, Wiskott-Aldrich)</td>
</tr>
</tbody>
</table>
It is frequently severe and occurs in the first pregnancy in almost 50% of cases. Thrombocytopenia may present prenatally (as early as 20 weeks’ gestation) in which case it is sometimes referred to as fetal alloimmune thrombocytopenia (FAITP) or at birth. The thrombocytopenia results from transplacental passage of maternal platelet-specific antibodies to human platelet antigens (HPA) which the mother lacks but which the fetus inherits from the father. The main clinical problem in NAITP is intracranial hemorrhage; this occurs in 10% of cases, with long-term neurodevelopmental sequelae in 20% of survivors. Affected neonates may present with seizures or other signs of intracranial hemorrhage, with petechiae or bruising, or with an incidental thrombocytopenia. The platelet count is usually <30 \times 10^9/L. The diagnosis of NAITP is made by demonstrating platelet antigen incompatibility between mother and baby (in 80% of cases of NAITP, the mother is HPA-la-negative and the baby is HPA-1a-positive). The recommended management for neonates with NAITP is to transfuse ‘severely affected’ (platelets <30 \times 10^9/L) babies with HPA-compatible platelets. If there is ongoing severe thrombocytopenia and/or hemorrhage despite HPA-compatible platelets, intravenous IgG (total dose 2 g/kg over 2-5 days) is often useful in ameliorating the thrombocytopenia until spontaneous recovery occurs 1-6 weeks after birth. Prenatal management of NAITP remains controversial. The principal options are an invasive approach

Table 5. Guidelines for platelet transfusion (British Committee for Standards in Hematology).

<table>
<thead>
<tr>
<th>Platelet count</th>
<th>Non bleeding neonate</th>
<th>Bleeding neonate</th>
<th>NAIT (Proven or Suspected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 30</td>
<td>Consider transfusion in all patients</td>
<td>Transfuse</td>
<td>Transfuse (with HPA compatible platelets)</td>
</tr>
<tr>
<td>30 – 49</td>
<td>Do not transfuse if clinicall stable Consider if &lt; 1000 gm and &lt; 1 week of age Clinically unstable (fluctuating BP) Previous major bleed Current minor bleed Concurrent coagulopathy Requires surgery or exchange Platelet count falling and likely to fall &lt;30,000</td>
<td>Transfuse</td>
<td>Transfuse (with HPA compatible platelets)</td>
</tr>
<tr>
<td>50 – 99</td>
<td>Do not transfuse</td>
<td>Transfuse</td>
<td>Transfuse (with HPA compatible platelets)</td>
</tr>
<tr>
<td>&gt;99</td>
<td>Do not transfuse</td>
<td>Do not transfuse</td>
<td>Do not transfuse</td>
</tr>
</tbody>
</table>
using fetal blood sampling plus fetal transfusion with HPA-compatible platelets if thrombocytopenia is detected or a non-invasive approach relying on maternal intravenous IgG therapy. Each approach has evidence to support.

Neonatal autoimmune thrombocytopenia

This is secondary to transplacental passage of maternal platelet autoantibodies in maternal idiopathic thrombocytopenic purpura and systemic lupus erythematosus, which affects 1-5 in 10000 pregnancies. Around 10% of infants of affected mothers develop thrombocytopenia. However, the thrombocytopenia is usually mild and intracranial haemorrhage occurs in <1 % of at-risk babies. In affected babies with severe thrombocytopenia, treatment with intravenous IgG is usually effective.

Management of thrombocytopenia.

Evidence-based guidelines for neonatal platelet transfusion therapy are yet to be defined, although consensus guidelines are available (Table.5).

Most recommend platelet transfusion for sick neonates where the platelet count is <50 X 10^9/L; however, for stable, relatively well preterm and term infants, platelet counts of 30-50 X 10^9/L are not associated with an increased risk of haemorrhage, This approach conforms to the current UK guidelines.23 Since platelet underproduction underlies the majority of neonatal thrombocytopenias, recombinant haemopoietic growth factors, including thrombopoietin and interkeukin-11, may be useful future therapies.

Points to Remember

- **The clinical setting in which bleeding occurs is an important clue to the underlying diagnosis.**

- **Majority of the bleeding disorders can be identified using simple screening tests.**

- **The hemophilies are the commonest inherited bleeding disorders to present in the neonatal period.**

- **Severe factor VII, X and XIII deficiency can cause ICH.**

- **A single dose of parenteral vitamin K is all that is needed in VKDB.**

- **Use blood components rather than whole blood wherever possible.**

References


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**CLIPPINGS**


**The diagnostic value of CRP, IL-8, PCT, and sTREM-1 in the detection of bacterial infections in pediatric oncology patients with febrile neutropenia Supportive Care in Cancer, 09/16/2010**

In this study, the authors evaluated C–reactive protein (CRP), interleukin (IL)–8, procalcitonin (PCT), and soluble triggering receptor expressed on myeloid cells–1 (sTREM–1) as predictors for bacterial infection in febrile neutropenia, plus their usefulness in febrile neutropenia during chemotherapy–induced gastrointestinal mucositis. IL–8 is the most useful marker for the early detection of bacterial infections, compared with CRP, PCT, and sTREM–1. IL–8 in combination with clinical parameters or PCT might be even more useful. Gastrointestinal mucositis alone does not affect PCT levels, in contrast to IL–8 levels and therefore, PCT might be more useful for the detection of bacterial infections during mucositis than IL–8.
ADVANCES IN PEDIATRIC CARDIAC EMERGENCIES

* Shreepal A Jain
** Kamal Kiran
** Krishna Kumar R

Abstract: With major advances in the field of pediatric cardiology over the past few decades, outlook for children with congenital heart disease has improved immensely. Congenital heart defects, which were once considered incompatible with life are now regularly managed immediately after birth with good survival. Better understanding of basic cardiovascular physiology behind such emergencies, has led to more focused and etiology oriented management resulting in better outcome. Through this article we aim to present some of the recent advances; both medical and interventional, which are changing the way we approach common pediatric cardiac emergencies.

We will focus on advances in the following common conditions presenting as cardiac emergencies in the pediatric age group:

1. Cyanotic spell
2. Heart failure
3. Neonatal emergencies: duct dependant circulation, transposition
4. Tachyarrhythmias

A. Cyanotic spell

Classically seen in patients with tetralogy of Fallot, these are characterized by episodes of paroxysmal dyspnea with marked cyanosis especially during infancy. These episodes result from drastic reduction of pulmonary blood flow, with an accompanying increase in the right-to-left shunt and a drop in systemic arterial oxygen saturation. The key factors incriminated in the etiology include increased infundibular contractility, peripheral vasodilation and hyperventilation. Kothari hypothesized that
stimulation of right ventricular mechanoreceptors secondary to either increased contractility (due to endogenous catecholamines) or a decrease in right ventricular cavity size (such as with valsalva-like manoeuvre) may trigger a reflex response resulting in hyperventilation, some peripheral vasodilation, without bradycardia and perpetuates the vicious cycle.\(^4\) Using this hypothesis he was able to correlate most of the precipitating events leading to cyanotic spells.

Traditional management included knee chest position, sedation with morphine, correction of acidosis with sodium bicarbonate and use of \(\beta\)-blockers (propranolol), which was thought to relieve the infundibular spasm. But with better understanding of the underlying pathophysiology, volume loading with IV fluids to increase right ventricular preload; increasing systemic vascular resistance along with sedation with the help of ketamine and antagonizing the vasodilatory effects of \(\beta\) adrenergic stimulants with the use of \(\beta\) blockers\(^5\) have become the cornerstone of management of cyanotic spells (Table.1). Recently, dexmedetomidine, a newer sedative agent has been shown to be of use in control of cyanotic spell.\(^6\)

Keeping a low threshold for mechanical ventilation in refractory cases may help by breaking the vicious cycle of hyperpnoea and further cyanosis and would also prevent the adverse outcomes associated with severe cyanotic spells.\(^7\)-\(^9\) Preoperative extra corporeal membrane oxygenation therapy has also been used in patients with severe refractory spells.\(^10\)

Long-standing cyanosis and repeated episodes of cyanotic spells have been shown to correlate with adverse neurodevelopment.\(^11\) With advances in peri operative cardiac care, the trend has been towards early primary correction of TOF in the infancy itself. In fact, many centers are performing primary repair within the first six months of age. But there is significant perioperative morbidity, which includes prolonged mechanical ventilation, increased inotrope requirement and end organ dysfunction.\(^12\)-\(^15\) The alternative to complete corrective surgery includes palliation with a BT shunt, which is especially true for centers from developing countries with limited resources. Advances in cardiac catheterization techniques have helped in performing a potential alternative form of palliation with balloon pulmonary valvotomy (BPV) in carefully selected cases of Tetralogy with severe valvular PS or PDA stenting in those with TOF and pulmonary atresia.\(^16\) Interim palliation with BPV has been regularly performed at our center for selected infants less than 3 months of age with predominantly valvar pulmonic stenosis. Data from our institution showed significant increase in the saturations, pulmonary annulus size and the branch pulmonary artery size after BPV for such cases.\(^17\) Thus, it can be considered as a safe and effective interim palliative procedure for symptomatic young infants with TOF and predominant valvar stenosis. Our experience with palliative PDA stenting in selected cases (> 18 months) with congenital cyanotic heart disease with reduced pulmonary blood flow, with no immediate prospect of definitive surgical correction because of unsuitable anatomy or economic considerations showed statistically significant alternative form of palliation, with no complications during the procedure and also led to significant improvement in oxygen saturations. However PDA stenting can only be offered to the minority of patients with a PDA in association with TOF.

**B. Heart failure**

Heart failure (HF) refers to a clinical state of systemic and pulmonary congestion resulting from inability of the heart to pump as much blood as required for the adequate metabolism of the
body. The clinical picture of HF results from a combination of “relatively low output” and compensatory responses to increase it.

Broadly, heart failure results either from an excessive volume or pressure overload on normal myocardium (left to right shunts, aortic stenosis) or from primary myocardial abnormality (myocarditis, cardiomyopathy). Arrhythmias, pericardial diseases and combination of various factors can also result in HF. The resultant decrease in cardiac output triggers a host of physiological responses aimed at restoring perfusion of the vital organs. Important among these are renal retention of fluid, renin-angiotensin mediated and sympathetic over-activity. Excessive fluid retention increases the cardiac output by increasing the end diastolic volume (preload), but also results in symptoms of pulmonary and systemic congestion. Vasoconstriction (increase in after load) tends to maintain flow to vital organs, but it is disproportionately elevated in patients with HF and increases myocardial work. Similarly, sympathetic overactivity results in increase in contractility, which also increases myocardial requirements. An understanding of the interplay of the four principal determinants of cardiac output - preload, alterload, contractility and heart rate is essential in optimizing the therapy of HF. It is clinically useful to consider HF in different age groups separately.

**Etiology of heart failure by age (Table.2)**

The most common causes of heart failure in infancy are congenital heart defects.

The single most significant advance in management of heart failure in children is the recognition of the fact that a specific cause is identifiable in the vast majority of children with heart failure. Most of these causes are correctable (Table.3) and can be addressed through surgery, catheter interventions or very specific medications. The term cardiomyopathy is now reserved for a small proportion of patients and this diagnosis should only be made after thorough evaluation. A number of conditions can masquerade as cardiomyopathy and these need to be recognized.

Emergency management of heart failure includes a rapid assessment to define the possible etiology and severity assessment directed investigations to confirm the same. But greater emphasis needs to be placed on rapid early stabilization irrespective of the underlying etiologic factor. Conventionally therapy is directed towards:

**I. Medical management**

The initial management involves the usual assessment of the patient’s airway, breathing and circulation (ABCs). This is followed by more specific therapeutic measures.
Table 2. Etiology of heart failure in various age groups.

<table>
<thead>
<tr>
<th>Age</th>
<th>Etiologies</th>
</tr>
</thead>
</table>
| Birth | 1. **Myocardial**  
Asphyxia; transient myocardial ischemia  
Sepsis and or myocarditis  
Hypoglycemia  
Hypocalcemia  
2. **Neonatal hematological abnormalities**  
Anemia or hyper viscosity syndrome  
3. **Neonatal heart rate abnormalities**  
SVT or congenital complete AV block  
4. **Structural abnormalities**  
Volume overload lesions -  
Tricuspid regurgitation  
Pulmonary regurgitation  
Systemic arteriovenous fistula  
Hypoplastic left heart syndrome |
| 1st week | 1. **Structural abnormalities**  
Critical aortic stenosis or pulmonic stenosis  
Coarctation or interrupted aortic arch  
Hypoplastic left-heart syndrome  
TAPVC (with obstruction)  
PDA (preterm infants)  
Duct-dependent lesions with a large PDA  
2. **Heart muscle dysfunction or arrhythmias**  
3. **Renal abnormalities**  
Renal failure or systemic hypertension  
4. **Endocrine disorders**  
Adrenal insufficiency |
| 2nd week | 1. **Shunt defects**  
2 months | Septal defects (ASD, VSD, AVSD)  
Aortopulmonary shunt (PDA, AP window, Truncus)  
2. **Single ventricle**  
3. **Obstructive lesions (see above)**  
4. **Myocardial dysfunction**  
Cardiomyopathy  
Anomalous origin of the left coronary artery  
Metabolic diseases  
5. **Pulmonary disease** |
Table 3. Correctable causes of left ventricular dysfunction in children

<table>
<thead>
<tr>
<th>Condition</th>
<th>Diagnostic clues</th>
<th>Specific treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Congenital cardiovascular disease</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anomalous left coronary artery from pulmonary artery</td>
<td>ECG changes of myocardial infarction typically in LI, aVL, V4-6. 2D and color doppler echocardiography is usually diagnostic</td>
<td>Surgery (Coronary translocation or the Takeyuchi operation)</td>
</tr>
<tr>
<td>Severe coarctation of aorta</td>
<td>Weak femoral pulses, echocardiography</td>
<td>Surgery, balloon angioplasty</td>
</tr>
<tr>
<td>Critical aortic stenosis</td>
<td>Auscultation, echocardiography</td>
<td>Balloon angioplasty</td>
</tr>
<tr>
<td><strong>Acquired cardiovascular diseases</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Takayasu’s arteritis</td>
<td>Careful palpation of all pulses; abdominal bruit, renal ultrasound, renal perfusion scans, doppler evaluation of the thoracic and abdominal aorta, aortography, high baseline renal parameters or extreme elevation following ACE inhibitors</td>
<td>Treatment of active disease (usually indicated by high ESR) may require steroids, immunosuppresants or both. After activity subsides, affected vessels may be treated by balloon angioplasty / stenting or surgery</td>
</tr>
<tr>
<td><strong>Tachyarrhythmias</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any long-standing ectopic atrial tachycardia permanent junctional re-entrant tachycardia, chronic atrial flutter</td>
<td>Disproportionate tachycardia that is not readily explained by the condition of the child. Careful ECG evaluation, esophageal or invasive electrophysiologic testing</td>
<td>Antiarrhythmic drugs Radiofrequency ablation when appropriate</td>
</tr>
<tr>
<td><strong>Metabolic and nutritional causes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypocalcemia</td>
<td>Typically occurs in newborns. Low calcium levels are universal. Adolescents with previously undiagnosed hypoparathyroidism. Chvostek and Trousseau signs may be positive. Prolonged QTc on ECG</td>
<td>Rapid response to restoration of calcium levels</td>
</tr>
<tr>
<td>Infantile beri-beri</td>
<td>1-4 month old infant who has been breast-fed by a thiamine deficient mother. Prominent edema, diarrhea and vomiting.</td>
<td>Rapid response to thiamine given intravenously</td>
</tr>
<tr>
<td>Carnitine deficiency</td>
<td>Hypoglycemia, coma and congestive heart failure in the infant. Ventricular hypertrophy may be seen. High ammonia levels, low serum carnitine and increased urinary excretion of carnitine</td>
<td>Rapid and sustained response to oral carnitine supplementation.</td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>Poorly controlled diabetes, alcoholism, following hyper alimentation nutrition recovery, syndrome and following recovery from severe burns, hyperparathyroidism, hypomagnesaemia, Fanconi syndrome, malabsorption and vitamin D deficiency</td>
<td>Careful restoration of phosphorous levels</td>
</tr>
<tr>
<td>Selenium deficiency</td>
<td>Well described as Keshan disease. Formerly endemic in parts of China. Outside China selenium deficiency has been described in individuals on chronic parenteral nutrition and in those with AIDS. Focal myocyte necrosis on biopsy</td>
<td>Selenium administration can result in partial or complete resolution</td>
</tr>
<tr>
<td>Drug</td>
<td>Mechanism</td>
<td>Dose</td>
</tr>
<tr>
<td>----------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>-----------------------</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Increases cardiac outpatient, BP and improves peripheral perfusion. Characterized by dose dependent pharmacodynamic response, (&lt;10\mu g/kg/min, \beta_1) stimulation by Norepinephrine release, (&gt;10\mu g/kg/min), alpha receptor stimulation.</td>
<td>5-15 (\mu g/kg/min)</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Potent inotropic effect with vasodilation</td>
<td>5-15 (\mu g/kg/min)</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>(\alpha) and (\beta) agonist. Effects are dose-dependent: at low doses, if can cause vasodilation ((\beta_2)-receptors); at high doses, it may produce vasoconstriction ((\alpha)-receptors) of skeletal and vascular smooth muscle, with a subsequent increase of myocardial oxygen consumption.</td>
<td>0.1-1 (\mu g/kg/min)</td>
</tr>
<tr>
<td>Sodium Nitroprusside</td>
<td>An NO donor that induces vascular smooth muscle relaxation and, thus, vasodilation. Nitroprusside seems to cause more systemic arterial (at the arteriolar level) dilation than systemic venous dilation. Therefore, it causes more reduction of after load than preload. Cardiac output increases and aortic and left ventricular impedance are decreased.</td>
<td>Initially 0.5 to 1 (\mu g/kg/min) by continuous I.V. infusion. Usual dose is 3 (\mu g/kg/min); maximum dose is 5 (\mu g/kg/min)</td>
</tr>
<tr>
<td>Phosphodiesterase</td>
<td>Selectively inhibit PDE III, increasing intracellular concentrations of cyclic adenosine monophosphate (cAMP). Increased cAMP in myocardial tissue results in increased intracellular calcium ion concentration and enhanced myocardial contractility. In vascular smooth muscle the increase in cAMP results in smooth muscle relaxation causing vasodilation.</td>
<td>Loading dose = 50 (\mu g/kg) administered over 15 minutes Maintenance dose : 0.35-0.75 (\mu g/kg/min)</td>
</tr>
<tr>
<td>Milrinone</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
1. Correction of low cardiac output state

Use of inotropes has been a well-established modality for stabilization of patients in heart failure irrespective of the underlying etiology. Inotropes other than digoxin are used for short-term support of circulation or to tide over the crisis (Table 4). Commonly used inotropes include

Newer agents

**Levosimendan**: It is a pyridazinone-dinitrate that belongs to a new class of drugs, the calcium sensitizers. In contrast to other inotropic agents, levosimendan is deemed to improve myocardial contractility without increasing intracellular calcium. It acts by binding to myocardial troponin C, causing a configuration change in tropomyosin that exposes actin and myosin elements, allowing for a more effective contraction. It offers the advantage of increasing systolic force without compromising coronary perfusion. Moreover, levosimendan opens adenosine triphosphate (ATP)-sensitive vascular potassium channels, causing vascular hyperpolarization and relaxation, coronary artery dilation and myocyte mitochondrial activation. Levosimendan is used in the treatment of decompensated cardiac failure and as an elective drug in patients with perioperative risk of ventricular failure. It has also been used in the rescue therapy of patients who have difficulty weaning from cardiopulmonary bypass or from mechanical circulatory support. There are also reports documenting its favorable effect in reducing pulmonary vascular resistance and endothelin-1 levels and in improving right ventricular failure.\(^ \text{19,20} \)

**Calcium infusion as an inotrope**: Inotropic state of left ventricle can be altered by alteration of circulatory calcium level. Calcium supplementation plays an essential role in augmenting left ventricular function in pediatric patients. Calcium, either in the form of gluconate or chloride salt have a significant inotropic effect in the ionised form but can cause increased resistance in coronary and systemic vascular beds leading to decrease in oxygen supply to the myocardium and increased left ventricular afterload. The usual recommended dose of calcium chloride in low cardiac output syndrome (LCOS) in infants and children is 10-20 mg/kg slowly into a central vein. The dose of calcium gluconate is three times more compared to calcium chloride.

2. Correction of congestive state

This is achieved with the use of diuretics (to reduce pulmonary or systemic congestion), and after load reducing agents (ACE inhibitors).

Diuretics afford quick relief in pulmonary and systemic congestion. 1-2 mg/kg of furosemide is the agent of choice. Secondary hyperaldosteronism does occur in infants with CHF and addition of spironolactone 1 mg/kg single dose to other diuretics conserves potassium.

3. Role of mechanical ventilation

A lower threshold for early elective mechanical ventilation especially for cases not responding to above line of therapy needs to be emphasized. Mechanical ventilation helps by reducing the work of breathing and thus translating into reduction of myocardial oxygen demand. Hypoventilation and low FiO\(_2\) help in reducing the amount of left to right shunt by increasing the pulmonary artery pressures.\(^ \text{21} \)

4. Role of neurohormonal modulation

ACE inhibitors: Several studies in infants and children with left-to-right shunts or dilated cardiomyopathy have demonstrated improved hemodynamics or significant clinical improvement after the introduction of an ACE inhibitor.\(^ \text{22-27} \) Three studies demonstrated a decrease in the pulmonary-systemic blood flow ratio and the left-to-right shunt following
the administration of an ACE inhibitor. Infants with a left-to-right shunt and increased systemic vascular resistance showed the greatest response. Oliguria, acute renal failure and hypotension are the most common side effects. Renal function should be monitored carefully when initiating therapy with ACE inhibitors. Hypotension is more frequent in patients who have elevated plasma renin activity and in patients who are volume depleted; therapy in these patients should be initiated with low doses. Captopril is the most commonly used ACEI. Dosage involves a test dose of 0.1 mg/kg with monitoring of blood pressure, followed by gradual increment in doses up to 1 mg/kg/dose every 8 hourly.

**β-blockers:** Like ACE inhibitors, β-blockers interfere with the endogenous neurohormonal system. ACE inhibitors interrupt the renin-angiotensin system, whereas β-blockers inhibit the effects of the sympathetic nervous system. Although the use of β-blockers in CHF may seem counterintuitive, low doses titrated slowly in adults with systolic dysfunction decrease symptoms of CHF and decrease both the risk of mortality and the combined risks of hospitalization or death. The rationale perhaps relates to downgrading of β-receptors due to chronic catecholamines stimulation. It is important to note that β-blockers are only used in stable patients. The therapy is best undertaken in hospital as careful monitoring is required. Carvedilol, a nonselective β-blocker with alfa-1 blocking and anti-oxidative properties has proven to be beneficial in infants with dilated cardiomyopathy and there is significant improvement in their functional status.

4. Correction of precipitating factors

Almost always, the worsening in clinical state of a patient with CHF can be traced to a precipitating event, the treatment of which leads to significant improvement. The checklist includes intercurrent infections, anemia, electrolyte imbalances, rheumatic activity, infective endocarditis, arrhythmia, pulmonary embolism, drug interactions, drug toxicity or non-compliance and other system disturbances.

5. Role of prostaglandin E₁

Neonates with transposition of great arteries, coarctation of aorta, aortic stenosis in failure or hypoplastic left heart syndrome, etc., improve remarkably with (PGE₁). The therapy is initiated at 0.05 μg/kg/min and may be gradually raised or lowered depending on the response. Apnea may occur during the infusion and ventilatory support should be available. Irritability, seizures, hypotension and hyperpyrexia are rare.

6. Miscellaneous

Extra corporeal membrane oxygenation, left ventricular assist device (LVAD) and the intra aortic balloon pump (IABP) have also found a place in the management of pediatric patients with heart failure. There is a 74% survival rate and the long-term outcome has been excellent in most cases. But the high cost of equipments and availability at select centers limits the use of these modalities for regular management of heart failure.

II. Catheter based management

With the recent advancements in pediatric interventional cardiology, it has become possible to treat certain congenital cardiac conditions without waiting for complete resolution of heart failure. Classic examples include transcatheter coil or device closure of PDA, especially in preterm newborns stuck on mechanical ventilation and balloon dilatation of critical aortic stenosis or severe coarctation of aorta presenting with severe heart failure. Balloon dilatation of critical aortic stenosis has now been achieved
even in smaller neonates through the umbilical artery route with the availability of smaller sized catheters.\textsuperscript{33}

### III. Emergency surgical management

Advances in cardiac surgical techniques and preoperative cardiac critical care have aided early primary repair of many congenital heart defects irrespective of the weight and age and with underlying refractory heart failure or respiratory tract infections. Classic examples include closure of heart defects especially large PDA in small newborns not amenable to transcatheter closure. ALCAPA repair at the time of diagnosis, repair or replacement for regurgitant valvular lesions, etc. Surgically treating critically ill cases with large VSD and respiratory infection has shown a success rate of 91.6% amongst the operated cases.\textsuperscript{34}

### C. Neonatal cardiac emergencies

These constitute a specific group of conditions with certain common presentations (Table 5). The three common presentations include:

1. Neonate with cyanosis
2. Neonate with cardiovascular collapse
3. Neonate with heart failure

### I. Medical management

A relatively well child presenting dramatically around 3 days to 1 week of life with either cyanosis or cardiovascular collapse strongly suggests duct dependent lesion. Oxygen may precipitate duct closure and therefore should be used with caution in duct dependent lesions. It may also worsen left to right shunts by decreasing pulmonary vascular resistance. Pulmonary blood flow can increase at the cost of systemic blood flow. Thus tailoring the FiO\textsubscript{2} to achieve a SpO\textsubscript{2} of 80-85% may be adequate to balance systemic and pulmonary circulations in patients with a single ventricle physiology. Cyanosis not associated with acidosis need not be corrected. PGE\textsubscript{1} infusion can be life saving. PGE\textsubscript{1} sensitive lesions may present with cyanosis and murmur or mild/no cyanosis with abnormal pulses. Clinical suspicion of obstructed TAPVC should be high, in situations with worsening of cyanosis after initiation of PGE\textsubscript{1} infusion. Dosage needs to be titrated according to the saturation.

Ideal ABG target should be to achieve a PO\textsubscript{2} and PCO\textsubscript{2} level of about 40 with a pH of 7.4. Adverse effects of PGE\textsubscript{1} include apnea, tachycardia, bradycardia, fever, NEC, seizures, thrombocytopenia. Major limiting factor for initiation of treatment is its high cost.

### II. Catheter based interventions

**Balloon atrial septostomy:** Neonates with transposition of the great vessels with intact ventricular septum and an inadequate sized foramen ovale have severe cyanosis due to poor intercirculatory mixing. Balloon atrial septostomy helps by improving this admixture between the systemic and the pulmonary venous blood to achieve a reasonable saturation and improve the hemodynamics till a time that an arterial switch operation can be performed. The advantage of this procedure is that it can be even performed on the bedside with the help of echocardiographic guidance especially in sick newborns.

**Balloon aortic valvotomy:** Neonates with critical AS suffer from low cardiac output and shock secondary to poor left ventricular function and/or mitral insufficiency. Outcome is usually fatal in most of these patients within the first weeks of life with medical treatment alone. The use of percutaneous balloon aortic valvuloplasty was first introduced in 1984 and has become the first-line treatment for critical aortic valve stenosis in neonates.\textsuperscript{35-38} Most studies
Table 5. Cardiac emergencies in the newborn

<table>
<thead>
<tr>
<th>Physiologic category</th>
<th>Conditions</th>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duct dependent</td>
<td>Hypoplastic left heart syndrome, critical coarctation, interruption of aortic arch, critical aortic stenosis</td>
<td>Heart failure</td>
</tr>
<tr>
<td>systemic blood flow*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duct dependent</td>
<td>Pulmonary atresia, critical pulmonary stenosis, Ebstein anomaly</td>
<td>Cyanosis, hypoxia,</td>
</tr>
<tr>
<td>pulmonary blood</td>
<td></td>
<td></td>
</tr>
<tr>
<td>flow*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obstruction of</td>
<td>Obstructed total anomalous pulmonary venous return, mitral atresia with a restrictive patent foramen ovale</td>
<td>Cyanosis, hypoxia, heart failure</td>
</tr>
<tr>
<td>pulmonary venous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>return</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parallel circulation</td>
<td>D transposition with intact ventricular septum</td>
<td>Cyanosis, hypoxia</td>
</tr>
<tr>
<td>with poor mixing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valve regurgitation</td>
<td>Congenital mitral valve regurgitation</td>
<td>Heart failure</td>
</tr>
<tr>
<td>High-output state</td>
<td>AV malformations (usually intracranial)</td>
<td>Heart failure</td>
</tr>
<tr>
<td>Myocardial</td>
<td>Myocardial diseases (inflammatory and metabolic)</td>
<td>Heart failure</td>
</tr>
<tr>
<td>dysfunction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tachyarrhythmia</td>
<td>Atrial flutter, neonatal atrio-ventricular reentrant tachycardias, ectopic atrial tachycardia</td>
<td>Tachycardia, heart failure</td>
</tr>
<tr>
<td>Bradyarrhythmia</td>
<td>Complete heart block</td>
<td>Bradycardia, heart failure</td>
</tr>
</tbody>
</table>

* Some of the duct dependent conditions (critical PS, AS, coarctation) manifest with varying severity. The most severe forms manifest early (in the first few days) with absolute dependence on the duct for survival. Others may manifest later in the neonatal period (first few weeks) with heart failure or cyanosis and may not be strictly “duct dependent”.

show that at 5-year follow-up, around 85% of patients are alive and 60% remain free of re-intervention.  

Balloon dilatation/Stenting of coarctation of aorta: Newborns with coarctation of aorta present in a critical state with left ventricular dysfunction, shock, pulmonary hypertension and end organ dysfunction. Emergency surgical correction may not always be feasible at short notice and may be associated with significant mortality and morbidity. Percutaneous balloon dilatation or stenting of coarctation of aorta achieves immediate relief of heart failure by reducing left ventricular pressure overload and allows postponing of surgery to a more appropriate time. The drawbacks of balloon dilatation are the high incidence of restenosis due to recoil and unfavorable scarring and aneurysm formation due to damage to the media of the vessel. Placement of a stent is said to interfere with the growth of the vessel. In spite of this,
these procedures are life saving measures and may be considered for interim palliation

**PDA stenting for duct dependant pulmonary circulation:** Conventional management of children with duct dependent pulmonary circulation (DDPC) includes prostaglandin infusion and/or surgically created aorto pulmonary shunts. Surgical shunts have significant mortality and morbidity. Maintaining the duct patency by stenting has been shown to be feasible with low complication rates.\(^{16}\)

**II. Surgical approach**

Emergency surgical intervention is indicated for certain congenital cardiac defects like total anomalous pulmonary venous connection with obstruction after basic stabilization (mechanical ventilation, inotropes, diuretics, correction of acidosis). This has shown to reduce the post-operative mortality.\(^{42}\)

**D. Tachyarrhythmias**

Confronting a new patient with a sustained tachycardia in the emergency room can be an intimidating experience for the uninitiated. However, prior knowledge of the finite list of diagnostic possibilities and familiarity with a standard sequence of therapy, can easily transform these encounters into an intellectually stimulating exercise with an optimal patient outcome. It is therefore imperative that the attending physician be well versed with the approach to tachyarrhythmias, which results in a rapid treatment response that is appropriately tailored to the underlying mechanism and severity of the arrhythmia.

Though there are several mechanisms for tachycardia, the clinician must be able to narrow the field to the one or two most likely possibilities in the acute setting in order to guide acute therapy and design a long-term management plan. This can be accomplished with reasonable accuracy on the basis of standard ECG recordings. A full 12-lead ECG should be obtained on all patients with a tachyarrhythmia whenever possible. A single lead rhythm strip or the monitor is a very poor substitute, because it lacks definition of the QRS and P-wave axis that can be the key to pinpointing the correct mechanism. In addition, the P-wave may not always be visible on a single given lead, nor can the maximum width of QRS complex be determined with certainty.

The only clinical situation where a full ECG should be deferred is when the suspected tachyarrhythmia is associated with hemodynamic compromise. This situation is seldom seen with narrow QRS tachycardia. However, selected children with wide QRS tachycardia may have serious hemodynamic compromise. Examples include, ventricular tachycardia, ventricular fibrillation and atrial fibrillation in the presence of pre-excitation. In all other settings, a 12 lead ECG should be obtained in the presenting tachycardia, after which a rhythm strip should be run during each therapeutic maneuver (especially while giving adenosine), followed by a repeat ECG after restoration of the patient’s normal rhythm.

**Emergency management**

The initial point to address from the ECG is whether the QRS complex in tachycardia is narrow or wide. This step is intended to provide a gross discrimination between supraventricular tachycardia (SVT) and ventricular tachycardia (VT). The QRS complex is labeled narrow only if its duration falls within the normal range for age in all\(^{43}\) 12 ECG leads. Though SVT is more common in children, a wide QRS complex suggestive of VT requires urgent evaluation and conversion.

An algorithmic approach to management of tachyarrhythmias is shown in Fig.1. This basic
approach is for the emergency management of arrhythmias without taking into consideration the underlying mechanism for tachycardia.

With advances in cardiac electrophysiology, it has become imperative to diagnose the underlying mechanism of the tachyarrhythmia to guide further management strategy. For eg. SVT due to accessory pathway is amenable to treatment with radiofrequency catheter ablation with high success rates. Certain mechanisms like ectopic atrial tachycardia and permanent junctional reciprocating tachycardia are well known to be associated with a pattern of heart failure known as tachycardia induced cardiomyopathy or tachycardiomyopathy. Heart failure and cardiomyopathy may improve completely or partially after control of such arrhythmias.

In certain SVT with very high rate, the underlying mechanism may be uncovered only after administration of I.V. adenosine; thus highlighting the importance of recording an ECG while giving adenosine.

Concept of tachycardiomyopathy

Coined by Gallagher JJ, the term tachycardiomyopathy refers to impairment in left ventricular function secondary to chronic tachycardia, which is partially or completely reversible after normalization of heart rate and/or rhythm irregularity.

Fenelon, et al further classified tachycardiomyopathy into two categories, namely “pure type” and “impure type”. In the former, chronic tachycardia causes LV dysfunction in a normal heart and completely recovers after termination of the tachycardia. In the latter, such a condition occurs in patients with structural heart diseases and the cardiac dysfunction may only recover incompletely after termination of the tachycardia. The incomplete recovery of LV function might be the result of long term tachycardia inducing irreversible myocardial injury.

Tachycardiomyopathy is induced by various supraventricular and ventricular arrhythmias. Ectopic atrial tachycardia (EAT) and permanent junctional reciprocating tachycardia (PJRT) are the two most common arrhythmias associated with tachycardiomyopathy in children. This is due to their incessant nature and refractoriness to anti-arrhythmic drugs.

Table 6. Key advances in management of tachyarrhythmias in children

1. Most tachyarrhythmias in children in the absence of structural heart diseases are well tolerated.
2. An episode of tachyarrhythmia should be looked upon as an opportunity to identify the mechanism (re-entrant or automatic) and arrive at a specific diagnosis.
3. Unless the tachyarrhythmia is associated with hemodynamic compromise, it is imperative to record a baseline ECG and document the response to adenosine or other forms of treatment.
4. Persistent tachyarrhythmia can manifest as heart failure and masquerade as cardiomyopathy.
**Fig 1. Approach to management of tachyarrhythmias in children**

- **Tachycardia with pulses and poor perfusion**
  - Assess and support ABCs as needed
  - Give oxygen
  - Attach monitor / defibrillator

  **Symptoms**

  **Evaluate QRS duration**

  **Narrow QRS (<0.08 sec)**
  - Evaluate rhythm with 12-lead ECG or monitor

  - **Probable sinus tachycardia**
    - Compatible history consistent with known cause
    - P waves present/normal
    - Variable R-R, constant P-R
    - Infants: rate usually <220 bpm
    - Children: rate usually <180 bpm
  
  - **Probable supraventricular tachycardia**
    - Compatible history (vague, nonspecific)
    - P waves absent / abnormal HR not variable
    - History of abrupt rate changes
    - Infants: rate usually ≥220 bpm
    - Children: rate usually ≥180

  - Search for and treat cause

  - Consider vagal maneuvers** (No delays)

  - If IV access readily available:
    - Give adenosine 0.1 mg/kg
    - (maximum first dose 6 mg) by rapid bolus
    - May double first dose and give once
    - (Maximum second dose 12 mg)
  
  - **OR**
    - **Synchronized cardioversion**:
      - 0.5 to 1 J/kg; if not effective,
      - Increase to 2 J/kg
      - Sedate if possible but don’t delay cardioversion

  - **Expert consultation advised**
    - Amiodarone: 5 mg/kg IV over 20 to 60 mts
    - **OR**
      - Procainamide: 15 mg/kg IV Over 30 to 60 minutes
      - Do not routinely administer amiodarone and procainamide together.

**During evaluation**
- Secure, verify airway and vascular access, when possible
- Consider expert consultation
- Prepare for cardioversion

**Treat possible contributing factors**
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-hyperkalemia
- Hypoglycemia
- Hypothermia
- Toxins
- Cardiac tamponade
- Tension pneumothorax
- Thrombosis (coronary or pulmonary)
- Trauma (hypovolemia)

*Vagal maneuvers: In infants or young children, use a bag filled with ice and cold water over the face for 15 to 30 seconds or rectal stimulation with a thermometer. In older children, encourage bearing down (Valsalva maneuver) for 15 to 20 seconds. Carotid massage and orbital pressure should not be performed in children.*
Radiofrequency ablation

In a very short period of time, there has been a revolution in the treatment of arrhythmias in both adults and children. This revolution in arrhythmia therapeutics has been facilitated largely by refinement of catheter ablation techniques, which allow arrhythmias to be diagnosed and “cured” in one session in the catheterization laboratory. In many circumstances drug therapy has been pushed down as a second line alternative therapy.

Catheter ablation is a procedure during which a patient’s cardiac conduction system is tested for its ability to sustain an arrhythmia. Once the arrhythmia is diagnosed, its substrate is electrically identified and anatomically localized. Subsequently, a critical component of the arrhythmia’s substrate is destroyed using energy delivered through the tip of a catheter. Currently, the energy most commonly used is radiofrequency energy. The electrophysiology study, which is performed in conjunction with this technique, identifies and localizes the arrhythmogenic substrate of a pathologic tachycardia. This substrate may be an accessory connection responsible for atrio-ventricular (AV) reciprocating SVT or Wolff-Parkinson-White (WPW) syndrome; an irritable focus in the atrium or ventricle responsible for an ectopic atrial tachycardia (EAT) or ventricular tachycardia (VT); a scar in the atrium or ventricle responsible forming a component of a re-entrant tachycardia circuit (atrial flutter or VT); or an area of slow AV node conduction that is critical for the perpetuation of the AV node re-entry tachycardia.

Almost all common pediatric arrhythmias can be treated with catheter ablation. The most commonly treated substrate is the accessory atrioventricular pathway (AP). The overall success rate regardless of the pathway location, the presence of multiple pathways, catheter approach, or patient age, can be as high as 98% and typically range 85% to 95%.47-49

On the basis of data from 1999 Pediatric RF Ablation Registry, AVNRT accounts for approximately 24% of ablations performed in children, with success rates of up to 97%.50,51

Major complications are minimal, but include complete AV block when ablating septal pathways,47,49 cardiac perforation and tamponade,50,51 inadvertent coronary damage,40 and vascular or embolic injury.51,52

Conclusion

Pediatric cardiac emergencies require very specific treatment in the emergency room setting. Considering the possibility of a cardiac problem as the cause for the presenting symptoms is the initial step in successful management. With advancements in the understanding of basic cardiovascular physiology behind such emergencies, their handling has become more focused and etiology oriented; resulting in better outcome. Cyanotic spells can now be readily managed with various medications and if required urgent surgical intervention. Management of cardiac failure is now more focused on unveiling the underlying cause along with modification of derangements of the various compensatory mechanisms that have taken place. Newborn cardiac emergencies are being regularly handled with the availability of prostaglandin E1 and with the advancements in transcatheter procedures to tide over the crisis. Approach to arrhythmias requires more focus on finding the underlying mechanism to provide a more specific pharmacological or interventional therapy.

To conclude, common pediatric cardiac emergencies are now managed more efficiently with better outcome seen in the worst case scenarios.
Points to Remember

- **Cyanotic spell:** IV fluids, low threshold for mechanical ventilation, early surgery
- **Heart failure:** Emphasis on identifying underlying etiology
- **Neonatal cardiac emergencies:** Early detection, role of prostaglandin E1 and transcatheter procedures.
- **Tachyarrhythmias:** Importance of 12 lead ECG at presentation and rhythm strip during therapeutic maneuvers.

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EVIDENCE BASED CHILD
HEALTH IN INDIA

* Meenu Singh

Abstract: A process of turning clinical problems into questions, then systematically locating, appraising and using clinical research findings as the basis for clinical decisions. Medical practice based on conclusions thus derived can be called as evidence based clinical practice. Simply following journal articles is not evidence based medicine. The research findings must be appraised and if multiple be synthesized to be put into proper perspective. Evidence gets graded depending on the quality of contributing studies and trials. Only grade one evidence is recommended to be included into guidelines. However when very high quality studies are not available evidence must be put into practice after a consensus approach.

Keywords: Evidence, Systematic review, Metaanalyses, Guidelines.

Abbreviations: Evidence based medicine, Randomized controlled trial

The need

Evidence based medicine is a term coined at McMaster University (Canada) in the 1980s. It is defined as a process of turning clinical problems into questions, then systematically locating, appraising and using clinical research findings as the basis for clinical decisions. Medical practice based on conclusions thus derived, can be called as evidence based clinical practice.

Achieving child health is a challenge in India as in other developing countries. However, there is evidence that policies made on the basis of available evidence can help achieve the goals for child health as laid down in the millennium development goals which have to be achieved by year 2015.

The methods

Evidence based medicine involves harnessing information for everyday clinical practice. Clinicians in the practice of medicine every day come across several problems which require discrete decisions. In today’s world of rapidly changing science it is essential to make all clinical decisions in light of the available evidence. However, it is important to develop the ability to discern what the available evidence is and its applicability in the given clinical situation. A large number of clinical observations and experiments are published every year. Information today is available not only in books but it is hovering all over in cyber space. More than two million articles are published annually in the biomedical literature. One generally gets about 100-500 articles per year about every relevant topic. One has to decide which ones to read. An ex-editor of medical journal – Lancet laments. “It is generally surprising to the lay that upto 99% of published
articles belong to the bin and certainly not be used to inform practice.”

A large amount of money is spent globally to study the effect of therapies. To develop a clinical approach in light of the published observations and to carry out research efforts which have not already been carried out one requires:

1. An access to all available literature needing state of the art library support. This job has been made partly simpler by the available electronic data bases like Medline, Index Medicus, Embase, Excerpta Medica and Current Contents.

2. An education in interpreting the findings of the previous research in light of its applicability in the given context. This involves systematic review of the available literature. Recently, application of the technique of meta-analyses has been able to provide answers in several clinical problems where limitless research had been going on.

3. After an evidence has been produced and interpreted it is important to disseminate it to the user i.e. the clinician. This requires an active group of individuals in a centralized facility who are able to supply this information to the doctors at the bedside. This dissemination of information from bench to bed side has been made easy again with help of electronic communication where on line access may be provided to the users of the electronic media. Several professional bodies are developing evidence based guidelines to improve the clinical practice.

**Evidence in India for India by India**

A concern has been expressed that research findings appraised and applied in other countries may not be applicable to our own country that has her own diversity and variability. A large body of scientific literature is produced in our country. It is a pity that not all is published or brought into the public domain. A recent effort to start websites of all scientific institutes containing archived research evidence produced by them and networking them with each other will go a long way in bringing to light the scientific evidence produced in India. A large volume of thesis is conducted by all medical institutions. However it has aptly been called fit for keeping in the fiction section of the libraries because the meager amount of resources that are spent on these make the students cook up their findings and lack of supervising time for the faculty makes all this data enter the literature. There is a need to introduce quality control at all levels of research. From planning to execution an attitude of utmost sincerity and relevance to the national needs is required.

Medical literature is vast. It is a stupendous task to review all the available evidence before making a decision in each and every clinical problem. However, this exercise has become essential in this age of specialized medicine and increased consumer awareness. Today, a patient has an access to available medical literature on his or her computer through internet or other agencies and a doctor may find it increasingly difficult to convince such a person unless he himself is well informed and knows the value of the available information. Such situations may not be very common in India at present but with ongoing revolution in the communication technology which is moving at a fast pace one can foresee such an implosion before it is too long.

Evidence based medicine has been practiced in many countries in different guises. There has been evidence to its clinical effectiveness. Short term trials have shown better and more
informed clinical decisions following even brief training in clinical appraisal. However this approach is very difficult to evaluate. It is a process for solving problems, and monitoring all possible outcomes is a formidable task. However it has been found that the graduates of a medical school that teaches lifelong, self directed evidence based medicine are still up to date as long as 15 years after medical graduation. Systematic reviews of computer based clinical decision support systems and implementing clinical practice guidelines have shown that, at least some of the time and in certain circumstances, it is possible to change what the clinicians do.

One of the international agencies which has taken up the task of building up evidence based medicine as a scientific discipline is the Cochrane Collaboration. This collaboration came into existence nine years ago and has grown at a very rapid pace. It is an international network named after the physician epidemiologist, Archie Cochrane. The collaboration was founded at a meeting of about 80 people from several countries in Oxford, England in the fall of 1993. The goals of the collaboration are to produce high quality systematic reviews (where possible meta-analyses) of trials of every sort of health interventions, to ensure that these are subjected to very high quality peer reviews, and when necessary, updating, and to disseminate these systematic reviews electronically both on CD-ROM and internet. The collaboration has already brought out Cochrane library which contains systematic information on research in number of research topics. The review groups undertaking a Cochrane are called Cochrane review groups and have the individuals representing these review groups have a lifelong commitment to carry out and update these systematic reviews and meta-analyses. This is in contrast to the reviews published in print journals which become outdated very soon.

This collaboration has the sort of promise that led David Naylor to write “the Cochrane Collaboration is an enterprise that rivals The Human Genome Project in its potential for the modern medicine”. At present there are Cochrane Centres in more than a dozen different countries. South Asian Cochrane Network has recently been upgraded to be a centre with coordinating node at CMC Vellore, Chandigarh, Delhi, Mumbai, Karachi and Dhaka are the other collaborating sites (www.SACN.org). Another giant leap was made with the procurement of the national license for the Cochrane Library of India by the ICMR which increased the access to evidence by all the academic and practicing pediatricians.

There is a need to spread the message of evidence based medicine to all professional bodies and agencies concerned with the health of children. The Indian Academy of Pediatrics in collaboration with Royal College of Pediatrics and Child Health has unrolled plan for teaching of evidence based child health to pediatricians in India with the help of David Baum International foundation. Three initial courses have already taken place in the Advanced Pediatrics Centre, PGIMER, Chandigarh since 2005. All other bodies can undertake projects for framing national guidelines for management of common disorders as has been done by IAP for some diseases. Indian Pediatrics also added a section on appraisal of local evidence in light of the one published in Archives diseases of Child Health under a section exclusively devoted to this as Eureca.

**Evidence based medicine and guideline formulation**

Clinical guidelines are needed in order to have uniform standardized clinical practice. The usual hierarchy of evidence is: Randomized controlled trials and systematic reviews of randomized controlled trials are classified as
Grade 1 evidence. Cohort studies, uncontrolled studies, cross sectional surveys come in the Grade 2 evidence and case reports and so called expert opinions come lower down as Grade 3 evidence. Only grade one evidence should be included in the recommendations for clinical practice.

**Points to Remember**

- Evidence based medicine revolves around well formulated research questions.
- It consists of accessing, appraising, applying and auditing research findings.
- Good quality evidence from RCTs and systematic reviews of RCTs forms the basis of clinical practice guidelines.
- The Cochrane library is one such source of quality evidence which can be accessed for free in India.

**References**


**CLIPPINGS**

Tomohiro Oishi, Kazuyuki Ueno, Kyoko Fukumoto, Kou Matsui, Shinya Tsukano, Tetsuo Taguchi, Makoto Uchiyama

Prophylactic cefdinir for pediatric cases of complicated urinary tract infection, Japan Pediatric Society 2010

This study evaluated the effect of prophylactic cefdinir (3 mg/kg given once daily) for the prevention of recurrent and complicated urinary tract infections (UTIs) in pediatric patients. The study included 14 infants who were observed for at least 6 months following the first signs of infection. Twelve patients had vesicoureteric reflux (grade I: 2; grade II: 3; grade III: 6; grade IV: 1), and 2 patients had ureteropelvic junction stenosis. No patients discontinued medication due to diarrhea or other adverse drug reactions. The patients had a 6-month recurrence-free rate of 93% (13/14); only 1 patient had recurrent UTIs. These results show that cefdinir given 3 mg/kg once daily is very effective and safe for preventing recurrent complicated UTIs in infants.
USE OF β-LACTAM ANTIBIOTICS IN PEDIATRIC INFECTIONS

* Jeeson C Unni

Abstract: β-lactam antibiotics are a large group of antibiotics that contain the beta-lactam ring in their molecular structure. They are the most commonly used antibiotics in pediatric practice. And hence a good understanding of their potential is essential for ensuring rational pediatric therapeutics. An attempt is made, in this review article, to highlight the role of each of these drugs in pediatric infectious disease today. Since the group includes all the various penicillins, cephalosporins and the carbapenems, it has required a thorough literature review to suggest some useful recommendations for their use.

Keywords: β-lactam antibiotics, Penicillins, Penicillinase-resistant penicillins, Extended spectrum penicillins, Beta lactamase inhibitor combinations, Cephalosporins, Carbapenems.

This is the most widely used and therefore also the most widely misused group of antibiotics. The beta-lactam antibiotics include all the various penicillins, cephalosporins and the carbapenems; basically any antibiotic agent which contains a β-lactam nucleus in its molecular structure. From the time penicillin was discovered in 1928 by Alexander Fleming, beta lactams have been the prime antibiotic used for prophylaxis and treatment of susceptible infections in pediatrics. Even today, newer beta lactams continue to be introduced. Whilst, traditionally, they were mainly active only against gram-positive bacteria, the development of broad-spectrum β-lactam antibiotics active against various Gram-negative organisms has increased the usefulness of the β-lactam antibiotics.

It is widely distributed in tissues, predominantly excreted in urine, does not cross un-inflamed blood-brain barrier though some do penetrate into CSF in meningitis. β-lactams should not be given intra-thecally as their effect is doubtful and it could cause a toxic encephalopathy.

The beta-lactam antibiotics are bactericidal and need to be intact when they bind to the penicillin binding protein to exert their effect of disrupting cell wall synthesis. Therefore, the two main mechanisms leading to resistance are destruction of the intactness of the β-lactam ring by β-lactamases produced by microorganisms and by alteration of the penicillin binding protein. Notable examples of this mode of resistance include methicillin-resistant staphylococcus aureus (MRSA) and penicillin-resistant streptococcus pneumoniae.

β-lactams are well tolerated. Their most important side effect is hypersensitivity.

Ampicillin and amoxyccilllin are known to produce a non-allergic maculopapular rash in 10-15% patients especially with concurrent EB virus or CMV infection.

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Cephalosporins and carbapenems demonstrate 5-10% cross reactivity with penicillins and therefore may be used with caution if the child is sensitive to penicillin but should be avoided if penicillin produces an anaphylactic reaction. All injectable β-lactams have potentially significant sodium levels.

**Penicillins (Benzylpenicillin / Penicillin G)**

Penicillin G is given parenterally because it is unstable to the hydrochloric acid of the stomach. It is effective against streptococcus, pneumococcus, gonococcus, meningococcus and some anaerobes. But of late there have been increasing reports of pneumococcus resistant to penicillin1,2 the world over but this has not been a problem till of late in India.3,4 Therefore, except in very highly resistant strains, the treatment of penicillin-resistant S. pneumoniae causing bacteremia, sinusitis, otitis, bronchitis, or community-acquired pneumonia could be with penicillin or any β-lactam. Only in pneumococcal meningitis caused by penicillin-resistant pneumococci, the pediatrician has to be careful in selecting an anti-pneumococcal antibiotic with adequate cerebrospinal fluid penetration and definite susceptibility.1 High penicillin resistance rates are being reported for gonococcus too all over the world, including India.5-7 Therefore, penicillin is no longer recommended for empiric treatment of pneumococcal meningitis and gonococcal infections. Important present day indications would include

1. Emergency treatment of suspected meningococcal infection.8
2. With aminoglycoside in the empirical treatment of neonatal sepsis.9
3. Empirical therapy of pneumococcal infection except for meningitis.1
4. When IV therapy is required for β-hemolytic streptococcal infection10

5. Specific conditions. – infective endocarditis, diphtheria, tetanus

**Phenoxymethylpenicillin (penicillin V)**

Phenoxymethylpenicillin or penicillin V is administered orally and has a spectrum similar to that of benzylpenicillin. It is however, not recommended for serious generalised infections due to its erratic absorption.

Indications would include streptococcal tonsillitis11,12 secondary prophylaxis of rheumatic fever12 and prophylaxis against streptococcal infections following splenectomy13,14 and in sickle cell disease.14 However, there are concerns regarding the compliance and effectiveness of phenoxymethylpenicillin prophylaxis following splenectomy14 and in sickle cell disease.14,15 Phenoxymethylpenicillin is preferred over penicillin G because food does not interfere with its absorption.

**Penicillinase-resistant penicillins (Flucloxacillin / Cloxacillin)**

These drugs are stable against staphylococcal penicillinases. They are therefore, both orally and parenterally, the drug of choice for the initial treatment of staphylococcus aureus infections because more than 90% of these organisms, regardless of the source, are resistant to penicillin.16 These β-lactamase-resistant drugs also have reasonable activity against streptococci and therefore provides adequate cover for mixed superficial infections (eg. impetigo) and may be reserved for cases with numerous lesions spread over a large area or where systemic symptoms are present. Flucloxacillin and cloxacillin, in combination with aminoglycosides or fusidic acid may be used for treating deep seated methicillin-susceptible staphylococcal infections like osteomyelitis and infective endocarditis17,18
Resistance of staphylococcus to flucloxacillin/cloxacillin is mediated by altering target penicillin binding protein.

Most hospital acquired coagulase-negative staphylococci and methicillin resistant staphylococcus aureus (MRSA) are resistant to these drugs.

These staphylococci are then often resistant to most β-lactams and other antibiotics.

Glycopeptide antibiotics vancomycin and teicoplanin and clindamycin are mainstay of therapy of infections with such strains.

**Extended spectrum penicillins (ampicillin / amoxycillin)**

Compared to benzylpenicillin, ampicillin and amoxycillin have greater activity against enterococci, listeria and gram negative organisms like H. influenzae and E. coli. They are however, not beta-lactamase stable and resistance due to altered penicillin-binding proteins also occurs. Almost all staphylococcus aureus, increasing numbers of M catarrhalis, 25% H influenzae, 70% E coli are resistant to these drugs and this limits its value in empiric therapy.

Amoxicillin is significantly better absorbed than ampicillin and remains the oral drug of choice in otitis media. In areas where drug-resistant streptococcus pneumoniae is prevalent, doubling the usual dose of amoxicillin improves the drug concentrations in the middle ear fluid and, due to a unique property that β-lactams possess, is thereby able to overcome the resistance conferred by alteration in the protein moiety. Children seem to tolerate these doses of 80mg/kg/day fairly well. Though, as mentioned earlier, the incidence of drug-resistant streptococcus pneumoniae is low in India to date, there are a few reports of this problem emerging of late in some parts of south India. Amoxicillin may also be used for treating non-severe pneumonia in children.

It should, however, not be used as first choice for treating uncomplicated sore throats as most are viral and the streptococcal throats are better treated with phenoxymethylpenicillin. It is however, a second line drug in a once daily dose of 750mg/day for streptococcal pharyngitis.

Intravenous ampicillin and amoxicillin should only be used for empiric therapy with another drug to ensure adequate cover. They may be combined with aminoglycoside as alternative to cephalosporin monotherapy in gastrointestinal and urinary tract infections. Combinations of amoxicillin or ampicillin with cephalosporins or aminoglycosides are effective against difficult to treat infections with enterococci and Listeria monocytogenes.

**β-lactamase inhibitor combinations**

Beta lactamase inhibitors (eg. clavulanic acid, tazobactam and sulbactam) are beta lactam compounds that have very little antibacterial activity but bind to the β-lactamases produced by micro-organisms more “efficiently” than the actual β-lactam antibiotic itself and thereby, are capable of inhibiting β-lactamases and protecting the parent penicillin with which they are associated from degradation by the β-lactamase enzymes. They inhibit β-lactamases of Staphylococcus aureus, Gram negatives like H influenzae, M catarrhalis, many E. coli, Klebsiella, and bacteroides; but are inactive against β-lactamases of Psuedomonas, Enterobacter, Serratia, Citrobacter sp.

The penicillin-β-lactamase-inhibitor combinations penetrate CSF poorly.
Further, it is not appropriate to use such combinations if the infecting organism is sensitive to the parent penicillin given alone.

Amoxicillin – clavulanic acid combination confers amoxicillin activity against all Staphylococcus aureus, except MRSA, and M catarrhalis over 90% of H influenzae, E coli and many other gram-negatives, including anaerobes. A high incidence of GI intolerance is the major side-effect. The combination, because it covers gram positive, gram negative and anaerobic organisms, is the drug of choice in human and animal bites, rhinosinusitis and mixed soft tissue infections and the second line drug for recurrent otitis media respiratory tract infection and UTI. It may be noted that amoxicillin-clavulanic acid would be effective for otitis media only if the resistance to amoxicillin is attributable to the production of beta lactamases by the infecting organisms. It would not be effective if alteration of protein is the cause for resistance.

Intravenous co-amoxiclav is alternative to cephalosporins/metronidazole combination for surgical prophylaxis.

Piperacillin + tazobactam and ticarcillin + clavulanic acid are combinations of anti-psuedomonal penicillin with beta lactamase inhibitor. They are very broad spectrum antibiotics but some studies show that they are not very effective against enterobacter and similar species and that is a significant disadvantage.

Some centres use them for empiric treatment of fever in a neutropenic child as monotherapy or in combination with aminoglycoside.

Others reserve these antibiotic combinations for serious infections resistant to other antibiotics.

Cephalosporins

This is a large, ever growing group of β-lactam antibiotics possessing a broad spectrum of anti-bacterial activity, which are useful and generally well tolerated. However, they are not active against enterococci, MRSA, listeria species and anaerobes.

The cephalosporin nucleus can be modified to innovate upon its properties. Cephalosporins are thus grouped into “generations” depending on their antimicrobial properties. The first cephalosporins were designated first generation while later, more extended spectrum cephalosporins were classified as second, third and fourth generation cephalosporins. Each newer generation of cephalosporins has significantly greater gram negative antimicrobial properties than the preceding generation sometimes at the expense of gram positive activity. Conversely, the earlier generations of cephalosporins have greater gram positive (staphylococcus and streptococcus) coverage than the newer generations. There is some disagreement on the definition of drugs to be included in the various generations of cephalosporins. For example, in Japan, the fourth generation of cephalosporins is not yet recognized, and are included in the third generation instead and cefaclor is classed as a first generation cephalosporin.

Ten-16% of patients allergic to penicillins are also allergic to cephalosporins. Patients who experience anaphylactic reactions to penicillins should not be given a cephalosporin. In general the new oral broad spectrum cephalosporins offer no clear advantage over previously available drugs and are far more expensive and should only be used as a second line agents.

First generation cephalosporins

Cephalexin and cefadroxil are examples of the first generation cephalosporins. They are active against streptococci, staphylococci and most community acquired E coli and klebsiella.
They do not afford H influenzae cover and they do not cross the blood brain barrier. The first generation cephalosporins may be considered for the following infections in children

1. As good urinary concentrations are achieved, they are useful in treating urinary tract infections especially because there is increasing resistance of urinary pathogens to amoxicillin and sulfamethoxasole-trimethoprim. They are also used for prophylaxis for recurrent urinary tract infections in children.

2. Skin and soft tissue infections

The first generation cephalosporins are of little use in respiratory infections due to lack of H influenzae activity. They are not recommended for use in newborn because less that 50% of the orally administered dose is absorbed in neonates.

**Second generation cephalosporins**

Oral second generation cephalosporins are cefuroxime, cefprozil, cefzil, cefaclor etc. and cefuroxime is the only one given parenterally.

They have a gram positive spectrum similar to 1st generation drugs but have increased gram negative activity (including H influenzae) and a greater $\beta$-lactamase stability and though they may be used for treating acute otitis media, respiratory infections, sinusitis, skin and soft tissue and urinary tract infections, none of them are first line drugs for any of these conditions. There are a number of brands of these second generation oral cephalosporins available in the market and as a result of their widespread misuse, resistance to them is likely to increase. Oral cefuroxime has been suggested as a second-line agent for treating acute otitis media in the past, but recent surveillance data suggest it may no longer be active against penicillin-resistant strains of S. pneumoniae. Though this is not yet a major problem in India, its use in pediatrics is restricted because it is extremely unpalatable. Further, most oral second generation cephalosporins have doubtful bioavailability and have little advantage over 1st generation cephalosporins. Cefuroxime given parenterally, however, is the only second generation cephalosporin used as a first line drug.

It may be used for a number of serious infections including that of the urinary and respiratory tract, skin and soft tissue, bone and joints and for prophylaxis and treatment of intra-abdominal infections in combination with metronidazole.

**Third generation cephalosporins**

Although these agents have better Gram negative cover than second generation cephalosporins, (sometimes including pseudomonas) this feature is not clinically important. However, there are some clinical situations where third generation parenteral cephalosporins have significant advantage over cefuroxime.

**Parenteral third generation cephalosporins**

Cefotaxime and ceftriaxone have identical spectrum including streptococci, staphylococcus aureus and Gram negative organisms. They are the most effective cephalosporin against penicillin resistant pneumococci and non beta-lactamase mediated amoxycillin resistant H.influenzae. Ceftriaxone has a longer half life and may be given once daily. It is excreted through the liver and may be associated with formation of crystalline biliary sludge (reversible on cessation of the drug) particularly in neonates.
Ceftazidime has the broadest Gram-negative cover including pseudomonas but has very poor Gram positive activity.

Common indications in pediatrics for cefotaxime are meningitis, meningococcal septicemia, empirical treatment of severe sepsis and epiglotitis after securing the airway. Emphasis is being made here of the fact that of today, the first choice broad-spectrum antibiotic for empirical therapy of severe sepsis with or without shock in children is cefotaxime or ceftriaxone. All other broad-spectrum antibiotics that are discussed in this article are to be used when certain specific conditions exist and these specifics are mentioned along with the review of those drugs.

Ceftriaxone has similar uses as cefotaxime and in addition may be used as fourth line drug for acute otitis media, as combination in sepsis and for multi-drug-resistant Salmonella typhi infection.

Ceftazidime may be reserved for gram-negative infections especially if pseudomonas is the cause of infection as multidrug-resistant pseudomonas aeruginosa is emerging and combination therapy is becoming increasingly necessary.

Oral third generation cephalosporins (cefixime, cefpodoxime, cefdinir)

Cefixime covers *H. influenzae, M. catarrhalis, Gr A streptococcus, Gram negatives like cefotaxime (but not effective against staphylococci or S pneumoniae). It may be used for treatment of blood and mucus diarrhoea and uncomplicated enteric fever.

Cefpodoxime has less Gram negative activity than cefixime but has good staphylococcal activity and better activity against S pneumoniae and therefore may be effective in respiratory tract infections.

Cefdinir has an extended spectrum of activity that includes many Gram-negative and positive aerobic organisms, including S pneumoniae, Staphylococcus, Gr.A.Streptococcus, *H.influenzae* and *M.catarrhalis*. It is, therefore, a possible second-line drug in treatment of acute otitis media, instead of cefuroxime, particularly among children who are likely to be noncompliant with a two- to three-times-daily dosing schedule or due to the unpalatability of cefuroxime. It may also be used in mild-to-moderate respiratory tract or skin infections, particularly in areas where β-lactamase-mediated resistance among common community-acquired pathogens is a concern.

However, it is important to be informed that none of the oral third generation cephalosporins are currently first line drugs for any pediatric illness. And therefore, a conscious effort should be made, to use the first line drugs first and reserve these drugs for treatment failures.

Fourth generation cephalosporins (cefpirome, cefpirome)

They have a very broad spectrum with Gram-positive cover comparable to the first and second generation cephalosporins and Gram negative cover similar to that of third generation cephalosporins. Activity against *Pseudomonas aeruginosa* of cefepime is similar to ceftazidime and superior to cefpirome. The fourth generation cephalosporins are effective against enterococci and enterobacter which are resistant to most of the other cephalosporins. Though cefepime may be used for empirical broad-spectrum antibiotic treatment for febrile neutropenia, carbapenems are a more reliable choice. Further, there are reports that increased ciprofloxacin-resistant *K. pneumoniae* and meropenem-resistant *Acinetobacter* species was significantly associated with the increased usage of extended-spectrum cephalosporins, including cefepime and ceftiraxone. Hence, there is a need to tread
carefully when prescribing 4th generation cephalosporins.

**Carbapenems**

These are exceptionally broad spectrum β-lactams for parenteral use.

Imipenem with cilastin is one of them - cilastin inhibits renal brush border inactivation of imipenem and prevents formation of nephrotoxic metabolites. But there is a risk of seizures especially if there is underlying CNS disease.

Meropenem is the latest introduction of carbapenem antibiotic. It does not need cilastin stabilisation and therefore is associated with less treatment induced seizure. But there are isolated reports of seizure worsening in children taking valproate and meropenem simultaneously. It has a similar spectrum but acinobacter respond poorly even though sensitive in vitro.

Most gram positives, gram negatives and anaerobes are susceptible to carbapenems. They are stable against all β-lactamases with penicillinase or cephalosporinase activity.

Therefore carbapenems are very active against enterobacter and other species resistant to cephalosporins. However, they are not active against MRSA, coagulase negative staphylococci and some enterococci. These agents achieve therapeutic concentrations in the CSF.

The present day indications for carbapenems in pediatrics is restricted to the treatment of serious hospital acquired infections not responding to standard therapy, treatment of febrile neutropenia and probably meningitis. There is limited experience with these agents in the newborn.

**Conclusion**

β-lactam antibiotics are indicated for the prophylaxis and treatment of bacterial infections caused by susceptible organisms. The earlier beta-lactams were only active against Gram-positive bacteria. However, the more recent development of broad-spectrum β-lactam antibiotics active against various Gram-negative organisms has increased their usefulness. It is important to make proper diagnosis before choosing the appropriate antibiotic for that particular illness. It is also important to use the β-lactam antibiotic that is first line drug for the given infection and to avoid using very broad spectrum antibiotics where a narrower spectrum antibiotic is as effective.

**Points to Remember**

- Benzylpenicillin may be used for emergency treatment of suspected meningococcal infection, empirical therapy of pneumococcal infection except for meningitis, for specific conditions like infective endocarditis, diphtheria and tetanus and along with aminoglycoside in the empirical treatment of neonatal sepsis.

- Phenoxyethylpenicillin is indicated for treatment of streptococcal tonsillitis, secondary prophylaxis of rheumatic fever and prophylaxis against streptococcal infections following splenectomy and in sickle cell disease.

- Penicillinase-resistant penicillins (Flucloxacillin / Cloxacillin), orally and parenterally, are the drug of choice for the initial treatment of Staphylococcus aureus infections. Most hospital acquired coagulase-negative staphylococci and methicillin resistant Staphylococcus aureus (MRSA) are resistant to these drugs. And staphylococcus resistant to penicillinase-resistant penicillins would only respond to glycopeptide antibiotics.
• **Amoxicillin** is the oral drug of choice for acute suppurative otitis media and community acquired pneumonia.

• **Amoxycillin – clavulanic acid** is the drug of choice in human and animal bites, rhinosinusitis and mixed soft tissue infections and the second line drug for recurrent otitis media, respiratory tract infection and UTI.

• **First generation cephalosporins (Cephalexin)** are useful in treating urinary tract and skin and soft tissue infections.

• **Cefuroxime**, given parenterally, is the only second generation cephalosporin recommended as a first line drug. It may be used for a number of serious infections including that of the urinary and respiratory tract, skin and soft tissue, bone and joints and for prophylaxis and treatment of intra-abdominal infections in combination with metronidazole.

• **Parenteral third generation cephalosporins** cefotaxime and ceftriaxone may be used for treatment of meningitis, meningococcal septicemia, empirical treatment of severe sepsis and epiglotitis after securing the airway. Emphasis is being made here of the fact that of today, the first choice broad-spectrum antibiotic for empirical therapy of severe sepsis with or without shock in children is cefotaxime or ceftriaxone. Ceftriaxone, in addition, may be used as fourth line drug for acute otitis media, as combination in sepsis and for multi-drug-resistant salmonella typhi infection.

• **Parenteral third generation antibiotic ceftazidime** may be reserved for gram-negative infections especially if pseudomonas is the cause of infection.

• **None of the oral third generation cephalosporins** are currently first line drugs for any pediatric illness.

**References**


10. American Academy of Pediatrics. Committee on Infectious Diseases. Severe invasive group


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**CLIPPINGS**


*Early Cranial Ultrasound Lesions Predict Microcephaly at Age 2 Years in Preterm Infants, Journal of Child Neurology, 08/24/2010*

To assess how well early ultrasound lesions in preterm newborns predict reduced head circumference at 2 years, the investigators followed 923 children born before the 28th week of gestation who were not microcephalic at birth. Six percent of children who had a normal ultrasound scan were microcephalic compared with 15% to 20% who had intraventricular hemorrhage, an echolucent lesion, or ventriculomegaly. Ventriculomegaly had a higher sensitivity for microcephaly than did an echolucent lesion (24% vs 16%, respectively). Focal white-matter lesion (echolucent lesion) and diffuse white-matter damage (ventriculomegaly) predict an increased risk of microcephaly.
PITYRIASIS ROSEA

* Vijayabhaskar C

Abstract: Pityriasis rosea is a common self-limiting condition seen in children and adolescents characterized by appearance of herald patch followed by skin lesions along the Langer’s line. Exact etiology is not known. Viral etiology has been proposed and may present without any symptoms to mild itching and rarely severe itching and lasts upto 8 weeks. The disease has to be differentiated from other conditions such as secondary syphilis, drug eruption, tinea corporis, erythema multiforme, etc. It is usually a clinical diagnosis in which reassurance of the patient and parents are important. Topical steroids and oral antihistamines are used. Erythromycin orally and acyclovir have been tried to shorten the course of the disease.

Keywords: Pityriasis rosea, Herald patch, Collarate of scales.

Pityriasis rosea (PR) is a common self-limiting skin condition described for more than 2 centuries. It is characterized by appearance of a herald patch followed by skin lesions along the Langer’s lines or cleavage lines. It is most commonly seen in adolescents and children with no gender predilection although a study has shown very slight predilection in females. This condition has been reported as early as in 3 months of age.

Etiology

The exact etiology is unclear, although some of the factors relate it to an infectious cause particularly viral.1 Several studies have shown association with human herpes virus 6 and 7 and an equal number have failed to show a causal link.2

Generally the condition occurs in epidemics proving that the infectious agent is present in the community.

Recurrence of the infection is rare suggesting long lasting immunity to the infectious agent.

More than 50% of the patients have prodromal symptoms before the onset of the herald patch.

Some of them have an increase in B lymphocytes and a decrease in T lymphocytes and increase in erythrocyte sedimentation rate.

Other infective agents considered as causes are Legionella pneumoniae, Chlamydia pneumoniae and Mycoplasma pneumoniae but there is no support in the form of rise in antibody titres in subsequent studies.

Clinical features

Patient presents with non specific clinical features. In 50% of these patients, there may be features of upper respiratory tract infection. Malaise, nausea, fever, joint pain, headache and lymph node enlargement may occur before the appearance of skin lesions in the form of ‘herald
patch’. The herald patch commonly appears over the trunk but rarely occurs over the neck or extremities and is usually 1 to 2 cm in diameter. It is ovoid or round in shape with a salmon coloured area in the centre which is wrinkled and a dark red peripheral zone. Secondary eruptions occur within 1 to 30 days. Secondary eruptions are symmetrical and consists of multiple erythematous macules progressing to small red papules localized predominantly to the trunk and adjacent area of the neck and proximal extremities, the ‘vest area’. The secondary eruption follows Langer’s lines. Involvement of the back takes a Christmas tree or fir tree pattern. On the lower abdomen and back it appears in a transverse manner.

The secondary rashes are erythematous oval patches with peripheral collarette of scales. Sometimes the rash is atypical including vesicular, purpuric and crusted type.

Usually the rash lasts from 4 weeks to 8 weeks. Mild itching is seen during the first week of occurrence of lesion and becomes asymptomatic there after. Very rarely they may present with severe itching.

Post inflammatory hyperpigmentation or hypo-pigmentation can occur.

Recurrence rate is as low as 3%.

**Other types of Pityriasis rosea**

Papular PR – Multiple papules occurs in the vest area.

Inverse PR – Here the extremities are affected and the trunk is spared. Facial involvement is seen in children. Axillae and groin are involved.

Localized PR - Lesion can occur in a localized area and diagnosis becomes difficult.

Pustular PR, vesicular PR, purpuric PR and erythema multiforme like PR are some of the other variants.

In Gigantean PR, the lesions are larger but less in number.

PR urticata presents more commonly with urticarial lesions.

Oral lesions in the form of erythematous plaques, hemorrhagic puncta and ulcers may occur.³

**Drugs causing rash similar to pityriasis rosea**

Arsenic compounds, barbiturates, bismuth, captopril, clonidine, gold, interferon, ketotifen fumarate, metronidazole, hepatitis B vaccine and Bacillus Calmette-Guérin vaccine.

**Investigations**

Investigations are rarely needed in case of PR. Though the blood count may be normal, sometimes leucocytosis, neutrophilia, basophilia and lymphocytosis may be seen.

When appropriate, VDRL test and Fluorescent Trepenomal Antibody test are done to rule out syphilis.

Skin scraping from the lesions in wet mount could be done to rule out superficial fungal infections.

**Differential diagnosis**

The following conditions should be considered and ruled out clinically and by investigation where ever necessary.

Herald patch of Pityriasis rosea has to be differentiated from tinea corporis, annular psoriasis, granuloma annulare and nummular eczema.

In tineacorporis the patches have papular, scaly border with central clearing and associated itching.
Secondary lesions of Pityriasis rosea have to be differentiated from the following conditions.

**Erythema multiforme**: Characterised by acute onset with oval or round fixed erythematous skin lesion which progress to form concentric zones of color change in which the central zone of dusky, blistered or crusted area surrounded by pallor and erythema. These are called as target lesions or iris lesions. Palms and soles are involved. Usually associated with herpes simplex.

**Guttate psoriasis**: The large herald patch is absent in guttate psoriasis. Skin lesions do not have peripheral collaratte of scales and the scales are usually thick.

**Pityriasis versicolor**: More common over face and trunk and lesions are scaly without collarette. They are non itchy and lesions have pencil lined borders.

**Drug reaction**: History of drug intake prior to the onset of rash and mucosal involvement will aid the diagnosis.

**Secondary syphilis**: There is no herald patch and itching is absent with involvement of palms and soles.

**Treatment**

Reassurance is very important. Parents have to be explained about the nature and course of the disease. The benign nature of the disease and the tendency to resolve after a few days or few weeks has to be stressed.

Oral antihistamines are advised if itching is present.

Topical steroids could be used for a period of 2 weeks. If itching is very severe, systemic steroids in the form of oral prednisolone is used for a period of 5 to 7 days.

In a small study, oral erythromycin for 2 weeks was found to be effective in bringing down the symptoms and disappearance of rash. Best results were obtained if erythromycin was started within 2 weeks of the appearance of the eruption. The exact mechanism is not known. Anti inflammatory property of the drug could have helped in the resolution of the symptoms. A study was done with azithromycin in children, in whom it was not found to be effective.

UV B therapy has been used in severe cases but the incidence of post inflammatory hyper pigmentation is high.

In one non randomized non blinded study, acyclovir was shown to hasten resolution if started within 1 week of the appearance of rash (800mg 5 times a day for 7 days).

Children may be allowed to attend school.

**Points to Remember**

- **Pityriasis rosea**, a common benign usually asymptomatic skin disease of varied etiology, needs to be differentiated from other similar diseases.
- Reassurance and symptomatic treatment is sufficient in many cases.
- Topical steroids, oral antihistamines and erythromycin form the main stay of management.
- Short course of systemic steroids may be given when required.
- Use of oral acyclovir needs further evaluation.

**References**


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**JIPMER NEOCON 2010**

The Annual convention of the Tamilnadu Neonatology forum will be held on the 20th & 21st of November 2010 at the Jipmer Auditorium, Puducherry. The programme schedule includes preconference workshops, CME on practical and problematic areas in neonatology and scientific paper presentations.

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Infarction is the result of vascular occlusion, be it arterial or venous. Arterial occlusion itself is rare in children. Venous thrombosis is even rarer and also presents with CNS symptoms as varied as seizures, neurological deficit and signs of raised intracranial tension. It is associated with anemia, infections like mastoiditis and meningitis, dehydration and various prothrombotic disorders. The diagnosis of venous thrombosis requires MRI and more specifically MR venography. But MRI as such, is not the first investigation in an acute presentation. Therefore there are certain features in an emergency CT that along with clinical features can lead to a suspicion of venous thrombosis. An MRI can then be done.

The thrombosed sinus can appear as a dense structure in a plain CT. The dense, thrombosed superior sagittal sinus can be seen in the midline posteriorly. This, with the dense falx gives the “delta sign”. It has to be remembered that neonates show increased density of sinuses due to raised hematocrit. On a contrast CT the thrombus can show as a filling defect in the superior sagittal sinus surrounded by the enhancing walls of the sinus. This is the classical “reverse delta sign” or “empty triangle sign”, though it is not always present. Fig.1. shows this sign in the falx posteriorly. The infarct is also seen on the right, anteriorly.

Parenchymal abnormalities include infarctions and hemorrhage. The infarcted areas do not follow specific arterial territorial patterns that we saw in the previous issue. Rather they are seen para sagittally in case of superior sagittal sinus thrombosis, and usually bilaterally. In case of transverse sinus thrombosis there is venous infarction in temporal and adjacent parietal regions on that side. When deep veins are thrombosed both thalami suffer.

Fig.2. shows a T2 weighted MRI image of venous infarction in the frontoparietal area on the right. The signal in MRI is dependant on water that is the largest constituent of our body. When ischemia sets in there is intracellular accumulation of ions which draws in water along the osmotic gradient. A little later, endothelial damage causes outflow of fluid from vessels. The presence of water and therefore more hydrogen atoms makes the infarcted area white in color. Fig.3. is an arterial infarct of the left middle cerebral artery for comparison. The infarct is white just like the ventricles that contain fluid.

Hemorrhage in the involved areas is common with venous thrombosis because the thin walls easily allow break through of blood when intravenous pressure rises due to distal obstruction. Infarctions involving white matter (Fig.1) and grey-white matter junctions are also

* Associate Professor,
*** Asst. Professor,
**** Professor,
Chengalpet Medical College Hospital, Chengalpet
Fig. 1. Empty triangle sign

Fig. 2. Venous infarction – Rt. frontal region (same patient as Fig 1)

Fig. 3. MRI image of arterial infarction – Lt MCA

Fig. 4. MRV- Superior sagittal sinus is not seen in its location.

Fig. 5. Posterior reversible encephalopathy syndrome (PRES)

Fig. 6. PRES now resolved- (Repeat film after 10 days)
features of venous infarction. Another important feature is cerebral edema due to venous hypertension. This is responsible for raised intracranial tension and its complications like cerebral herniation and coma.

MRI is the investigation to establish a diagnosis of cerebral venous thrombosis. It will show the clot itself and its sequelae. The acute clot is isodense on T1 and hypointense in T2 films. The subacute clot is hyperintense on T1.

MR venography, using specific techniques (gradient recoil echo), will demonstrate lack of flow in the venous channels. Fig.4. is the MR venogram of the same patient as Fig.1. The superior sagittal sinus is not seen in its location which is along the contour of the vault. Only the internal cerebral vein and the more inferior basal vein are seen emptying into the vein of Galen and then into the straight sinus.

Fig.5. is that of a nine year old boy who presented with headache and altered consciousness. He had glomerulonephritis with hypertension. A CT was taken suspecting intracranial hemorrhage. Instead, what we see is bilateral hypodensity in the occipital regions just like infarction. This is called posterior reversible encephalopathy syndrome (PRES). The term arose because the common region involved is the posterior parietal and occipital regions though it can also occur in the temporal and frontal lobe, cerebellum and basal ganglia. It is reversible as you can see in the repeated study (fig.6.) once the hypertension is controlled. The densities are due to vasogenic edema or leakage of fluid due to increased intravascular pressure. Drugs like cyclosporine and tacrolimus which cause hypertension are also known to cause PRES.

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