Acute Flaccid Paralysis
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ABSTRACT
Acute flaccid paralysis is an emergency and includes a variety of diagnostic possibilities. Identification of life threatening problems and emergency stabilization should precede further diagnostic evaluation. The common differential diagnoses in the post polio eradication era include Guillain Barre syndrome, acute transverse myelitis and traumatic neuritis. It is essential to rule out reversible causes such as hypokalemia and snake envenomation in the appropriate clinical setting. Stabilization of spine is imperative when trauma is suspected, and early neuroimaging is essential in any child who presents with features of myelopathy to rule out compressive lesions. Careful monitoring of respiratory muscle strength is essential, especially in children who progress rapidly and early institution of mechanical ventilation is essential. Management of these children is largely supportive, and includes appropriate ventilator support, provision of early enteral nutrition, physical therapy, strict asepsis, good nursing care, bowel and bladder care and psychosocial support. Immunotherapy is useful in certain patients with Guillain Barre syndrome, transverse myelitis and myasthenic crisis. Prolonged ventilation is anticipated in children with severe disease and necessitates tracheostomy to increase patient comfort and facilitate weaning from ventilation. Children who are admitted in intensive care for various disease processes may develop weakness due to critical illness related neuromuscular weakness, no effective treatment exists and management is aimed at identification of risk factors and prevention strategies.

Key words: Neuromuscular weakness, flaccid paralysis, children, Guillain Barre, ventilation.

Introduction
Acute flaccid paralysis (AFP) is a clinical syndrome characterized by rapid onset of hypotonic weakness, progressing to maximum severity within several days to weeks. The syndrome assumes importance in an emergency setting owing to respiratory and bulbar muscle weakness, which when present, may lead to respiratory failure and death if not managed appropriately. The public health aspect of this syndrome which is extremely crucial consists of two important components: 1) all AFP cases are to be notified to a surveillance team and 2) appropriate stool samples are to be sent to WHO accredited laboratory.

Initial Assessment and Stabilization in Pediatric Emergency
Any child presenting with AFP should be evaluated systematically for life threatening emergencies and stabilized before proceeding to detailed neurological evaluation and specific therapy.

a) Airway stabilization: These children are prone to airway problems due to bulbar weakness and retained secretions. Symptoms such as weak cry, voice change, difficulty in swallowing, pooling of secretions, difficulty in clearing secretions, ineffectual cough, regurgitation of food particles, choking during feeding are pointers towards bulbar weakness. Children with bulbar weakness require repeated oral suctioning to prevent pooling and aspiration. In an unstable airway, the risk of aspiration can be minimized by nasogastric feeding and intubation with a cuffed endotracheal tube.

b) Detection of respiratory weakness: Weakness of diaphragm and/or intercostal muscles results
in respiratory failure. Examination should include respiratory rate, chest expansion, breathing pattern, accessory muscle use, and single breath count. The normal early tachypneic response to hypoventilation is lacking in these children due to neuromuscular weakness. Hence there is a danger of progression to respiratory failure if not carefully monitored. Single breath count is a useful bedside tool for older children who can cooperate with the examiner and a count up to 10, and up to 25 suggests a forced vital capacity of at least 15-20 ml/kg and 30-40 ml/kg respectively. Serial monitoring is important and appropriate ventilator support should be provided when progressive respiratory weakness is detected. There could be signs of aspiration due to bulbar muscle weakness.

### Table 1: Differential diagnosis of Acute flaccid paralysis in children (Adapted from Reference 2):

<table>
<thead>
<tr>
<th>Anatomical Site</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal cord</td>
<td>Acute transverse myelitis, Ascending myelitis, Traumatic spinal injury, Epidural abscess, hematoma, discitis</td>
</tr>
<tr>
<td>Anterior horn cell</td>
<td>Poliomyelitis, Enteroviral myelitis, Guillain Barre Syndrome</td>
</tr>
<tr>
<td>Nerve roots/ Nerves</td>
<td>Traumatic sciatic neuritis, Post diphtheritic neuropathy, Acute porphyria, Arsenic poisoning</td>
</tr>
<tr>
<td>Neuronal muscular junction</td>
<td>Myasthenia gravis, Snake envenomation, Organophosphates, Botulism, Critical Illness Neuromuscular weakness, Hypermagnesemia</td>
</tr>
<tr>
<td>Muscle</td>
<td>Viral myositis, Polymyositis, Rhabdomyolysis, Hypokalemia, Periodic paralysis</td>
</tr>
</tbody>
</table>

### Table 2: Salient features of the common differential diagnosis of Acute flaccid paralysis (Adapted from Reference 3):

<table>
<thead>
<tr>
<th>Feature</th>
<th>Poliomyelitis</th>
<th>Guillain Barre Syndrome</th>
<th>Acute transverse myelitis</th>
<th>Acute traumatic neuritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progression of paralysis</td>
<td>24-48 hours to full paralysis; descending; asymmetric</td>
<td>Hours to 4 weeks; mostly ascending; symmetrical</td>
<td>Hours – 4 days; symmetrical; lower limbs</td>
<td>Hours – 4 days; affects only limb</td>
</tr>
<tr>
<td>Deep tendon reflexes</td>
<td>Decreased/ Absent</td>
<td>Absent</td>
<td>Absent in lower limbs (early); hyperreflexia (late)</td>
<td>Decreased/ Absent in affected limb</td>
</tr>
<tr>
<td>Fever at onset of weakness</td>
<td>High</td>
<td>Uncommon</td>
<td>May be present</td>
<td>Present, if underlying infection being treated</td>
</tr>
<tr>
<td>Sensory signs and symptoms</td>
<td>Severe myalgia, backache, no sensory changes</td>
<td>Tingling, cramps</td>
<td>Anaesthesia of lower limbs with sensory level</td>
<td>Pain in gluteal region</td>
</tr>
<tr>
<td>Cranial nerve involvement</td>
<td>Only when bulbar involvement present</td>
<td>Often present; affecting VII, IX, X, XI, XII</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Respiratory insufficiency</td>
<td>Only when bulbar involvement present</td>
<td>In severe cases</td>
<td>Sometimes</td>
<td>Absent</td>
</tr>
<tr>
<td>Autonomic signs and symptoms</td>
<td>Rare</td>
<td>Frequent in severe cases</td>
<td>Present</td>
<td>Hypothermia in affected limb</td>
</tr>
<tr>
<td>Bladder disturbance</td>
<td>Rare</td>
<td>Occasionally (transient, at the peak of weakness)</td>
<td>Present- early and persistent</td>
<td>Never</td>
</tr>
<tr>
<td>Cerebrospinal fluid</td>
<td>Mild elevation of lymphocytes (10-200/ ml)</td>
<td>Albumin-cytologic dissociation (usually &lt;10 cells/ml, never &gt;50)</td>
<td>Normal or pleocytosis</td>
<td>Normal</td>
</tr>
<tr>
<td>Nerve conduction velocity; third week</td>
<td>Abnormal; anterior horn cell disease (normal during first 2 weeks)</td>
<td>Abnormal; slowed conduction, decreased motor amplitudes</td>
<td>Normal</td>
<td>Abnormal; motor sensory axonal damage</td>
</tr>
<tr>
<td>Diagnostic test</td>
<td>Stool viral detection</td>
<td>Nerve conduction studies</td>
<td>MRI Spine</td>
<td>Nerve conduction studies, Electromyography</td>
</tr>
</tbody>
</table>
c) **Detection of cardiovascular instability:**
Autonomic instability is common in Guillain Barre Syndrome and spinal cord dysfunction. It can result in cardiac rhythm disturbances and blood pressure changes thus necessitating continuous ECG and BP monitoring.

d) **Suspect Toxins:** Neuroparalytic snake envenomation is an important cause of flaccid paralysis, and should be ruled out in all cases of acute onset bulbar and/or respiratory weakness. ASV should be administered in a timely fashion when there is circumstantial evidence pointing towards snake envenomation. Organophosphate poisoning could also present with profound weakness, and can be identified by the classical toxidromes of anticholinergic excess.

e) **Spine stabilization:** A history of trauma requires initial immobilization of spine until imaging studies and neurological evaluation rules out trauma related myelopathy. Urgent imaging of spine is warranted in any child with features of myelopathy, to exclude extrinsic cord compression.

f) In a child presenting with AFP, it is also essential to rule out hypokalemia as a cause for paralysis, as potassium correction rapidly reverses weakness in these children. Table 1 gives the differential diagnosis of children presenting with AFP and the salient features of common clinical conditions are described in Table 2.

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**Table 3: Investigations in a child with AFP:**

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrospinal fluid</td>
<td>Elevated cell count Transverse myelitis, infectious myelitis</td>
</tr>
<tr>
<td>Albuminocytological dissociation</td>
<td>Guillain Barre syndrome, Post diphtheritic polyneuropathy</td>
</tr>
<tr>
<td>Normal</td>
<td>Traumatic neuritis, Early course of illness</td>
</tr>
<tr>
<td>Nerve conduction studies</td>
<td>Abnormal Characteristic pattern in Guillain Barre syndrome</td>
</tr>
<tr>
<td>Repititive nerve stimulation</td>
<td>Decremental response in myasthenia gravis</td>
</tr>
<tr>
<td>MRI Spine</td>
<td>Extrinsic compression Trauma, tumor, abscess, hematoma</td>
</tr>
<tr>
<td>Abnormal Spinal cord signal with enhancement</td>
<td>Transverse myelitis</td>
</tr>
<tr>
<td>Spinal nerve root enhancement</td>
<td>Guillain Barre syndrome</td>
</tr>
<tr>
<td>Creatine kinase</td>
<td>Elevated levels Viral myositis, Inflammatory myopathy</td>
</tr>
<tr>
<td>Electromyography</td>
<td>Myositis, Critical illness myopathy, Traumatic neuritis</td>
</tr>
<tr>
<td>Stool viral studies</td>
<td>Poliomyelitis, enteroviral myelitis</td>
</tr>
</tbody>
</table>

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Investigations in a child with AFP:
The choice of investigations depends on the clinical history and examination, and must be undertaken only after emergency stabilization. The urgency of investigation is also dictated by the differential diagnosis considered and its impact on patient management. Table 3 gives the utility of common investigations performed in a child with AFP.
The brief overview of the common clinical conditions presenting as AFP and their specific management strategies are outlined below:

1) **Guillain Barre Syndrome:**
Guillain-Barre syndrome (GBS) is the most common cause of AFP in children, with an incidence of 0.34-1.34 per 100,000 person years. It is a post-infectious disorder characterized by a rapidly progressive, symmetrical weakness of limbs in combination with hyporeflexia or areflexia. Approximately 50% of the children reach the maximum severity of weakness by 2 weeks, 80% by 3 weeks and the rest by 4 weeks. The diagnosis of GBS may be challenging in young children who present with irritability, fussiness or refusal to walk. Cranial nerve involvement is common, especially facial weakness, swallowing difficulties. Sensory symptoms include paresthesias, muscle pain, backache and may precede the onset of weakness. A short interval between antecedent infection and symptoms onset, cranial nerve involvement and
increased CSF proteins have been found to predict respiratory failure in children\(^5\). Indications for intensive care admission are rapidly progressive weakness with impaired ventilation, need for assisted ventilation, bulbar weakness and severe autonomic dysfunction\(^6\). Autonomic dysfunction is more frequently seen in patients with respiratory failure, quadriplegia or bulbar weakness and manifests as sinus tachycardia, bradycardia, arrhythmias, blood pressure lability, hypertension, and diaphoresis. Investigations required for definitive diagnosis are nerve conduction studies and lumbar puncture. The two common subtypes are Acute Inflammatory demyelinating polyradiculoneuropathy (AIDP) and Acute motor axonal neuropathy (AMAN). Cerebrospinal fluid (CSF) analysis can help in excluding myelitis as a cause of weakness. The classical albuminocytological dissociation is found in about 60% of patients, and CSF may be normal in the first week of illness. Presence of high fever and bladder or bowel dysfunction at onset, sharp sensory level, persistent marked asymmetry of weakness and increased CSF pleocytosis>50/ ml makes GBS a rather unlikely diagnosis. Intravenous immunoglobulin (IVIg) administered in a dose of 2 g/kg given over 2 to 5 days, is favored over plasma exchange due to the ease of administration, availability and safety profile. Plasma exchange is aimed at removing antibodies, complements factors and requires five exchanges over two weeks. The need for specialized equipment, central venous access and volume shifts resulting in cardiovascular instability makes the option difficult in children. Combination therapy is not better than either treatment option administered alone\(^6\).

2) **Acute Non Traumatic Myelopathy:**

Acute myelopathy represents a heterogenous group of disorders of varied etiologies which present with spinal cord dysfunction. The clinical presentation is dramatic with paraparesis or quadriplegia, sensory disturbances and bladder/bowel dysfunction. The first priority is an emergent neuroimaging of the spine to rule out a compressive lesion. Once compressive lesions are ruled out, inflammatory, infectious and vascular causes can be considered. The following section describes common conditions which present with acute myelopathy.

a) **Acute transverse myelitis:**

Acute transverse myelitis (ATM) is an immune-mediated inflammatory disease of spinal cord which can be idiopathic, secondary to infection/immunization or associated with other conditions such as acute disseminated encephalomyelitis (ADEM), multiple sclerosis, neuromyelitis optica, systemic lupus erythematosus, antiphospholipid antibody syndrome. The incidence is quoted as 2 cases/ million children with peak incidence between 0 and 2 years of age and 5 to 17 years of age\(^7\). This should be suspected in any child who presents with acute onset motor deficits of lower extremities, often with sensory loss and loss of bladder and bowel control. Presence of encephalopathy and seizures point to associated ADEM. Upper cervical lesions result in pharyngeal weakness and loss of airway patency, lesions above cervical level 5 results in diaphragm weakness and respiratory compromise. In the initial phase of spinal shock, deafferentation from sympathetic control centres in brainstem could present as arterial hypotension and bradycardia due to unopposed vagal activity. Autonomic dysreflexia could occur in children with lesions above the splanchnic sympathetic outflow at thoracic level 5-6, and present with imbalanced reflex sympathetic discharge, occurring after the beginning of spinal shock resolution. This presents as systemic hypertension, headache and reflex bradycardia in response to noxious sensory stimuli such as urinary retention or muscle spasm below the lesion\(^8\). Diagnosis of ATM involves excluding other causes of spinal cord dysfunction and demonstration of spinal cord inflammation as an enhancing spinal cord lesion or cerebrospinal fluid finding of pleocytosis (more than 10 cells /mm\(^3\)) or increased immunoglobulin type G index and time to maximum disability greater...
than 4 hours to less than 21 days. Treatment involves care of airway, breathing and circulation, initial spinal immobilization if there is history of trauma and bladder catheterization if there is urinary retention. Specific treatment in noninfectious immune mediated ATM includes intravenous methyl prednisolone 30 mg/kg/d (max. 1 g/d) for 5 to 7 days, followed by an oral corticosteroid taper starting at 1 mg/kg/d over 3-4 weeks. Prognosis in children is reported as complete recovery in 33% to 50% of patients, with significant deficits in others.

b) **Infectious Myelitis**
AFP due to spinal cord involvement can be caused by infectious agents such as poliovirus, non-polio enterovirus, herpes group of viruses, rabies, Japanese encephalitis among others.

**Anterior Horn Cell Poliomyelitis**
Poliovirus causes destruction of motor neurons resulting in asymmetric flaccid paralysis. Children less than 5 years are most frequently affected, only about 0.1-1% of infected individuals manifest with paralysis. The biphasic illness has initial nonspecific symptoms such as fever, fatigue, limb pain, headache followed by acute onset flaccid paralysis that is asymmetric, involving proximal more than distal group of muscles and progresses rapidly over 24 – 48 hours. Mortality is due to complications from bulbar weakness and respiratory failure, and management is largely supportive. AFP surveillance and nationwide Pulse Polio initiative have however come a long way and India has being accorded “polio free” status by World Health Organisation in 2014.

c) **Compressive myelopathy**
Extramedullary tumors such as neuroblastoma, Ewing sarcoma, lymphoma, granulocytic sarcoma and spinal cord abscesses, hematoma could present with mass effects on the spinal cord. The constitutional symptoms vary depending on the underlying etiology.

d) **Spinal Epidural Abscess**
Spinal epidural abscess (SEA) requires special mention as prompt diagnosis and initiation of treatment is essential for optimal outcome. The classical triad of fever, back pain and neurologic deficits is found in a minority of children, and diagnosis requires high index of suspicion in a febrile child with weakness. Recent skin/soft tissue infection and trauma have been identified as risk factors. Staphylococcus aureus is the most common organism causing SEA and empirical antibiotic regimen should be broad spectrum with staphylococcal cover, and further dictated based on sensitivity patterns. Duration of intravenous antibiotic therapy should be 4-6 weeks, dependent on the clinical improvement and response to treatment. Pediatric data on surgical decompression is limited, favorable results have been reported when surgery was performed within 24 hours of neurological deficit. The length of time before initiation of treatment and the severity of neurological deficit are the factors predictive of outcome.

e) **Ischemic Myelopathy**
Anterior spinal artery infarction is a rare entity reported after trauma, aortic dissection, thromboembolism. Absence of CSF pleocytosis and normal protein levels help to distinguish this entity from inflammatory myelopathy. Diffusion weighted sequences demonstrate restricted diffusion in the distribution of a T2 hyperintense lesion.

3) **Trauma to Spinal Cord**
Spinal cord can be injured as a result of falls, motor vehicle accidents, sports related injuries or abuse. The common levels of injury are occiput-C1 and C1-C2 in children under 8 years age, lower cervical region in older children and thoracic spine in all ages. Spinal cord trauma should be suspected in all patients with motor vehicle collision, fall from height > 10 ft, child abuse, neurological deficits, unexplained hypotension, spine tenderness, or evidence of encephalopathy/intoxication precluding clinical examination. Immediate priorities in management include airway, breathing and hemodynamic stabilization,
and spine immobilization. Children with neurogenic shock are treated with vasopressors such as dopamine, norepinephrine in addition to fluid resuscitation. Whenever there is evidence of spinal cord injury, early MRI should be performed to determine the need for surgical decompression and spine stabilization. High dose corticosteroids are often used within 8 hours after injury despite lack of concrete evidence.

4) Myasthenia Gravis
Myasthenia gravis (MG) is a disorder of neuromuscular junction of voluntary skeletal muscles and characterized by easy fatigability and fluctuating weakness. Clinical history, a decremental response to repetitive nerve stimulation, electromyography and demonstration of antibodies to Acetylcholine receptor are diagnostic. Myasthenic crisis is an exacerbation of myasthenic symptoms resulting in respiratory failure from either involvement of upper airway muscles or diaphragm and respiratory muscles. It is precipitated by infections, surgery, initiation of steroids, rapid tapering of immunotherapeutic drugs and certain medications such as aminoglycosides, beta blockers, vancomycin etc. Cholinergic crisis occurring with overdose of anticholinesterase drugs can also result in severe weakness and have additional features of cholinergic excess such as bronchorrhea, salivation, lacrimation, diarrhea, sweating, pupillary constriction, muscle fasciculations.

During acute exacerbation, treatment with cholinergic drugs should be withheld, potential triggers should be identified and treated. Use of noninvasive ventilation (bilevel positive airway pressure) may prevent the requirement for intubation. Plasmapheresis and IVIg in a dose of 2g/kg over 2 to 5 days have a role in treatment of acute exacerbations.

5) Traumatic Neuritis
Traumatic neuritis is considered when there is history of injection, usually less than 24 hours prior, with limb involvement ipsilateral to the injection site. Pain at gluteal region and hypothermia of the affected limb are usually present. Presence of sensory deficits and absence of CSF pleocytosis differentiates the entity from poliomyelitis. Management is largely supportive.

6) Hypokalemic Paralysis
Hypokalemic paralysis is an important reversible cause for AFP, especially in a young child. Diarrheal diseases, renal tubular acidosis and hyperaldosteronism need to be ruled out before the rarer primary channelopathies are considered. ECG monitoring is essential to prevent fatal cardiac arrhythmias. Correction of potassium rapidly reverses the paralysis, and must be followed with identification and treatment of the primary disease.

7) Critical Illness Neuromuscular Abnormalities
Children admitted in PICU could develop flaccid paralysis, respiratory muscle weakness and ventilator dependency secondary to critical illness polyneuropathy (CIP) and myopathy (CIM). Risk factors identified include sepsis, systemic inflammatory response syndrome, multiple organ failure, corticosteroids, neuromuscular blockers, parenteral nutrition, hyperglycemia. Nerve conduction studies, electromyography and tissue biopsy could help in differentiating, however the two entities also occur in combination. No specific therapy is recommended, early physiotherapy, good nutrition, aggressive treatment of sepsis and control of risk factors are the preventive options.

General Care of Child with Flaccid Paralysis in PICU
Children with AFP who develop respiratory muscle weakness, bulbar weakness, evidence of autonomic instability and those with rapid progression require transfer to intensive care facility. General care is directed towards anticipation and prevention of complications arising from immobilization, prolonged ventilation and ICU stay of the children with neuromuscular weakness. Good handwashing, strict asepsis policies during intravenous, urinary catheter care and tracheal suctioning are essential to prevent healthcare associated infection in these children.
Positioning
In a child with flaccid paralysis, proper positioning and padding of limbs is important to prevent nerve compression. Principal sites at risk for nerve compression are the ulnar nerve at the posteromedial aspect of elbow and peroneal nerve at the anterolateral aspect of knee, and these areas should be cushioned. A trochanteric roll helps to prevent peroneal nerve compression and foot drop. Frog positioning of limbs (arms at the side, elbows partially flexed slightly, wrists cocked up in functional position, leg rotated externally and draped over pillows, knees flexed and heel off the bed surface) provides comfort to the patient.

Prevention of Bed Sores
These children are at increased risk of pressure sores, and require regular inspection of occiput, sacrum, heels, shoulders and other pressure points for evidence of injuries. They should be turned and repositioned every 2 hours or more frequently as judged by clinical needs. Use of water or air mattresses to prevent pressure sores is recommended. A conscious child should be repositioned to keep him comfortable, during the night frequency of turning could be reduced to facilitate uninterrupted sleep.

Physiotherapy
During the acute stage, gentle passive range of motion exercises reduce the risk of contractures and deep venous thromboses. During recovery, repetitive active-assisted exercises without exhausting the child help to reduce the progression of disuse atrophy.

Nutrition
Early provision of enteral nutrition is crucial, nasogastric feeding is recommended in children with swallowing difficulty. Parenteral nutrition is reserved for children with ileus.

Bowel Care
Constipation occurs due to autonomic involvement, altered gut motility, prolonged immobilization and use of opiate analgesics. Daily monitoring of stool frequency, bowel sounds are essential.

Bladder Care
Urinary retention could be encountered especially in children with myelopathy. Bladder catheterization should be performed as a part of general nursing care, to prevent distension and avoid patient discomfort, and as a prophylaxis measure against autonomic dysreflexia. A sterile, closed bladder drainage system should be used and the risk of urinary infection should be balanced against the benefit.

Pain Relief
Pain could result from a variety of reasons such as pressure sores, bladder distention, constipation, muscle spasms, myalgia among others. Paracetamol and non-steroidal analgesics are used as first line drugs after avoidance of triggers. Narcotic analgesics are reserved for intermittent use in severe cases. Gabapentin and carbamazepine are used for neurogenic pain.

Psychosocial Support
Psychological and emotional support to the child and caregiver are an essential part in the management of children with prolonged intensive care stay. Measures such as family participation, enhancing cognitive development, increased interaction with environment outside of the intensive care unit could meet the psychosocial and developmental needs of the children.

Respiratory support
Respiratory failure in children with AFP occurs due to respiratory muscle weakness resulting in alveolar hypoventilation, poor cough clearance, and poor airway protection from aspiration.

Noninvasive Ventilation (NIV)
NIV provides positive pressure support with help of nasal masks or prongs and helps to prevent atelectasis. Expiratory positive airway pressure (EPAP) helps to overcome upper airway obstruction and maintain
alveolar recruitment, and inspiratory pressure provides effective tidal volumes. The evidence for use of NIV in children with neuromuscular diseases is based on observational studies, and the effectiveness should be evaluated on each child as it is used. The main barriers for use of NIV are major air leaks around interface, patient cooperation, and additionally in children with AFP, the presence of airway instability and failure to clear secretions which may necessitate invasive ventilation.

**Intubation**
Intubation is indicated for
a) airway protection in children with bulbar weakness to prevent aspiration,

b) children progressing to respiratory muscle paralysis and
c) to optimize pulmonary toilet.

Elective intubation is preferred to emergency procedure, and intubation after respiratory arrest has been associated with anoxic encephalopathy. Neuroumscular blocking drugs should be avoided especially in children with myasthenia, as there can be a prolonged paralysis.

**Invasive Ventilation**
In children with intact respiratory drive, synchronized intermittent mechanical ventilation, pressure support or a combination of both are appropriate. Gradual weaning could be begun once the respiratory muscles regain strength and there are no adverse pulmonary findings such as atelectasis. Weaning can be accomplished by reducing mandatory breaths or by increasing the time duration of spontaneous breaths off ventilator. A vital capacity of >10 ml/kg and a maximum inspiratory force of atleast 20 cm H2O indicate the ability to wean. Respiratory fatigue should be avoided and full ventilator support may be used at night times to provide rest. With increasing muscle strength, the patient can be kept on continuous positive airway pressure or T piece for increasing periods of time. Clinical assessment of bulbar muscle weakness is important prior to extubation consideration.

**Tracheostomy**
Tracheostomy is performed in children with anticipated prolonged ventilation, especially in those ventilated for more than 14 days. Tracheostomy offers various advantages such as increased mobilization, comfort, airway safety, participation in swallowing, speech activities and help in weaning from ventilation.

**Conclusion**
AFP is a broad clinical syndrome with numerous diagnostic possibilities. It is a medical emergency as these children could progress rapidly to bulbar weakness and respiratory failure, hence careful monitoring is warranted. The immediate priorities are to identify and manage respiratory, bulbar weakness, monitor for autonomic dysfunction and rapidly exclude causes such as dysaesthesia and envenomation. Emergent spine imaging is warranted in children with features of myelopathy, and once extrinsic causes are ruled out, immune mediated spinal cord lesions are treated with immunomodulatory therapy.

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**Source of Funding:** None

**References**