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- Editorial Board
FROM THE EDITOR’S DESK

The Journal Committee of IJPP sincerely thank all the Central IAP office bearers and members of the Executive board for giving us an opportunity to jointly organize the PEDICON 2004 along with IAP-Tamilnadu State Chapter and IAP Chennai City Branch at Chennai. The dedicated team of IJPP team have extended their whole hearted support and cooperation to the organizing committee in various capacities and made the PEDICON 2004 a memorable one. The conference was a resounding success and we had a good share of profit from PEDICON 2004. This will help us to obtain our own office space at Chennai, so that a lot of improvements can be done in the new premises.

This special issue will reach all the 15000 IAP members all over India including our regular IJPP subscribers. This is in response to our earlier commitment to the Central IAP who had contributed Two Lacks and Twenty nine thousand Rupees towards the corpus fund of IJPP during its inception in 1993.

From this issue the President’s page will be included. We thank Dr.MKC Nair, President IAP for contributing an article “Child Labour”. He has stressed every pediatrician’s responsibility to join hands with nation in abolishing the child labour.

This issue will focus on “Fluid and Electrolytes” management in pediatric practice. The topics and academic content were carefully chosen to suit our readers by Dr.Malathith Sathiayasekaran our Associate Editor and Executive Board members of the Journal Committee Dr.T.L.Ratnakumari and Dr.S.Santhi. We hope the articles will be very useful, informative and serve as a desktop reference for our readers.

The editorial on ‘Fluid management in electrolyte and mineral disturbance’ by Dr.Uma Sankari Ali is very well narrated with illustrations. Dr.Sathish B Deopujari nicely written the practical aspect on ‘Maintenance fluid therapy in pediatrics’. ‘The neonatal fluid and electrolytes therapy’ is well discussed by Dr.Arvin Shenoi. With his vast experience and knowledge on Diarrheal disorders Dr.S.K.Mittal stressed well on various aspects of ‘Fluid therapy and electrolyte management in diarrheal disorders’. The ‘Fluid management in renal disorders’ is a real academic feast by Dr.M.Vijayakumar and Dr.B.R.Nammalwar. Dr.Raghupathy et al have dealt in detail on ‘Fluid and electrolytes management on various situations in endocrine disorders in pediatrics’. The fluid management for various surgical problems in children is narrated in a vivid manner by Dr.Shanbhogue et al. For the sake of easy reference the composition of commercially available IV fluids and stock solutions is tabulated neatly by Dr.T.L.Ratnakumari, Dr.S.Santhi and Dr.Malathi Sathiayasekaran. In radiologist talks to you section, abdominal masses is discussed in detail by Dr.Vijayalakshmi et al.

The article on ‘Management of multiple injuries in children’ by Dr.KLN Rao et al will be of immense use and serve as reference guide to practitioners. Dr.Malathith Sathiayasekaran and Dr.So Shivbalan have enumerated the various laboratory tests in the evaluation of the liver.

We thank all the contributors of various articles in this issue. We welcome suggestions and guidance from our readers.

Dr. A.Balachandran
Editor-in-Chief
CHILD LABOUR

The UN Declaration stipulates that the child shall be protected against all forms of neglect, cruelty and exploitation. He/she shall not be the subject of traffic in any form. The child shall not be admitted to employment before an appropriate minimum age and not be engaged in any occupation or employment which will prejudice his health or education or interfere with physical, mental or moral development.

30th of April is observed world over as anti child labour day, yet the reality is that their rights are often violated, their aspirations ignored, their hopes blighted and their dreams shattered. Despite progressive laws and court orders to protect them, millions of Indian children are still the victims of circumstances. For understanding the issue in some detail, the child population can be grouped into three categories - school going children, child labour and nowhere children (No labour and non school goers). According to National Sample Survey data the last category of nowhere children are 35 % and they are potential child labourers.

Definition: Child Work Vs Child Labour

Child work refers to children’s participation in economic activity - that does not negatively affect their health and development or interfere with education. Work that does not interfere with education (light work) is permitted from the age of 12 years under the International Labour Organization (ILO) Convention 138. Child labour is more narrowly defined and refers to children working in contravention of the above standards. This means all children below 12 years of age working in any economic activities, those aged 12 to 14 years engaged in harmful work, and all children engaged in the worst forms of child labour.

The worst form of child labour involves children being enslaved, forcibly recruited, prostituted, trafficked, forced into illegal activities and exposed to hazardous work. They may be trafficked (1.2 million), forced into debt bondage or other forms of slavery (5.7 million), into prostitution and pornography (1.8 million), into participating in armed conflict (0.3 million) or other illicit activities (0.6 million).

Bonded labor takes place when a family receives an advance payment to hand over a child—boy or girl—over to an employer. In most cases the child cannot work off the debt, nor can the family raise enough money to buy the child back. The workplace is often structured so that “expenses” and/or “interest” are deducted from a child’s earnings, in such amounts that it is almost impossible for a child to repay the debt. Millions of girls work as domestic servants and unpaid household help and are especially vulnerable to exploitation and abuse. However, the vast majority of child labourers—70 percent or more work in agriculture.

Prevalence and the social milieu

It may be totally shocking but absolutely true that child labour contributes to 20 % of the national product (GNP) and subsidizes higher education for privileged children. A country like India, which cannot find jobs for 60 million adults, can find space for 111 million child labourers. An estimated 246 million children are
engaged in child labour. Of those, almost three-quarters (171 million) work in hazardous situations or conditions, such as working in mines, working with chemicals and pesticides in agriculture or working with dangerous machinery. They are everywhere but invisible, toiling as domestic servants in homes, labouring behind the walls of workshops, hidden from view in plantations.

According to the Indian census of 1991, there are 11.3 million working children under the age of fourteen years in India. Over 85% of this child labour is in the country’s rural areas, working in agricultural activities such as farming, livestock rearing, forestry and fisheries. The number of working children was estimated at around 17 million according to the 43rd round of the National Sample Survey (1987). In India, Kerala has the lowest incidence of child labour and Andra Pradesh the highest. 82.5% child labour is in the agrarian sector. Apart from agriculture and construction work, children are mostly engaged in forest operations, beedi making, weaving and brick kilns in rural sector and hotels and eating houses, garages and workshops in the urban sector. It was in the late eighties that attention was drawn to the visible, unpaid and unrecognized work being done by rural women within their families, a substantial amount of it being contributed by girl children.

**Labour laws**

Several laws have been passed with regard to child labour, the earliest one being the Factories Act of 1881 and the most recent one being the Child Labour Prohibition Act of 1986. India’s first act on the subject was the enactment of the Children (Pledging of Labour) Act of February 1933. The Employment of Children Act followed this in 1938. Subsequently, twelve additional legislations were passed that progressively extended legal protection to children. Provisions relating to child labour under various enactment such as the Factories Act, the Mines Act, the Plantation Labour Act etc. have concentrated on aspects such as reducing working hours, increasing minimum wage and prohibiting employment of children in occupations and processes detrimental to their health and development.

The Child Labour (Prohibition & Regulation) Act 1986 of India was the culmination of efforts and ideas that emerged from the deliberations and recommendations of various committees on child labour, the significant among them being National Commission on Labour (1966-69), Gurupadaswamy Committee on Child Labour (1979), and the Sanat Mehta Committee (1984). The Act aims to prohibit the entry of children into hazardous occupations and to regulate the services of children in non-hazardous occupations.

**Eradication of child labour**

Though restrictions on child labour exist in most nations, many children do participate in the work force. This vulnerable state leaves them prone to exploitation. They endure work conditions which include health hazards and potential abuse. However, there are problems in proceeding with the intuitive solution of immediately abolishing child labour in order to prevent such abuse. First, there is no international agreement defining child labour, making it hard to isolate cases of abuse, let alone abolish them. Second, many children may have to work in order to be able to attend school and hence abolishing child labour may only hinder their education. Problem with complete abolition of child labour is that education and employment for children are not mutually exclusive.

The Government of India is determined to eradicate child labour in the country. The world’s largest child labour elimination program is being implemented at the grass root level in India, with
primary education targeted for nearly 250 million. In this a large number of non-governmental and voluntary organizations are also involved. Special investigation cells have been set up in states to enforce existing laws banning employment of children in hazardous industries.

It is heartening to note that the Hon’ble Prime Minister of India had declared in 1994, on the eve of the Independence Day that child labor would be abolished in hazardous occupations by the year 2000, reflecting a national consensus and commitment. It is also true that after this declaration, several far-reaching initiatives have been taken by the Government to effectively tackle the problem. In this context it may be relevant to point out that Kannur District in Kerala is tipped to become the first child labour free district in India. Efforts for eradicating child labour in Kannur are reaching the final leg, with the department of labour deciding to get a final survey done by the department of economics and statistics.

Policy aspects

Unicef has taken many concrete steps to uphold child rights and naturally have plans to assist in setting up a model ‘Child Care Institute’ in each of the states for developing a model institute for training of functionaries. Vocational training for destitutes have been set up with the assistance of Unicef by SOS Children’s Village association. Policy prescription for eradicating child labour must include some income-generating programs for parents, providing children with jobs suitable to their age and also bringing awareness among employers to treat them humanely. Establishment of compulsory education for children is a prerequisite for the reduction and the abolition of child labour. It may be noted that non-coercive means can be used for promoting compulsory education. Weekend schools should be started to educate these children in the workforce.

Government of India’s policy consists of three complementary measures:

- Legal action plan: This policy envisages strict enforcement of the provisions of the Child Labour (Prohibition & Regulation) Act, 1986 and other child-related legislation.

- Focus on general development programs benefiting children wherever possible: The policy envisages the development of an extensive system of non-formal education for working children withdrawn from work and increasing the provision for employment and income generating schemes meant for their parents. A special cell - Child Labour Cell - was constituted to encourage voluntary organizations to take up activities like non-formal education, vocational training, provisions of health care, nutrition and education for working children.

- Area specific projects: To focus on areas known to have high concentration of child labour and to adopt a project approach for identification, withdrawal and rehabilitation of working children.

As a responsible and responsive citizen of India and as the advocate for the children as a whole and specially for the child in need, each one of us - pediatricians have a responsibility to participate in this national effort, offering our expertise at least in the health sector. It may be a most humbling experience to realize that the amount of food that we waste during meals may be enough to feed another hungry child, giving him/her an option to attend the school and not join the work force. It may not be possible for any one of us to get at it individually, yet every IAP local branch could spearhead a mission to reduce child labour in your locality, in association with well meaning NGOs.

Dr. MKC Nair
IAP National President 2004.
FLUID MANAGEMENT IN ELECTROLYTE AND MINERAL DISTURBANCES

Electrolyte and mineral disturbances are frequently encountered in both critical and chronic illnesses. The type of disturbance may provide an important clue to the nature of the underlying disease e.g. hypernatremia with polyuria may suggest the presence of central or nephrogenic diabetes insipidus, hyperkalemia suggests the presence of renal failure whereas hypocalemia may indicate the presence of rickets or hypoparathyroidism. Besides being pointers to the presence and nature of the underlying diseases, electrolyte disturbances may be responsible for much of the symptomatology; lethargy, somnolence and seizures may be seen with hyponatremia, muscular weakness and abdominal distension with hypokalemia and irritability and jitteriness with hypocalemia. While many of the electrolyte disturbances may be asymptomatic, some others can be life threatening.

Hyponatremia

Hyponatremia is the commonest electrolyte disturbance encountered in acutely ill children. It is defined as serum sodium less than 135 mEq/L.

Pathophysiology

As sodium is the predominant cation of the ECF and behaves as an impermeant ion due to the Na K ATP ase pump on the cell membrane, it is the major determinant of the tonicity of the ECF. Therefore disturbances of sodium levels in the serum can have profound effects on cellular volume. When serum sodium falls, the serum becomes hypotonic relative to the ICF. Water moves from the ECF into the ICF to maintain osmotic equilibrium leading to cellular edema. Although this occurs in all cells, it has disastrous consequences in the central nervous system which is encased in a rigid case. The swelling of CNS cells gives rise to cerebral edema with risks of herniation.

In contrast, when hyponatremia develops slowly, the cells have time to adapt to the hypotonic external milieu. The cells extrude osmotically active particles to protect themselves against swelling. Hence, a slowly developing hyponatremia may be relatively asymptomatic even when the serum sodium falls to very low levels.¹,²,³

Causes

Hyponatremia may develop due to either net loss of sodium or net gain of water. Losses of sodium may be external through the extrarenal or renal route. In addition, there may be hypovolemic hyponatremia without external losses because of third spacing of body fluids. Gain of water may be seen in oedematous states or in euvolemic states with inappropriate secretion of ADH. (Table1)

Diagnosis

The diagnosis of hyponatremia requires

1. Exclusion of pseudohyponatremia
2. Exclusion of factitious hyponatremia
3. A careful clinical evaluation for a possible etiology
4. Assessment of the volume status
5. Measurement of urinary sodium levels
6. Measurement of serum osmolality

**Pseudohyponatremia:** is a methodology dependent artifact that may be seen when the measurement of sodium is done by flame emission spectrometry (FES). FES measures the sodium in the total serum i.e. serum water plus the solids whereas the true sodium is present only in the water component. Normally, the serum solids constitute just 7% of the total sodium; hence serum sodium measured by FES is close to the value of sodium in the serum water. However, when the serum solids are increased as in hyperlipidemia or hyperproteinemias the serum solids may occupy >10% of the serum. Therefore serum sodium although normal in the aqueous phase may appear falsely low because it is measured in the total serum. When serum sodium is measured using ion specific electrode (ISE) measurements are made directly in the serum water. Thus the measured sodium is the true sodium and no corrections need to be applied for elevated levels of lipids or proteins. Most modern laboratories use ISE.  

**Factitious hyponatremia:** When serum osmolality is elevated because of the presence of osmotically active particles other than sodium such as glucose, urea or mannitol, water is drawn out from the ICF into the ECF thus lowering the serum sodium. Although the serum sodium is low, the serum osmolality is high. For each 100mg/dl of blood glucose elevation sodium drops by 1.6 mEq/L. As the blood glucose falls the serum sodium gradually returns to normal.

**Table 1. Causes of hyponatremia**

<table>
<thead>
<tr>
<th>Loss of sodium</th>
<th>Gain of water</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypovolemic hyponatremia</strong></td>
<td><strong>Hypervolemic hyponatremia</strong></td>
</tr>
<tr>
<td><strong>Extrarenal losses</strong></td>
<td>Nephrotic syndrome</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Cirrhosis of liver</td>
</tr>
<tr>
<td>Sweat</td>
<td>Congestive cardiac failure</td>
</tr>
<tr>
<td><strong>Renal losses</strong></td>
<td>Acute renal failure</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Chronic renal failure</td>
</tr>
<tr>
<td>Recovering ATN</td>
<td><strong>Euvolemic hyponatremia</strong></td>
</tr>
<tr>
<td>Post-obstructive diuresis</td>
<td>Pain</td>
</tr>
<tr>
<td>Salt losing nephropathy</td>
<td>Stress</td>
</tr>
<tr>
<td>Bartter’s syndrome</td>
<td>Anxiety</td>
</tr>
<tr>
<td>Congenital adrenal hyperplasia</td>
<td>Drugs</td>
</tr>
<tr>
<td>Adrenal insufficiency</td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Hypoaldosteronism</td>
<td>Cortisol deficiency</td>
</tr>
<tr>
<td>Cerebral salt wasting</td>
<td>SIADH</td>
</tr>
<tr>
<td><strong>Third spacing</strong></td>
<td><strong>Euvolemic hyponatremia</strong></td>
</tr>
<tr>
<td>Peritonitis</td>
<td>Pain</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Stress</td>
</tr>
<tr>
<td>Ileus</td>
<td>Anxiety</td>
</tr>
<tr>
<td>Necrotizing enterocolitis</td>
<td>Drugs</td>
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<tr>
<td></td>
<td>Hypothyroidism</td>
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<tr>
<td></td>
<td>Cortisol deficiency</td>
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<td></td>
<td>SIADH</td>
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In pseudohyponatremia the serum osmolality is normal and in factitious hyponatremia it is high despite low serum sodium. In all other causes, hyponatremia is associated with hypoosmolality. In both these entities, therapy does not specifically address the sodium levels.

A careful clinical evaluation with attention to the volume status and the urinary sodium values helps to arrive at the right cause of hyponatremia (Fig 1).

**Symptoms**

Much of the symptoms of hyponatremia are non-specific and can lead to a mistaken diagnosis of a primary CNS disorder or a worsening of the primary neurological problem. This may lead to a number of unnecessary imaging studies that are not only futile but may lead to a fatal delay in diagnosis. A high index of suspicion coupled with early electrolyte estimation in children with unexplained CNS symptomatology may be life saving as timely intervention can be instituted (Table 2).

**Treatment**

**Acute hyponatremia:** The treatment of acutely developing hyponatremia depends on the cause, the rapidity of the fall, the severity of the hyponatremia and the presence or absence of symptoms. Serum sodium above 130 mEq/L is generally asymptomatic even when it develops acutely and does not require rapid correction.

---

**Fig 1. Differential diagnosis of hyponatremia**

* Although the effective circulating volume is low, the child may not look dehydrated
** Acute tubular necrosis
# Congenital adrenal hyperplasia
When serum sodium falls rapidly to less than 125 mEq/L cerebral edema is generally present. When CNS symptoms result, the serum sodium should be corrected rapidly with hypertonic saline to ameliorate the cerebral edema. 10 to 12 ml/kg of 3% NaCl should be given over a period of 2 to 4 hours. 1 ml of 3% NaCl = 0.5 mEq of sodium. Once the acute symptoms have been taken care of, the serum sodium should be corrected more gradually over a period of 48 hours.

In cases of hypovolemic hyponatremia, the sodium required is calculated as 0.6 x body weight x (135 – serum sodium). This should be given over 48 hours. Serum sodium should be increased at a rate of 0.5 mEq/L per hour or not more than 12 mEq/day.

Hypervolemic hyponatremia occurs due to water gain and not due to sodium loss. In fact the total body sodium is high despite the low serum sodium. Administration of sodium would result in worsening of edema and more fluid overload. The treatment here would consist of fluid restriction. Fluid administered in edematous hyponatremia should be less than the insensible water loss plus urine output. The fluid excess can be calculated as follows: TBW x [1 – measured sodium/ expected sodium]

For example a 20 kg edematous child with serum sodium of 120 mEq/L would have a fluid excess of 0.6 x 20 [1 – 120/140] = 1.7 L. If this child’s insensible water loss is 300 ml and Urine output 300 ml, restricting his fluid to 600 ml per day will prevent further lowering of serum sodium. However, to correct hyponatremia fluid administered should be less than 600 ml. If this child is given only 300 ml of fluids daily he will sustain a negative fluid balance of 300 ml. Serum sodium will gradually return to normal over 6 days. 300 ml x 6 = 1800 ml negative balance. If in addition he is given furosemide to which the child responds by increasing his UO to 600 ml he will increase his serum sodium to normal levels within 3 days provided the fluid administration is kept at 300 ml/day.

Euvolemic hyponatremia as in SIADH

Restrict fluids to insensible water losses
Give fluids as isotonic saline.
Do not administer hypotonic fluids
Administer furosemide to improve free water clearance.

If serum sodium is less than 120 mEq/L and is accompanied by CNS symptoms administration of 3% NaCl would be needed to counteract the cerebral edema.

Chronic hyponatremia: In slowly developing hyponatremia the CNS has had time to adapt to the hypotonic milieu. The cells do so by extruding osmotically active particles and lowering intracellular osmolality. Rapidly correcting serum sodium to normal levels in such a situation will restore normal ECF tonicity. However as sodium cannot enter the cells, the ICF will be unable to increase its osmolality so rapidly. The osmotically challenged cells of the CNS undergo a characteristic change called osmotic demyelination that can lead to pseudo bulbar palsy, quadriaparesis and terminate in death. These changes are seen as central pontine myelinolysis at autopsy or on magnetic resonance imaging (MRI).

Table 2. Symptoms of hyponatremia

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea</td>
<td>Aphasia</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Echolalia</td>
</tr>
<tr>
<td>Lethargy</td>
<td>Seizures</td>
</tr>
<tr>
<td>Somnolence</td>
<td>Respiratory irregularities</td>
</tr>
<tr>
<td>Muscle cramps</td>
<td>Cheyne Stokes respiration</td>
</tr>
<tr>
<td></td>
<td>Coma</td>
</tr>
</tbody>
</table>
Therefore, all asymptomatic hyponatremias and slowly developing hyponatremias should be corrected gradually by not more than 12 mEq/L/day to prevent CNS damage secondary to treatment.

**Hyponatremia**

Serum sodium above 150 mEq/L is diagnostic of hyponatremia. It occurs much less commonly than hyponatremia. When it occurs, it is most commonly due to increased free water losses and occasionally due to a true gain in sodium.

**Pathophysiology:** An acute increase in serum sodium increases serum osmolality and makes it hypertonic. This leads to fluid shifts from the ICF to the ECF in order to maintain osmotic equilibrium resulting in shrinkage of cells. In the CNS this shrinkage can lead to a tear of the bridging vessels resulting in subarachnoid, subdural or intracerebral bleeds and sometimes venous thrombosis. When hyponatremia develops more gradually the cells generate osmotically active particles as a volume protective strategy and prevent fluid loss from the ICF. Some of these idiogenic osmoles have been identified as myo-inositol, taurine, glutamine, glycerophosphorylcholine and betaine. Thus a slowly developing hyponatremia may not manifest severe symptoms.\(^3,4\)

**Causes**

1. **Losses of free water in excess of sodium** seen in GI losses due to diarrhea. This is most commonly seen in infants who still have renal immaturity that limits their concentrating capacity, especially when they are fed high solute containing fluids leading to losses of free water in excess of solutes. Infants fed inappropriately prepared oral rehydration solutions are at special risk.

2. **Pure free water deficits** may be due to evaporative losses through the skin as in extremely premature babies under radiant warmers who may have large insensible water losses through the highly immature skin. Exclusively breast fed term neonates when there is lactation failure or inadequate fluid intake in hot weather.

**Renal losses** Absence of ADH or non responsiveness to ADH as in central diabetes insipidus (DI) or nephrogenic DI.

3. **Hyponatremia due to excess sodium** intake is rarer and is clinically associated with euvoolemia or fluid overload. It can occur due to accidental or intentional salt poisoning, sea water drowning, or iatrogenically due to the administration of excessive hypertonic saline or sodium bicarbonate.\(^4,6\)

**Symptoms:** Symptoms of hyponatremia consist of marked thirst, irritability, excessive crying, tremulousness, drowsiness alternating with irritability, seizures, focal deficits and coma.

**Diagnosis:** The cause of hyponatremia can be arrived at by a careful clinical evaluation with specific attention to the volume status, the urine output and the urinary osmolality or specific gravity (Fig 2).

**Treatment:** Children who are volume overloaded and hyponatremic due to excess sodium intake require omission of sodium administration and enhancement of sodium excretion by the use of hypotonic fluids along with diuretics in milder cases and by dialysis in severe cases.

In the more common hyponatremic dehydration which is due to free water deficit, correction of the hyponatremia should be by slow gradual rehydration. The fluid deficit should be corrected over 48 hours or longer. The maintenance fluid can be normal or slightly restricted.

Volume of fluid to be administered over 48 hours equals.
2 x Maintenance fluid + Calculated deficit to be given over 48 hours in 48 equal hourly instalments. No fluid boluses should be given unless the child is in shock.

The rate of fall of serum sodium should be not more than 0.5 mEq/L/hour or not more than 12 mEq/L/day.

Free water deficit = 0.6 x wt x [(Measured Na / Expected Na) – 1] L

For a 10 kg child with a serum Na of 160 mEq/L, this would amount to

\[0.6 \times 10 \times [(160/140) - 1] = 0.857 \text{ L} = 857 \text{ ml}\]

This free water deficit accounts for the total deficit in pure water losses. However in children with hypernatremic dehydration due to diarrheal losses there is not only free water losses but also sodium losses. Therefore the total deficit is greater than that calculated by free water deficit alone. Thus, if the same child has dehydration estimated to be 10%, the total fluid deficit will be 1000 ml; of this 857 ml would be free water, and 143 ml would be equivalent to isotonic saline. The total sodium lost is very little and administration of the total calculated fluids as 0.2 NS may be mathematically appropriate.³,⁴,⁷

However, in the presence of severe oliguria denoting the presence of high levels of ADH with poor free water clearance, it may be safer to give fluids with a higher osmolality to prevent a rapid fall in serum sodium. The fluid administered should be 0.45 normal saline prepared by adding equal amounts on 0.9 NS with 5% glucose. The resulting fluid will therefore have 2.5% glucose with 0.45 NS. When the hypernatremia is very severe, the initial fluid can be 0.9 NS for the first few hours after which it can be replaced by ½ DNS.

---

**Fig 2. Diagnosis of hypernatremia**

UO - Urine Output; Usp gr - Urine specific gravity; UOsm - Urine Osmolality; DI - Diabetes insipidus
In polyuric children with DI the concentration of sodium in the fluid administered should not exceed 0.2 DNS. The total fluid for 24 hours should include:

Maintenance fluid + 1/2 deficit + hourly replacement of urinary losses.

The deficit may be given as free water administered orally or by nasogastric tube as a continuous infusion or as equal hourly instalments. In central DI vasopressin administration is needed and should be administered to keep urine output of 2 ml/kg/hr. Once vasopressin response is obtained as seen by a fall in urine output to normal levels, the use of hypotonic fluids should be limited to prevent the development of hyponatremia.

Complications

Several complications could occur during correction of hypernatremia. If the serum sodium is brought down rapidly the serum osmolality and tonicity gets lowered rapidly. To protect the cell volume from shrinkage the cells generate idiogenic osmoles to maintain osmotic equilibrium. During correction of hypernatremia the cells are unable to dissipate these idiogenic osmoles rapidly. As the intracellular osmolality is now high, water moves into the cells leading to cerebral edema. This may manifest as convulsions or deterioration in sensorium during correction. There may be seizures, pupillary irregularities, respiratory irregularities and even death. The occurrence of CNS worsening during therapy would paradoxically require the administration of 3% NaCl to prevent a rapid fall in serum sodium. Changes of extrapontine myelinolysis has been described on MRI in some children with hypernatremia that has been corrected rapidly. Hyperglycemia is frequently encountered in hypernatremic dehydration as the high serum sodium has an inhibitory effect on insulin secretion. Insulin should not be administered to correct this hyperglycemia as the rapid fall in glucose may precipitate cerebral edema. The glucose administration in the IV fluids can be reduced to 2.5% or the water deficit administered as free water by nasogastric tube in equal hourly instalments to prevent worsening of the hyperglycemia. Hypocalcemia is another common disturbance seen during hypernatremia.

Hypernatremia is a medical emergency. Optimal monitored therapy is required to prevent CNS damage that could result in death or morbidity.

Potassium Disturbances

Potassium is the most abundant cation in the ICF. Although the total body potassium is to the tune of 50 mEq/kg, only 2% is in the ECF. The bulk is in the intracellular compartment. This large transmembrane gradient is important for maintaining the normal resting membrane potential and is maintained by Na-K-ATP ase pump on the cell membrane. Normal serum K levels are 3.5 to 5 mEq/L.

Hyopokalemia

This is defined as serum K of less than 3.5 mEq/L

Causes: Almost all dietary articles contain potassium. Dietary deficiency of potassium therefore occurs mainly in protein energy malnutrition. GI fluids are rich in potassium. Hence GI losses often lead to hypokalemia. Potassium excretion is mainly through the kidneys. Excessive losses through the kidneys may occur due to diuretic therapy, osmotic diuresis, and in ketonuria. It may also occur in renal tubular acidosis, Bartter’s syndrome and with steroid therapy. In all the above conditions hypokalemia is associated with a decrease in total body potassium.

Hypokalemia without a decrease in total body potassium can occur due to transcellular
shifts of potassium. This may occur transiently in response to the administration of glucose, insulin, catecholamines and bicarbonate which favour the movement of potassium into the cell.\textsuperscript{9,10}

**Diagnosis:** The clinical history coupled with the blood pressure evaluation along with the analysis of urinary potassium and chloride levels as well as the acid base status helps to arrive at the right diagnosis (Table 3).

**Symptoms:** Hypokalemia can lead to several symptoms. The gastrointestinal tract, the neuromuscular and the cardiovascular systems are the main targets of this deficiency. Symptoms include muscular weakness, hypotonia, proximal muscle weakness, abdominal distension, paralytic ileus, bradycardia and arrhythmias. Quadriplegia with respiratory paralysis occurs occasionally. Characteristic ECG changes may be seen in the form of low voltage QRS complexes, depressed ST segments, flattened T waves and the appearance of u waves.\textsuperscript{9,10}

**Treatment:** Hypokalemia needs to be corrected slowly; orally if feasible; if not, then parenterally. The daily potassium administration should not exceed 4 mEq/kg/day. If given IV it should be suitably diluted to avoid cardiotoxicity. The maximum concentration that can be given safely without ECG monitoring is 40 mEq/L. Concentrations up to 80 mEq/L can be given if required under continuous ECG monitoring in the ICU.

Rapid correction may sometimes be needed in the following circumstances. When hypokalemia is associated with cardiac arrhythmias, bradycardia, shallow respiration, severe muscular weakness as well as in patients on digoxin and in critically ill children at risk for arrhythmias. 0.5 to 1 mEq/kg can be given suitably diluted over 1 hour with continuous ECG monitoring.

**Table 3. Differential diagnosis of hypokalemia**

<table>
<thead>
<tr>
<th>Normal BP</th>
<th>High BP</th>
</tr>
</thead>
<tbody>
<tr>
<td><em><em>U K</em> &lt; 20 mEq/L</em>*</td>
<td><strong>High renin</strong></td>
</tr>
<tr>
<td>Low bicarbonate</td>
<td>Renovascular hypertension</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>Malignant hypertension</td>
</tr>
<tr>
<td>High bicarbonate</td>
<td>Renin producing tumors</td>
</tr>
<tr>
<td>vomiting</td>
<td></td>
</tr>
<tr>
<td><em><em>U K</em> &gt; 20 mEq/L</em>*</td>
<td><strong>Low renin, low aldosterone</strong></td>
</tr>
<tr>
<td>Low bicarbonate</td>
<td>CAH</td>
</tr>
<tr>
<td>RTA</td>
<td>Cushing’s syndrome</td>
</tr>
<tr>
<td><em><em>U K</em> &gt; 20 mEq/L U Cl</em>*\textsuperscript{**} &lt; 10 mEq/L</td>
<td><strong>Low renin, high aldosterone</strong></td>
</tr>
<tr>
<td>vomiting</td>
<td>Primary hyperaldosteronism</td>
</tr>
<tr>
<td><em><em>U K</em> &gt; 20 mEq/L U Cl &gt; 10 mEq/L</em>*</td>
<td></td>
</tr>
<tr>
<td>Diuretics</td>
<td></td>
</tr>
<tr>
<td>Bartter’s syndrome</td>
<td></td>
</tr>
<tr>
<td>Mg depletion</td>
<td></td>
</tr>
<tr>
<td>Extreme K depletion</td>
<td></td>
</tr>
</tbody>
</table>

\* Urinary potassium \textsuperscript{**} Urinary chloride
Potassium should never be given undiluted or as a bolus as it can cause fatal cardiotoxicity. Persistent low levels of serum potassium should raise the suspicion of accompanying hypomagnesemia which needs to be corrected to restore potassium levels to normal. Correction of concomitant hypocalcemia or metabolic acidosis should be deferred till potassium levels improve as the administration of bicarbonate will further lower serum potassium levels and administration of calcium in the presence of hypokalemia may precipitate cardiotoxicity.\textsuperscript{9,10}

**Hyperkalemia**

This is defined as serum potassium more than 5 mEq/L.

**Causes:** As the kidney is the main route of potassium excretion, any factor that impairs this ability can lead to hyperkalemia. This includes renal failure, use of drugs such as angiotensin converting enzyme inhibitors and aldosterone antagonists. Adrenal insufficiency and decreased mineralocorticoid activity is another major cause of severe hyperkalemia.

Transcellular shifts favouring movement of potassium from ICF to ECF occurs in acidosis and with the use of beta blockers with potassium moving out of the cells leading to hyperkalemia. Muscles are a rich source of potassium and crush injuries or burns lead to massive release of potassium into the ECF that may exceed the capacity of the kidney to excrete the same. Spurious hyperkalemia may be encountered due to excessive pressure or tourniquet application during blood collection. It may also be seen when the WBC counts are very high as in leukemia due to in vitro release of potassium from the cells.\textsuperscript{9,10,11}

**Clinical manifestations:** The clinical manifestations are sparse. When they occur it is generally catastrophic in the form of arrhythmias. Therefore hyperkalemia should be anticipated and children at risk should have monitoring of serum electrolytes and ECG.

ECG changes may be seen as tall T waves that may progress to widened QRS complexes, ventricular ectopics, ventricular tachycardia and ventricular fibrillation. Cardiotoxicity is more likely to occur when the serum potassium is more than 6.5 mEq/L. However, there is no strict correlation between the level of serum potassium and cardiotoxicity.

**Treatment:** The presence of renal failure invariably leads to hyperkalemia, as the kidney is the main route of potassium excretion. The rate of rise may be extremely steep when there is increased potassium liberation because of catabolic stresses due to sepsis, hemolysis, muscle injury, GI bleeding or administration of blood products. The finding of electrocardiographic changes of hyperkalemia (tall T waves) or serum potassium levels above 6 mEq/l calls for the institution of immediate cardioprotective measures. These measures protect the heart but do not remove potassium out of the body. They have to be followed by measures that get rid of potassium. These include the use of ion exchange resins which exchange Na or Ca for potassium and the institution of dialysis (Table 4).\textsuperscript{9,10,11,12}

All patients at risk for hyperkalemia should have a careful review of the fluids administered, foods and liquids consumed to avoid all intake of potassium. Drug review is absolutely essential to avoid use of drugs that may either impair potassium excretion or favour transcellular shifts into the ECF. Drugs specifically to be avoided include potassium sparing diuretics such as spironolactone, amiloride, ACE inhibitors, ACE receptor blockers and NSAIDs. The potassium sparing effect of some of these drugs such as spironolactone may persist for some days even after the drug is omitted. In the ICU setting the
use of succinylcholine should be avoided for rapid sequence intubation.\textsuperscript{11,12}

**Calcium**

Calcium is a divalent cation that plays an important role in maintaining membrane potential and in various intracellular enzyme processes. 99\% of the calcium resides in the bones. In the blood 40\% is protein bound, 10\% is complexed to citrate, phosphate or bicarbonate. 50\% exists as ionic calcium. The biological activity depends on the free or ionic calcium. The calcium level in the blood is maintained at a constant level by the actions of PTH, Vitamin D and calcitonin acting on the GI tract, bones and the kidney. Deficiency or defects in the metabolism of Vitamin D, absence or resistance to the actions of PTH are the two major mechanisms that result in hypocalcemia.

**Hypocalcemia**

**Definition:** Hypocalcemia is defined as total serum calcium less than 8.5 mg/dl in children, less than 8 mg/dl in term neonates and less than 7 mg/dl in preterm babies.

**Causes:** The causes of hypocalcemia vary with age (Table 5).

**Pathophysiology:** Low ionized calcium levels decrease the threshold of action potentials in neurons leading to hyperexcitability in both sensory and motor nerves resulting in paresthesias, tetany and even psychiatric manifestations and cardiac dysfunction.\textsuperscript{13,14}

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Onset/Duration</th>
<th>Mechanism</th>
<th>Risks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium gluconate (10%)</td>
<td>0.5 to 1 ml/kg over 5 min Can be repeated once more.</td>
<td>Immediate Lasts for 30 min.</td>
<td>Membrane stabilization</td>
<td>Cardiac monitoring or auscultation needed. Stop if HR &lt;100 Can cause asystole</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>1mEq/kg diluted over 10 to 15 min.</td>
<td>15 minutes; lasts for 60 min</td>
<td>Shifts potassium intracellularly</td>
<td>Hypernatremia, volume overload, hypertension</td>
</tr>
<tr>
<td>Glucose insulin</td>
<td>0.5-1 g/kg 0.1 u/kg as infusion over 30 min</td>
<td>30 - 120 min may last for 4 hrs</td>
<td>Shifts potassium into cells</td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>B\textsubscript{2} agonist salbutamol</td>
<td>5 10 mg nebulised</td>
<td>30 min may last for 2 hours</td>
<td>Shifts potassium into cells</td>
<td>Tremors, tachycardia</td>
</tr>
<tr>
<td>Kayexalate</td>
<td>1 g/kg PO or PR in sorbitol</td>
<td>30 to 60 min; may take up to 4 to 6 hrs</td>
<td>Exchanges Na for potassium across colonic mucosa</td>
<td>Sodium overload</td>
</tr>
<tr>
<td>Calcium - Potassium exchange resin</td>
<td>1g /kg PO or PR</td>
<td>30 to 60 mins PR 4-6 hours PO</td>
<td>Exchanges Ca for potassium across colonic mucosa</td>
<td></td>
</tr>
</tbody>
</table>

Table 4. Cardioprotective measures in hyperkalemia
Table 5. The causes of hypocalcemia at different ages

<table>
<thead>
<tr>
<th>Early neonatal hypocalcemia 48-72 hours after birth</th>
<th>Late neonatal hypocalcemia 3-7 days</th>
<th>Hypocalcemia in infants and children</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Prematurity</td>
<td>* Phosphate rich cow’s milk or formula feeding</td>
<td>* Vitamin D deficiency</td>
</tr>
<tr>
<td>* Birth asphyxia</td>
<td>* Transient hypoparathyroidism of newborn</td>
<td>* Hypoalbuminemia</td>
</tr>
<tr>
<td>* Infant of diabetic mother</td>
<td>* Magnesium deficiency</td>
<td>* Alkalosis</td>
</tr>
<tr>
<td>* Intrauterine growth retardation</td>
<td>* Hypoparathyroidism</td>
<td>* Hypoparathyroidism</td>
</tr>
<tr>
<td></td>
<td></td>
<td>* Renal failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>* Malabsorption syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td>* Pancreatitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>* Hungry bone syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td>* Hypophosphatasia</td>
</tr>
</tbody>
</table>

Clinical features: Patients with hypocalcemia may be totally asymptomatic or may present with lethargy, poor feeding, vomiting, abdominal distension, seizures, tetany, cramps, laryngospasm or apnea. The ECG may show a prolonged QTc interval.

Diagnosis: Estimation would be required not only of the total calcium but also the ionized calcium. In addition estimations of phosphorous, alkaline phosphatase, magnesium may be required. Estimation of PTH levels and levels of 25 OH D3 and 1-25 (OH) 2 D3 may also be sometimes required. (Table 6)

Treatment: Asymptomatic hypocalcemia should be treated with oral calcium supplements of 100 to 200mg/kg/day of elemental calcium. This can be given as calcium glucobionate (115 mg of elemental calcium/5ml) or calcium carbonate (250 mg of elemental calcium/5ml). Vitamin D supplements may be required in patients with renal disease and vitamin D deficiency states.

For critically ill patients with hypocalcemia intravenous therapy is indicated in the form of 10% calcium gluconate in a dose of 1-2 ml/kg IV over 3 to 5 minutes upto a total of 10 ml with cardiac monitoring. Calcium gluconate contains 9.8 mg/ml or 0.45mEq/ml of elemental calcium.

Calcium chloride 10% contains 27mg/ml or 1.4 mEq/ml of elemental calcium and so is more irritating to the veins. The short term treatment is followed by 100 to 500mg/kg of IV calcium gluconate infusions over 24 hours in neonates and 100-200mg/kg/d IV in older children with blood calcium level monitoring. If magnesium is low, 50 % magnesium sulfate may be given as 0.1 to 0.2 ml/kg IM every 12 to 24 hours as needed.

Hypercalcemia

Hypercalcemia is a state where serum calcium levels are > 12 mg/dl. Symptoms are invariably present when serum calcium levels are above 15 mg/dl but symptoms may be present even when the level is 12mg/dl.

Pathophysiology: Hypercalcemia in a person with normal regulatory mechanisms suppresses PTH. This helps to regulate serum calcium. This regulatory effect is not so effective when serum calcium levels are below 7.5mg/dl or above 11.5 mg/dl. At these levels homeostasis is dependent on direct exchange of calcium between bone and ECF. A high ECF calcium concentration impairs the ability of the renal tubule to respond to ADH. This leads to polyuria, dehydration and azotemia.
The causes of hypercalcemia vary with age (Table 7).

**Clinical features:** The predominant clinical features are nausea, vomiting, constipation, polyuria, polydipsia, dehydration and marked irritability. Weight loss is an important feature. Some children may have renal failure and rarely seizures. On examination there may be bradycardia, proximal muscle weakness or hypertension. Rare manifestations include pancreatitis, peptic ulcer disease and band keratopathy. Metastatic calcification and renal stones can also occur. At levels above 17mg/dl calcium phosphate may precipitate in the blood or tissues leading to cardiac arrest or coma.

**Diagnosis:** Besides calcium estimations, it is important to estimate phosphorus, alkaline phosphatase, PTH, 25 OH D3 and 1-25 (OH) 2 D3. Urinary calcium estimation as well as USG evaluation for the presence of nephrocalcinosis is required.

**Treatment:** The primary line of treatment is to augment urinary losses of calcium by saline diuresis coupled with IV furosemide. Fluids should be administered at 1.5 to 2 times the maintenance requirement with close monitoring of serum electrolytes and urine output.

If serum calcium is greater than 14mg/dl calcitonin 2-4 units /kg every 6 to 12 hours may

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**Table 6. Differential diagnosis of hypocalcemia**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Phosphorous</th>
<th>Alkaline phosphatase</th>
<th>PTH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D deficiency</td>
<td>L</td>
<td>H</td>
<td>H</td>
</tr>
<tr>
<td>Vitamin D dependent rickets</td>
<td>L</td>
<td>H</td>
<td>H</td>
</tr>
<tr>
<td>Hypoparathyroidism</td>
<td>H</td>
<td>N</td>
<td>L</td>
</tr>
<tr>
<td>Pseudohypoparathyroidism</td>
<td>H</td>
<td>N</td>
<td>H</td>
</tr>
<tr>
<td>Magnesium deficiency</td>
<td>H</td>
<td>N</td>
<td>L</td>
</tr>
<tr>
<td>Renal failure</td>
<td>H</td>
<td>H</td>
<td>H</td>
</tr>
<tr>
<td><strong>L</strong> low</td>
<td><strong>H</strong> high</td>
<td><strong>N</strong> normal</td>
<td></td>
</tr>
</tbody>
</table>

**Table 7. Causes of hypercalcemia**

<table>
<thead>
<tr>
<th>Neonate</th>
<th>Infants</th>
<th>Older children</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Excess calcium supplementation</td>
<td>* Subcutaneous fat necrosis</td>
<td>* Vitamin D excess</td>
</tr>
<tr>
<td>* Secondary hyperparathyroidism due to maternal hypocalcemia</td>
<td>* Vitamin D excess</td>
<td>* Primary hyperparathyroidism</td>
</tr>
<tr>
<td>* Primary hyperparathyroidism</td>
<td>* Idiopathic infantile hypercalcemia</td>
<td>* Immobilization</td>
</tr>
<tr>
<td></td>
<td>* William’s syndrome</td>
<td>* Milk alkali syndrome</td>
</tr>
<tr>
<td></td>
<td>* Blue diaper syndrome</td>
<td>* Thiazide diuretics</td>
</tr>
<tr>
<td></td>
<td>* Severe autosomal recessive hyperphosphatasia</td>
<td></td>
</tr>
</tbody>
</table>
be used. Biphosphonates such as pamidronate at 0.5 to 1mg/kg/dose over 4-5 hours can be used for 2 days. Hydration and furosemide may lower the serum calcium levels by 1-3 mg/dl within a day and biphosphonates serve to block bone resorption by osteoclasts over the next 24 -48 hours. Biphosphonates have the potential toxicity of lowering serum phosphorous and can cause low grade fever, lymphopenia, myalgia and reversible hepatotoxicity. Biphosphonates have only rarely been used in children. Oral glucocorticoids may play a role in vitamin D induced hypercalcemia by blocking GI absorption of calcium. In severe hypercalcemia, especially if associated with renal failure, dialysis may be life saving. Surgical subtotal parathyroidectomy may be required in cases of primary hyperparathyroidism. 13, 14, 15

**Magnesium**

Magnesium is the second most abundant intracellular cation in the body. Approximately 60% of the total body magnesium is located in the skeleton and about 38%in the soft tissues. Less than 2% is in the ECF. Serum concentration ranges from 1.8 to 2.5 mEq/L Approximately 33% is protein bound. The free Mg is the biologically active component.

**Definition:** Hypomagnesemia is defined as serum Magnesium <1.8 mEq/L.

**Causes:** Magnesium loss may occur in diarrhea, nasogastric suction, and inflammatory bowel disease. There may be congenital defect in magnesium absorption or in tubular reabsorption of magnesium. Increased renal losses may occur due to drugs such as thiazides, loop diuretics, aminoglycosides, amphotericin B and cisplatinum. Other situations when hypomagnesemia may occur include poorly controlled diabetes and during recovery of diabetic ketoacidosis.14,16

**Role of magnesium in the body:** Extracellularly magnesium ions block neurosynaptic transmission by interfering with the release of acetylcholine. It also interferes with the release of catecholamines from the adrenal medulla. Intracellularly almost all enzymes involved in phosphorous reactions require magnesium for activation.

**Clinical features:** Early manifestations of hypomagnesemia include nausea, vomiting, lethargy and muscle cramps. Major manifestations are as CNS and neuromuscular irritability. At levels less than 1mEq/L there may be tremors, exaggerated deep tendon reflexes, carpopedal spasm, and tetany. Mental changes include irritability, disorientation, depression and psychosis. Respiratory muscle failure may occur in severe cases. Refractory ventricular tachycardia – torsades de pointes can occur in magnesium deficiency. Magnesium deficiency may lead to hypokalemia and hypocalcemia. Therefore decrease in any one of these should prompt an evaluation of the other two electrolytes.

**Laboratory evaluation:** The most specific method of estimating total serum magnesium is by atomic absorption spectrophotometry. Ion selective electrodes to measure ionic magnesium are not yet available in clinical laboratories. ECG changes may be seen as ST depression, altered T waves, prolonged PR or widened QRS and low voltage.

**Treatment:** For mild asymptomatic deficiencies serum magnesium <1.2 mEq / L oral replacement with 10 to 20 mg/kg/day of elemental magnesium in 3 or 4 divided doses is given. In infants with hypomagnesemic tetany 0.4 to0.8 mEq/kg (5 to10 mg/kg) magnesium is given IV or IM as a 50%solution of magnesium sulphate.50% MgSO4 contains 4 mEq per ml(48mg). For severe symptomatic hypomagnesemia IV magnesium is given 1 mEq/kg/over 2 to 6 hours
on day 1 followed by 0.5 mEq/kg over 2 to 4 hours for the next 3 days. The total replacement required would be about 4 mEq/kg. In renal failure with documented hypomagnesemia, half the amount should be given. The usual concentration that can be given intravenously ranges from 5 to 20%. Maximum concentration that can be given IV is 20%. Risks of IV magnesium include hypermagnesemia, hypocalcemia and hypotension. Careful monitoring of electrolyte and hemodynamics is required during IV magnesium infusions. 14, 16

**Hypermagnesemia**

Serum magnesium levels above 2.5 mEq/L constitutes hypermagnesemia.

**Causes:** Patients in renal failure who with excessive magnesium intake from medications or magnesium containing enemas, following trauma, shock, burns or cardiac arrest as a result of rapid mobilization from soft tissues, in the initial dehydrated phase of DKA and neonates whose mothers have received magnesium sulfate for pregnancy induced hypertension.

**Clinical features:** Nausea, vomiting, lethargy, weakness and dizziness are early symptoms. Neuromuscular depression, hypotonia, absent deep tendon reflexes, respiratory depression, disorientation, hypotension and altered sensorium may be seen in more severe cases. Hypermagnesemia has a negative effect on the heart rate. Depression of the sino-atrial node and atrial fibrillation may occur. Higher Magnesium levels cause widening of QRS and can cause conduction delays. In severe cases complete heart block or asystole may occur. Vasodepression of the vascular smooth muscle may occur resulting in hypotension.

**Diagnosis:** Monitoring serum levels is important in children with renal failure who have any of these symptoms. Other electrolytes should also be measured as there may be associated hyperkalemia and hypercalcemia. BUN and serum creatinine should be estimated. In refractory hypermagnesemia thyroid function and serum cortisol needs to be estimated. ECG should be done in all cases and cardiac monitoring may be needed.

**Treatment:** IV Calcium gluconate 1 ml/kg diluted is given slowly with cardiac monitoring to antagonize the effects of magnesium at the cardiac membrane and neuromuscular level. Where the urine output is good, saline diuresis with furosemide may be given. Non-magnesium containing enemas or cathartics may be given to enhance GI clearance. Dialysis is indicated in patients with renal failure, in asymptomatic hypermagnesemia with serum levels above 8 mEq/L and in any patient with cardiovascular or neuromuscular effects of hypermagnesemia independent of serum level. 14, 16

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**References**


NEWS AND NOTES

Pediatric Intensive Care Chapter’s
6th National Congress on Pediatric Critical Care

**Date:** 30th and 31st October 2004
**Venue:** IIT, Kanpur

<table>
<thead>
<tr>
<th>Registration fee</th>
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For further details please contact: Dr. Vivek Saxena, Organising Secretary, Regency Hospital Ltd, A-2, Sarvodaya Nagar, Kanpur 208 005. Ph: 0512-2212001-05, Fax: 0512-2213407, 2295429, Email: rashmik@ncpcckanpur.com, viveks@ncpcckanpur.com
FLUID AND ELECTROLYTES

MAINTENANCE FLUID THERAPY IN PEDIATRICS

* Satish B. Deopujari  
** Sangeeta Gedam  
** Sakina Vali  
*** Prajakta Kulkarni

Abstract: It is imperative that volume and composition of extracellular fluid is maintained. The fluid and electrolyte therapy in pediatrics is based on the age, weight and surface area of an individual child. The conventional maintenance fluids may not be able to subserve basal sodium needs for increasing age and weight of the child, raising the concern of hyponatremia especially in older age group. Hence the maintenance fluid should contain 5% dextrose, 3mEq/kg/24hrs of sodium and 2 mEq of potassium/100 ml of fluid.

Keywords: maintenance fluid, sodium, potassium.

Understanding fluid and electrolyte management is thus an important component of efficient supportive care.

Applied physiology of body fluids

It is essential to know the distribution of fluids in the body for proper understanding of the topic.

Total body water (TBW): TBW content in an average built adolescent/adult is about 60% of the body weight. In an obese person the TBW is proportionately less (as water content of fat is less) as compared to a lean person. In a newborn the TBW content could be as high as 80% of the body weight.

Distribution of body fluids: TBW is distributed in the body in two major compartments i.e. the extracellular compartment and the intracellular compartment. Out of the TBW, 2/3rd is intracellular fluid (ICF) and 1/3rd is extracellular fluid (ECF) as shown in figure 1.

ICF is essentially rich in potassium (only 2% being extracellular) and serum potassium levels do not reflect total body potassium while ECF is rich in sodium.

Water and electrolytes requirements are high in infants and children as compared to adults. This is because of the increased turnover rate of fluid and electrolytes in this age group which in turn is related to high metabolic rate.

Metabolism creates two by products to be actively eliminated for maintenance of homeostasis:
Figure 1: Shows the distribution of total body water (60% of the body weight) in an adolescent boy of 45 kg.

1. Heat: It is dissipated by insensible losses from skin and lungs.

2. Solute waste products of metabolism: They are excreted into the urine. (Table 1)

To determine the daily fluid requirement of the body we need to know insensible fluid losses, which constitutes about 1/3rd of the total maintenance fluid. It is called insensible as we are unaware of it. Sweating is not considered as an insensible loss but it is important for heat regulation and can be a major source of water and solute loss.

Maintenance therapy

Maintenance intravenous fluid (MIF) is necessary considering the sensible and insensible losses of the body. MIF comprises of 5% dextrose, sodium and potassium. Provision of 5% dextrose minimizes the endogenous breakdown of proteins and fats thus preventing the formation of ketones. Maintenance therapy also subserves basal sodium and potassium needs. It does not repair fluid deficit or replace abnormal fluid losses, such as in diarrhea and vomiting. These losses need to be replaced as they occur in addition to the maintenance fluid requirement. Basic principles of parenteral maintenance fluids in children were laid down by Holliday and Segar’s landmark paper in 1957.

Classical indications for maintenance therapy

1. For children who cannot be fed enterally.
2. For preoperative and postoperative surgical patients.

Volume and composition of maintenance fluid

The conventional composition of maintenance fluid is 2 to 3 mEq of sodium and 1 to 2 mEq of potassium per 100 ml of MIF. The maintenance fluid requirement can be easily calculated depending on the body weight as given in Table 2.

Maintenance fluid contains 5% dextrose which provides 17cal/100ml i.e. approximately 20% of the daily caloric needs. The minimum amount of glucose required to prevent protein catabolism is 3g/kg/day. The solutions containing 5% dextrose provide adequate carbohydrates that

Table 1. Maintenance of homeostasis by elimination of metabolic byproducts

<table>
<thead>
<tr>
<th>Site of fluid loss</th>
<th>Proportion by site</th>
<th>Fluid</th>
<th>Electrolytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heat Skin and Respiratory tract</td>
<td>50%</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Metabolism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solute Urine</td>
<td>50%</td>
<td>All</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Maintenance fluid requirements

<table>
<thead>
<tr>
<th>Body weight (kg)</th>
<th>Fluid requirement (ml/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>3000</td>
</tr>
<tr>
<td>20</td>
<td>4000</td>
</tr>
<tr>
<td>30</td>
<td>5000</td>
</tr>
</tbody>
</table>
prevents gluconeogenesis, protein catabolism and ketogenesis. Therefore all parenteral maintenance fluids should contain 5% dextrose.

Maintenance fluids contain 3mEq/kg/day of sodium and when calculated as per table 2 approximates to 5% dextrose with 1/4th to 1/2 normal saline. Higher the body weight higher is the concentration of sodium in maintenance fluid. Potassium in maintenance fluid is 2 mEq/100ml. It is imperative that potassium should be added only after ensuring adequate urine output.

Most of the commercially available maintenance fluids are approximately 1/6th normal saline* with 5% dextrose and potassium. (*154 mEq/1000 ml of Na+ in normal saline and 25.6 mEq/1000 ml of Na+ in 1/6 normal saline)

- For a 15 kg child (see Table 2) maintenance fluid requirement for 24 hrs would be 1250 ml of 5% dextrose with 45 mEq of sodium (i.e. 3 mEq/kg/day) and 25 mEq of potassium (i.e. 2 mEq/100ml).
- For a 50 kg child maintenance fluid requirement for 24 hrs would be 2100 ml of 5% dextrose with 150 mEq of sodium (i.e. ~1/2 Normal Saline) and 42 mEq of potassium (i.e. 2mEq/100ml). (Fig 2).

### Table 2. Maintenance fluid and electrolyte requirement

<table>
<thead>
<tr>
<th>Body weight</th>
<th>Maintenance Fluid Requirement</th>
<th>Na⁺</th>
<th>K⁺</th>
<th>Glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate</td>
<td>mEq/kg/day</td>
<td>mEq/100ml</td>
<td>g/100ml</td>
</tr>
<tr>
<td>0 - 10 kg</td>
<td>4 ml/kg/hr</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>11 – 20kg</td>
<td>40 ml + 2ml/kg/hr for each kg above 10kg</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>&gt;20 kg</td>
<td>60 ml + 1ml/kg/hr for each kg above 20kg</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

**Calculation of rate of fluid infusion**

Most commonly used method for infusion of IV fluids is by conventional IV sets or microdrip sets. The following table depicts a simple practical approach for calculation of rate of fluid infusion (Table 3).

E.g. for a 10 kg child the rate of maintenance fluid is 4 ml/kg/hr i.e. 40 ml/hr = 40 micro drops/min and by the conventional I.V. set 40 ÷ 4 = 10 drops/min.

### Alterations in maintenance fluid

Maintenance fluid has to be modified as per clinical situations. Table 4 depicts the alterations in maintenance fluid.

### Limitations and problems of maintenance fluid

1. **Duration:** Maintenance fluids alone will minimize the excessive breakdown of proteins and prevent ketone production, but the patients on this regimen will actually lose 0.5 - 1% of weight each day. Thus it is imperative that patients should not be on maintenance therapy indefinitely. Some form of nutritional supplements are mandatory if maintenance therapy is to be continued beyond seven days. This is
Figure 2: Shows Glucose, Sodium and Potassium in MIF in relation to body weight. Note that with the increase in body weight, the concentration of sodium in maintenance fluid increases but that of potassium and glucose remains constant.

Table 3. Practical approach for calculation of rate of fluid infusion

<table>
<thead>
<tr>
<th>Rule</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rule 1</td>
<td>Calculate maintenance fluid rate as ml/hr.</td>
</tr>
</tbody>
</table>
| Rule 2 | For Microdrip set: ml/hr = No. of microdrops/min  
(Presumption: 1ml = 60 microdrops) |
| Rule 3 | For Conventional IV set: Divide the above figure (No. of micro drops) by 4.  
(Presumption: 1ml = 15 drops) |
| Rule 4 | Do not take the drop rate for granted, check it frequently |

Table 4: Modification of maintenance fluid in various clinical situations

<table>
<thead>
<tr>
<th>Skin</th>
<th>Increased maintenance fluid</th>
<th>Decreased maintenance fluid</th>
</tr>
</thead>
</table>
| 1. Radiant warmer (20 ml/kg/day).  
2. Phototherapy (20ml/kg/day).  
3. Fever (10-15% for each 1°C rise above normal) | Increased ambient humidity. |
| Lungs | 1. Tachypnoea.  
2. Bypassing the upper airways. | Humidification of inhaled gases. |
especially important in children who are malnourished.

2. **Other nutrients**: Maintenance fluid is lacking in such crucial nutrients as proteins, fats, vitamins and minerals. It only provides sodium, potassium and 20% of the daily caloric needs.

3. **Potassium**: There is a risk in adding potassium chloride in routine maintenance fluid without confirming the renal status. Vast majority of sick children are usually not potassium depleted and are unlikely to have significant hypokalemia. There is a risk of dangerous hyperkalemia in patients with renal disease, acidosis of varying etiology or in tissue breakdown such as in trauma, sepsis and hemolysis.

Low potassium levels are well tolerated in children over at least a short period of time.

**Controversies / Newer concepts**

**Volume**: Regarding volume fortunately there are no major controversies.

**Composition**: Sodium – There is a growing concern regarding development of hyponatremia\(^2\,3,4\) in children on the maintenance fluid therapy with 0.2 normal saline (most of the commercially available maintenance fluids). This stems from the following issues:

1. It is believed that especially in an older child the requirement of sodium is more than 0.2 Normal Saline.
2. Utilization of dextrose in maintenance fluid makes this fluid all the more hypotonic in vivo.
3. Release of vasopressin during stress (not uncommon during illness) leads to retention of free water causing hyponatremia.

Hence maintenance fluid in older child should contain 0.45% normal saline with 5% dextrose.

**Summary**

Sodium requirement is 3 mEq / kg / day while potassium requirement is 2 mEq / 100ml of MIF. Always add potassium after the patient has voided urine. This ensures that the sodium concentration in the fluid increases with increase in weight but that of potassium remains the same.

Administration of the parenteral fluid should be considered as an invasive procedure with deleterious effect which may culminate in long term morbidity and mortality. Patients on parenteral fluids need to be closely monitored.

The essence of successful management of fluid and electrolyte abnormalities is frequent clinical and laboratory monitoring of patients. The younger the child, the more frequent this needs to be done.

**Points to remember**

- The commercially available maintenance fluid may not be able to meet the sodium requirement for all the age groups. Iatrogenic hyponatremia is known to occur in the patients on maintenance therapy especially in older age group.
- Potassium and dextrose concentration in maintenance fluid is constant irrespective of the age.
- Some form of the nutritional support is necessary if maintenance therapy is to be continued for a long time.

**References**

1) Holliday MA, Segar WE. The maintenance need for water in parenteral fluid therapy. Pediatrics. 1957; 19: 823-832


3) Halberthal M, Halperin ML, Bohn D. Acute hyponatraemia in children admitted to hospital:

BOOK REVIEW

Title : Immunization for children
Authors : Dr. Niranjan Shendurnikar
Dr. Mukesh Agrawal
Review : Immunization is one of the important cost effective technologies to improve child survival. Now its scope has been extended to achieving improved quality of life for both children and adults. Information on vaccines and the research on newer vaccines is taking place at such a pace that it is difficult for the general pediatricians and practitioners to keep abreast all the time. The need for a comprehensive book on immunization with references similar to the red book of American Academy of Pediatrics was long felt. The book on ‘Immunization for children’ fulfils this need to a great extent. The topics are well chosen and the authors have presented scientific evidence for and against some of the newer vaccines. They have tried to give guidelines wherever possible. Besides, new information on conventional vaccines is also included. Newer vaccines such as pneumococcal and meningococcal vaccines and not so new vaccines such as Hepatitis B, MMR, H influenzae, varicella and Hepatitis A are covered in depth. Extensive references given from standard journals will stimulate the readers to study the pros and cons of each new vaccine and arrive at some evidence-based decision. Under the heading DPT, information on DTaP vaccine is also provided and this will ensure that the practitioners look at all aspects of any vaccine including cost-benefit, local epidemiology and feasibility before they include it in their practice. The only vaccine of topical interest, which is not covered, is Japanese encephalitis vaccine. Information on combination vaccines and Internet sources are well thought out additions.

This book is a well-compiled collection of scientific data and recommendations. Pediatricians and postgraduates will find it immensely useful and valuable for improving their knowledge and practice on immunization. We hope that periodic updated editions will be brought out to provide latest information to the readers on the ever-expanding subject of vaccinology.

Publisher : Paras Medical Publishers, 5-1-473, Jhambagh Road, PO Box No.544,
Hyderabad 500 095.

Price : Rs.125/-

CONGRATULATIONS

Dr. H. Paramesh, Past President of IAP – Respiratory Chapter had been awarded Fellowship by the Indian College of Allergy, Asthma and Immunology (FICAAI) for the year 2003.
FLUID AND ELECTROLYTES

NEONATAL FLUID AND ELECTROLYTE THERAPY

* Sandeshkiran PS
* Arvind Shenoi

Abstract: Fluid and electrolyte therapy is crucial in the care of sick and convalescing neonates. This communication discusses therapy based on the physiology of the newborn. Fluid therapy is based on the postnatal age, factors which influence fluid balance like warmers, phototherapy and certain pathologies like broncho-pulmonary dysplasia. Electrolytes are added after 48 hours. Fluid and electrolyte therapy is monitored with weight monitoring and appropriate biochemical tests. Dyselectrolytemia is diagnosed by biochemical tests and corrected rapidly if life threatening and gradually, if mild. Commercially available fluids may be used based on these principles after tailoring them to the needs of the neonate.

Key words: Infant, newborn, fluid therapy, dyselectrolytemia.

Disorders of fluid and electrolytes are among the commonest derangements encountered in preterm and critically sick neonates. This article describes important principles and specific guidelines of fluid and electrolyte management in newborns. Fluid and electrolyte management in the context of acid base disorders, calcium, magnesium and parenteral nutrition disorders are not discussed here. Fluid and electrolyte (FE) management is important because most sick neonates in a neonatal intensive care unit (NICU) require IV fluids. Appropriate fluid therapy ensures maintenance of homeostasis and assists recovery. If inappropriate fluids are administered, serious morbidity can result from fluid and electrolyte imbalances.

Well managed fluid and electrolyte balance is the cornerstone of treating sick neonates. Neonatal fluid therapy differs from that of an older child and an adult in that the fluid requirement changes with the postnatal and gestational age, therapy (e.g. phototherapy) and the underlying illness. This is because of the dramatic changes in the physiology of body fluid compartments which occurs soon after birth.

Physiology

At birth there is an increase in renal perfusion and glomerular filtration followed by diuresis. This results in loss of extracellular water and weight. After preterm birth changes in body composition are similar to a term infant. Preterm infants thus tend to lose more weight. Thereafter water and electrolyte homeostasis is regulated by the balance between losses and intake. Fluid intake is controlled by the physician and must thus be dictated by the knowledge of losses and regulatory mechanisms. The two main areas of fluid loss are the kidney and transepidermal water loss. Renal water loss is regulated by renal perfusion and anti-diuretic hormone (ADH). Renal perfusion is reduced in asphyxia, hypotension or any major systemic illness. ADH is secreted in response to rise in plasma
osmolarity, hypovolemia and hypotension, or stress (intraventricular hemorrhage, pneumothorax etc). ADH action on the distal tubule is impaired in the sick neonate. This results in relatively high losses of water and sodium in a sick neonate. This is further aggravated by renal immaturity in neonates less than 32 weeks gestation. Preterm infants less than 32 weeks gestation are also hampered by high transepidermal water loss which may be as high as 200 ml/kg/d in neonates less than 28 weeks gestation. Pathologic conditions such as sepsis induced systemic inflammatory response syndrome and necrotising entero-colitis are associated with a large amount of third space loss. These are hidden losses and are difficult to estimate clinically. Extracellular fluid losses are seen in conditions such as diarrhea, or in surgical conditions like intestinal obstructions, ileostomy losses etc. These can be measured and appropriate replacements made. Clinically changes in the fluid status can be gauged by measuring urine output, blood pressure and body weight, but dyselectrolytemias are difficult to diagnose clinically. Thus a clinician needs to have a fair idea of the patho-physiology, normal fluid balance in a neonate as well as access to a reliable laboratory.

Goals of fluid and electrolyte therapy

The aim of fluid and electrolyte therapy is to ensure a smooth transition from the aquatic in-utero environment to the dry ex-utero environment. The goal of early management is to allow initial extracellular fluid loss over the first 5-6 days as reflected by weight loss(1-3% of body weight / day), while maintaining the tonicity and intravascular volume as reflected by blood pressure, heart rate, urine output (1-3 ml/kg/hr), urine specific gravity(1005-1015)serum electrolytes(serum Na+ 135 – 145 mEq/L ,serum K+ 4-5 mEq/ L ), euglycemia(blood glucose 60-100 mg / dl). Fluid management should maintain water and electrolyte homeostasis and provide for body growth and recovery from illness.

Putting the principles into practice

The clinician has to order the correct amount of fluids based on the understanding of physiology. A few rules are:

1. All fluid requirements are calculated on the basis of the birthweight till the neonate’s weight exceeds this.
2. These guidelines are for initiating fluid therapy; urine output, weight, and if available plasma sodium, other electrolytes and osmolality guide further therapy.
3. In very small and sick neonates one would need to reassess fluid therapy as frequently as every 6 hours while for stable neonates once daily charting may be adequate.

Initiating fluid therapy: This exercise is guided by some clinical questions:

What is the postnatal age of the neonate?

In view of the physiological diuresis which occurs after birth the fluid requirements are lowest soon after birth and rise sequentially (60 ml/kg, 90 ml/kg, 120 ml/kg on day 1, 2, 3 respectively) to plateau at 150 ml/kg per day on day 4 or thereafter. There are some variations in VLBW infants as shown in table1.

Are there any factors which increase or decrease water loss?

Phototherapy and radiant warmers are the two commonest causes of increased insensible water losses. We add 20ml/kg of fluids extra for each of these. If the neonate has diarrhea one needs to factor in the increased stool water losses. Acute renal failure with oliguria is a condition which requires that total fluid therapy be restricted to 400 ml/m²/day(approximately 40ml/kg/d). Broncho-pulmonary dysplasia and patent ductus arteriosus are two common conditions
which require fluid restriction. Necrotising enterocolitis and severe sepsis have large third space losses and need fluid therapy approximating 200ml/kg/day. Surgical conditions like exomphalos may have large water losses unless appropriate care is taken to treat the primary problem. Surgical ostomies and catheters may also contribute to water losses, which need to be replaced volume per volume.

**What is the glucose requirement?**

Neonates at high risk for hypoglycemia, including all sick neonates are often the candidates for intravenous (IV) fluid therapy. They thus depend on glucose in the IV fluid as a sole source of energy. In such situations the fluid volume is often dictated by the glucose requirements. The standard intravenous fluid has 10% dextrose although one can infuse up to 12.5% dextrose through a peripheral vein. One starts with a glucose infusion rate of 4 - 6 mg/kg/min. As a rule of the thumb one starts a one day old newborn baby at 3ml/kg/hour (equivalent to 5mg/kg/min of glucose, and 72ml/kg/day of fluids). Then one checks the blood glucose levels at regular intervals and increases or decreases the glucose infusion rate as required. It is important to note that glucose and fluids are calculated independently of each other.

**When and which electrolytes do we add?**

Sodium and potassium are the two electrolytes which are added after the initial 48 hours. Term infants require about 2-3 mEq/kg/day of sodium and preterm infants 3-5 mEq/kg/day. Some extremely preterm infants require higher intakes of sodium. Potassium requirements for term and preterm infants are uniform, amounting to 2-3 mEq/kg/day. Calcium may be used in a dose of 4 ml/kg/day (36 mg/kg/day) of calcium gluconate for the first 3 days in certain high-risk situations (birth asphyxia, IDM, preterm sick neonate).

**Do we reconstitute fluids or use commercially available fluids?**

Ideally one should reconstitute fluids and have them prepared by an in house pharmacy with due asepsis. However this is far from a reality for most of us. Hence one has to manage with ready made fluids which are available commercially. The neonate is started on 10% dextrose on day 1 or 2 at the required infusion rate. The pediatric intravenous fluid contains sodium 25 mEq/L, potassium 20mEq/L, and small concentrations of chloride, magnesium, acetate and phosphorous along with 5% dextrose concentration. This can be used for most term infants after the addition of 10 ml of 50% dextrose for every 100ml. The reconstituted solution provides a dextrose concentration of 10%, and composition of 22.7mEq/L of sodium and 18mEq/L of potassium. At an infusion rate of 100 - 150 ml/kg/day this meets the requirements.
of most term and preterm infants more than 32 weeks gestation. More preterm infants however require additional sodium (2-5 mEq/kg/d). Calcium gluconate is added to this as indicated above.

**Monitoring fluid therapy**

Once intravenous fluid therapy has been initiated one has to monitor it regularly. Clinically one looks for signs of dehydration (loss of skin turgor, tachycardia etc) and overhydration but it is far better to prevent either by monitoring:

a. **Body weight:** An infant on IV fluids should lose about 1% of body weight on each day of therapy. Weight loss of greater than 2% body weight/day suggest inadequate fluids; while weight gain suggests excess fluid intake (provided the baby is not on parenteral nutrition).

b. **Urine output:** A neonate receiving adequate amounts of fluids and normal renal function passes 1-3ml of urine kg/hour. Urine output of <1ml/kg/hour suggests inadequate intake; while urine output >4ml/kg/hour may suggest overhydration or diuresis.

c. **Plasma sodium and osmolality:** The level of plasma sodium is well regulated to maintain a level of 135 - 145 mEq/L, barring in extreme preterms. Rise in plasma sodium is a good sign to detect dehydration and rarely is due to excess sodium infusion. Similarly a fall in plasma sodium is a good indicator of overhydration. Plasma osmolality is also maintained between 270 - 285 mOsm/kg. A rise in plasma osmolality relates to dehydration and a fall in osmolality suggests overhydration. However in the present scenario of lack of standardisation amongst labs, it is difficult to have fluid therapy based on plasma sodium values or osmolality alone.

d. **Other electrolytes:** One needs to measure plasma potassium and chloride in certain situations when one expects large variations in electrolyte balance (e.g. in diarrhea).

It is thus essential to take clinical assessments in conjunction with lab values from a reliable lab for monitoring fluid therapy.

**Fluid therapy in special situations**

Certain illnesses have altered fluid balance and in them it is essential to have varied fluid administration regimes. Some common conditions are illustrated in table 2. In conditions where there is excessive fluid loss (e.g. diarrhea) it is important to replace these along with maintenance fluids and electrolytes. In case of body fluid losses (e.g. ryle’s tube losses) one needs to replace not only fluids but also electrolytes in the proportions lost.

Practical tip: Ringer’s lactate or N/2 normal saline is adequate for replacing most body fluid losses.

---

**Table 2 : Fluid therapy in special situations**

<table>
<thead>
<tr>
<th>Situation</th>
<th>Total fluids</th>
<th>Watch for signs of</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patent ductus arteriosus</td>
<td>120 ml/kg/d</td>
<td>Heart failure</td>
</tr>
<tr>
<td>Bronchopulmonary dysplasia</td>
<td>120 - 140 ml/kg/d</td>
<td>Worsening oxygenation</td>
</tr>
<tr>
<td>Necrotising enterocolitis</td>
<td>200 ml/kg/d</td>
<td>Hypotension, Shock</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>400 ml/m²/d + urine output</td>
<td>Fluid overload</td>
</tr>
</tbody>
</table>
Extreme prematurity (gestation <28 weeks, birth weight <1000 gram): These neonates have large insensible water losses due to thin, immature skin barrier. The stratum corneum matures rapidly in 1-2 weeks and therefore fluid requirements become comparable to larger infants by the end of the second week. Fluid requirement in the first week may be decreased substantially by reducing the insensible water loss with the use of plastic transparent barriers or using double walled incubators. The initial fluids on day 1 should be electrolyte free and should be made using 5% dextrose solution to prevent risks of hyperglycemia. Sodium and potassium should be added after 48 hours of life.

Fluid restriction: There has been a lot of interest in the amount of fluid therapy and outcome of preterm neonates in terms of mortality and morbidity. The Cochrane meta-analysis on this topic could identify four eligible studies. Their findings state that, although restricted fluid therapy may lead to greater weight loss and dehydration, it is associated with a decreased incidence of death, PDA and NEC. There also seems to be a beneficial effect of restricted fluid therapy on the incidence of BPD. The volume of fluids used in the restricted groups differs from the above-described fluid therapy by 20-50 ml/kg/day in the initial 3-4 days. Based on their meta-analysis, the investigators had concluded that fluid therapy needs to be balanced enough to meet the normal physiological needs without allowing significant dehydration.

Fluid therapy and dyselectrolytemia

The general principles in managing any electrolyte disorder are
- Correct life threatening dyselectrolytemia quickly
- Slow correction thereafter till physiological levels are attained
- Treat or remove underlying cause

Hyponatremia (Plasma sodium < 130 mEq/L): Emergency management in a symptomatic neonate and plasma sodium < 120 mEq/L - bolus of 3% saline (0.5 mEq/ml) 2 ml/kg slow intravenous.

Definitive therapy

1. Hyponatremia due to sodium loss. Calculate sodium deficit using the formula for sodium deficit = (135 - plasma sodium) x 0.6 x body weight. Replace 1/3 in 8 hours, 1/3 in the next 16 hours and 1/3 over subsequent 24 hours.

2. Hyponatremia due to water excess - restrict intravenous fluids to 2/3rd maintenance.

Hypernatremia (Plasma sodium >145 mEq/L): If there is evidence of fluid overload, reduce sodium administration.

If there is evidence of hypovolemia - give a small bolus of albumin or plasma 10 ml/kg followed by relatively more water than sodium by using N/3 or N/5 solutions.

Note: Hypernatremia often indicates relative water loss to sodium rather than sodium retention. Thus, it is important to provide small quantities of sodium along with fluid and also avenues of free water loss like trans-epidermal loss need to be cut off.

Hypokalemia (Plasma potassium < 3mEq/L): Provide additional potassium by increasing its content in the 24 hour fluid.

Hyperkalemia (Plasma potassium > 6 mEq/L): Note: The commonest cause of high potassium values in blood samples is hemolysis, and high potassium levels must preferably be confirmed by ECG and lab analysis of nonhemolysed samples.
ECG signs of hyperkalemia: Peaked T waves, prolonged PR interval, absent P waves, QRS widening and slurring and arrhythmias.

Therapy should preferably also be under ECG monitoring

1. Calcium gluconate 1-2 ml/kg slow intravenous route over 0.5 to 1 ml
2. Sodium bicarbonate 1-2 ml/kg by slow intravenous route over 0.5 to 1 hour.
3. Remove all sources of exogenous potassium.
4. Salbutamol 4 mcg/kg IV bolus or as salbutamol nebulisation
5. Glucose - insulin therapy: Bolus of insulin and glucose in the dose of 0.05 units/kg of human insulin with 2 ml/kg of 10% dextrose to be followed by continuous infusion of 10% dextrose at 2-4 ml/kg/hour and human regular insulin (10 units/100ml) at 1 ml/kg/hour
6. Peritoneal dialysis
7. Kayexalate 1g/kg per rectally may be used if available with caution in a neonate who has no gut pathology and is not very premature.

**Conclusions**

Fluid therapy in the neonate is dictated by the changes that occur around birth and also the pathophysiology of the various illnesses. A clinician can adopt a rational approach to fluid therapy by answering certain questions.

**Key messages**

1. **Total fluids administered are calculated based on the birth weight till the neonate becomes heavier than the birth weight.**
2. **Electrolytes are added after the neonate is older than 48 hours.**
3. **Fluid therapy has to be altered when there are excessive fluid losses or fluid retention.**
4. **Correct life threatening dys-electrolytemia quickly. Slow correction thereafter till physiological levels are attained. Treat or remove underlying cause.**
5. **Monitor fluid therapy regularly and make changes particularly in sick neonates.**

**Bibliography**


**NEWS AND NOTES**

The IAP National Guidelines on Asthma Management are available with the Respiratory Chapter of IAP in the form of a book, ‘Asthma by Consensus’. You will be mailed the book after receiving a cheque for Rs.150/- (50 for mailing charges) written to ‘IAP Respiratory Chapter’, along with your name and mailing address. Please write to Dr.R.P.Khubchandani, kailas Darshan, Kennedy Bridge, Nana Chowk, Mumbai 400 007. Email: dr_rajukay@hotmail.com
FLUID AND ELECTROLYTES

MANAGEMENT IN ACUTE DIARRHEA

*Mittal SK
*Amit Agarwal

Abstract: Diarrhea continues to be a serious problem in many areas of the world. It results in large losses of water and electrolytes, especially sodium and potassium. Dehydration in a child could be isonatremic, hyponatremic or hypernatremic. To institute an appropriate treatment plan, hydration status of the patient with diarrhea should be assessed. This article focuses on the practical aspects regarding assessment and management of dehydrated child with oral rehydration therapy and intravenous fluid, with reference to the guidelines laid down by WHO and experiences at our center in the management of these cases.

Key words: Acute diarrhea, dehydration, fluid therapy

Fluid and electrolyte disturbances are major complications of acute diarrhea syndrome, which includes acute watery diarrhea and acute dysentery (invasive diarrhea). Dehydration and dyselectrolytemia are the major cause of morbidity and mortality in children with acute watery diarrhea.

Dehydration in a child could be isonatremic, hyponatremic or hypernatremic, depending on the cause of diarrhea and the rehydration solutions patient is given prior to examination. 70-80% of patients lose water and sodium proportionately leading to isonatremic dehydration.

To institute an appropriate treatment plan, patient with diarrhea should be assessed to determine

- Nature and pattern of diarrhea by a detailed history
- Status of hydration and nutrition
- Presence of other problems like fever, vomiting and abdominal distension
- Screening for concurrent illness(s) like pneumonia, otitis media, meningitis.

Assessment of dehydration

The first step is to assess degree of dehydration as it dictates the urgency of intervention and the volume of fluid, needed for rehydration. The following table summarizes the clinical features that are present with varying degrees of dehydration (Table 1).

- As per WHO guidelines mild and moderate dehydration has been clubbed as ‘some dehydration”
- Skin pinch is less useful in severely malnourished or obese child.
- Change in sensorium in severe dehydration sets in earlier than signs and symptoms of shock and should be carefully looked for
- Signs that remain useful for assessing hydration status in malnourished child are – Dry mouth and tongue, eagerness to drink (some dehydration) or absence of radial pulse in severe dehydration.

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Oral rehydration therapy (ORT)

It is used for prevention and treatment of mild to moderate dehydration and for maintenance of hydration in child with diarrhea.

Fluids used for oral rehydration therapy

- WHO Oral Rehydration Solution (ORS) (Table 2)
- Sugar and salt solution made at home by (mixing 40 g sugar and 4g salt in 1 liter of water)
- Food based solution like rice water with salt, or lassi with salt
- Home made fluids lemon water, rice kanji along with continued feeding


Table 1. Clinical features of dehydration

<table>
<thead>
<tr>
<th>Components</th>
<th>Mild dehydration</th>
<th>Moderate dehydration</th>
<th>Severe dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degree of wt.loss</td>
<td>3-5%</td>
<td>6-9%</td>
<td>≥10%</td>
</tr>
<tr>
<td>Mental status</td>
<td>alert</td>
<td>irritable</td>
<td>Lethargy/unconscious</td>
</tr>
<tr>
<td>Thirst</td>
<td>thirsty</td>
<td>decreased thirst</td>
<td>absent</td>
</tr>
<tr>
<td>Skin turgor</td>
<td>normal</td>
<td>Mild tenting</td>
<td>Tenting of skin</td>
</tr>
<tr>
<td>Tears</td>
<td>present</td>
<td>reduced</td>
<td>none</td>
</tr>
<tr>
<td>Buccal mucosa</td>
<td>moist</td>
<td>dry</td>
<td>parched</td>
</tr>
<tr>
<td>Eyes</td>
<td>normal</td>
<td>Deep set</td>
<td>sunken</td>
</tr>
<tr>
<td>Fontanelle</td>
<td>flat</td>
<td>soft</td>
<td>sunken</td>
</tr>
<tr>
<td>Urinary output</td>
<td>normal</td>
<td>decreased</td>
<td>anuria</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>normal</td>
<td>Slight tachycardia</td>
<td>Rapid pulse decreased BP</td>
</tr>
</tbody>
</table>

Table 2. Composition of WHO ORS

<table>
<thead>
<tr>
<th>Components</th>
<th>Amounts in gms</th>
<th>Final composition mEq/litre</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium chloride</td>
<td>3.5</td>
<td>Sodium 90</td>
</tr>
<tr>
<td>Sodium citrate</td>
<td>2.9</td>
<td>Citrate 10</td>
</tr>
<tr>
<td>Potassium chloride</td>
<td>1.5</td>
<td>Potassium 20</td>
</tr>
<tr>
<td>Glucose</td>
<td>20</td>
<td>Glucose 110 mOsm/L</td>
</tr>
<tr>
<td>Water</td>
<td>1 liter</td>
<td>Total 330 mOsm/L</td>
</tr>
</tbody>
</table>

Oral rehydration therapy (ORT)

The efficacy of WHO ORS to treat diarrheal dehydration is based on the fact that glucose linked enhanced sodium absorption remains largely intact during acute diarrhea of diverse etiology. Similarly the home available fluids containing sugar, starch etc. also enhance glucose linked absorption, as these carbohydrates break down to glucose and other monosaccharides in the gut. ORS should be used within 24 hours of its preparation. WHO has recently suggested a revised formulation as a universal ORS.

Hypooisomolar ORS

In hypooisomolar ORS, sodium and glucose concentrations have been lowered to 75 mEq/L each with final osmolarity made up to
245 mOsm/L (Table 3). This should not be confused with low sodium (type B) ORS currently available in the market.

It has been shown to be more effective in not only restoring hydration but also decreasing diarrhea losses by 20-30% and better control of vomiting.

**Low sodium ORS**

Drug controller of India also allows manufacturing and marketing of type B (low sodium) ORS which contains sodium equivalent of 60 mEq/L with a high glucose content (about 150-200 mOsm/L). The total osmolarity of these solutions is also comparable to standard WHO ORS i.e 330 mOsm/L. These low sodium ORS could be useful in prevention and maintenance phase of rehydration therapy and in the treatment of dehydration among neonates and young infants.

**Super ORS:** It is postulated that if glycine or starch is used instead of glucose in ORS, in addition to rehydration it will cause decrease in purge rates and improvement in diarrhea by enhancing absorption but improvement in efficacy has been marginal in randomized controlled trials. Rice based ORS (50g of puffed rice cooked in 1litre of water with a pinch of salt has also proved to be efficacious).

**Clinical application of ORT**

**Acute diarrhea with no dehydration:** Most children at the time of initial presentation to any medical facility do not have any dehydration. The fluid therapy at this stage is required to prevent dehydration, fluids available at home like coconut water, dal water, rice kanji etc. or sugar salt solution are advocated. Low sodium ORS or hypo osmolar ORS may also be used in this phase.

**Acute diarrhea with mild to moderate or some dehydration:** In a dehydrated child the fluid therapy consists of

- Correction of deficit (rehydration therapy)
- Replacement of ongoing or concurrent losses
- Provision for daily requirement

WHO ORS is given in the first 4 hours at the rate of 75 ml/kg in case of some dehydration. If the child is still dehydrated after 4 hours, same therapy could be repeated. If after 8 hours, dehydration persists then intravenous therapy should be used. In infants less than 3months of age it is preferable to use low sodium containing ORS.

**In the maintenance phase** ORS should be replaced at the rate of 10-20 ml/kg for each liquid stool. In young infants (<3mo of age) plain water should be given in between ORS in the ratio 1:2.

**Table 4. Maintenance fluid requirements**

<table>
<thead>
<tr>
<th>Age</th>
<th>Fluid ml/kg/24 hr</th>
<th>Age</th>
<th>Fluid ml/kg/24 hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>7days-3months</td>
<td>150</td>
<td>1-3 year</td>
<td>100</td>
</tr>
<tr>
<td>3-6 months</td>
<td>130</td>
<td>3-7 yrs</td>
<td>80</td>
</tr>
<tr>
<td>6months-1 year</td>
<td>120</td>
<td>&gt;7year</td>
<td>60</td>
</tr>
</tbody>
</table>
Breast-feeding should be continued and alternatively low osmolarity ORS can also be used. However the improvement in efficacy has been marginal in randomised controlled trials. If there is vomiting, small amount of ORS should be given with spoon and cup. This helps to control vomiting and to prevent dehydration. But if vomiting persists, then ORT should be abandoned in favour of intravenous therapy.

**ORT is ineffective when**
- there is high purge rate
- persistent vomiting
- severe acidosis / severe dehydration (failure to pass urine for >8 hrs)
- shock
- convulsion and abdominal distention
- sepsis
- severe dyselectrolytemia

In all these cases ORT should be terminated and replaced by intravenous therapy.

**Intravenous therapy**

The maintenance and deficit requirement of fluid and electrolyte should be computed. The daily fluid requirement can be calculated by using age and body weight, the readily available parameter (Table 4).

**Deficit therapy**

Deficit fluid requirement should be assessed by degree of dehydration. When correcting the dehydration with intravenous fluids it should be endeavored to correct it in first 8 hrs of fluid therapy. Fluid administered at this time should contain both deficit and maintenance fluid requirement in 8 hours. The following table gives total fluid requirement in first 8 hours of admission in various grades of dehydration (Table 5).

In the first 8 hrs 50% of deficit is replaced and rest of deficit is allowed to be taken care of by body’s own homeostatic mechanisms. As the loss of sodium is more in diarrheal stools, deficit correction is given as 0.45% saline or N/2 isotonic saline. Potassium should only be added to infusion when adequate urination is established.

**Replacement of concurrent losses**

It should be started after correction of dehydration. In this phase maintenance fluid should be given and concurrent losses should be replaced appropriately (Table 6).

**Management in case of severe dehydration with or without peripheral circulatory failure**

**WHO regimen**

WHO recommends intravenous therapy in case of severe dehydration, ringer lactate is to be given according to following schedule (Table 7).

There are certain limitations to the above fluid regimen as this is meant only for severe dehydration with circulatory failure but i.v. therapy may also be required in certain moderate dehydration circumstances as detailed above. Further as per this regimen, in older children the rate of fluid replacement is very fast which may lead to rapid change in electrolyte balance especially sodium.

An alternate plan which is much safer and easily applicable at all ages, is to give fluid intravenously in amounts of 150ml/Kg in first 8 hours, as mentioned in table 5. While in children above 3 years of age Ringer lactate or isotonic (0.9%) saline in 5% dextrose should be used, for younger children it may be safer to use N/2 (0.45%) saline with 5% dextrose saline. Of the calculated fluid (that is 150ml/kg), almost 40%, i.e. 60ml/Kg can be given in the first 2 hours at a rate of 20 to 30 ml/kg/hr and the remaining fluid be given over next 6 hours.
Practical steps for management of severe dehydration

- Set up immediate i/v access, obtain samples for electrolyte and arterial blood gas

- 20ml/kg bolus by Ringer lactate or isotonic saline in 20 minutes, repeat until intravenous volume is replenished, (i.e radial pulse becomes palpable) may give up to 50-60ml/kg in first 2 hours.

- No potassium is given till adequate urination is established

- If urination is not established even after adequate hydration, child should be assessed for parenchymal renal disease. It is absolutely important to ensure that the child is appearing clinically hydrated, with good volume pulse before giving the test dose of furosemide (Fig. 1).

- If possible CVP should be measured and furosemide should not be given if CVP is low (<6 cm of water). ABG should be done and acidosis to be corrected appropriately. Monitor of vitals like pulse, blood pressure every 15-30 min, intake output fluid balance, clinical signs of volume depletion or overload and electrolytes.

Hyponatremic dehydration (sodium <130mEq/L): It occurs when large amounts of electrolytes are lost as compared to water loss, or fluid loss is replaced by plain water. The signs of isonatremic dehydration along with neurological features like lethargy, convulsions may be seen. Asymptomatic hyponatremia is frequently observed in diarrheal diseases. The management is essentially on the lines of isonatremic dehydration but if serum sodium is less than 120 mEq/L and accompanied by symptoms like convulsions, rapid correction of sodium is required.

<table>
<thead>
<tr>
<th>Degree of dehydration</th>
<th>Body wt loss</th>
<th>Estimated fluid deficit ml/kg</th>
<th>Deficit replacement in 8 hrs ml/kg</th>
<th>1/3rd * Total fluid in first 8 hrs ml/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>mild</td>
<td>3-5%</td>
<td>30-50</td>
<td>nil</td>
<td>50</td>
</tr>
<tr>
<td>moderate</td>
<td>6-9%</td>
<td>60-90</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>severe</td>
<td>10% or more</td>
<td>120-150</td>
<td>60-75</td>
<td>50</td>
</tr>
</tbody>
</table>

* Calculated for 7 days - 3 years of age. The amounts are decreased thereafter as per maintenance requirements in different ages (see Table 4)

Table 6. Estimation of concurrent losses of electrolytes (mEq/Litre)

<table>
<thead>
<tr>
<th></th>
<th>Sodium</th>
<th>Potassium</th>
<th>Chloride</th>
<th>Bicarb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vomiting</td>
<td>90</td>
<td>20</td>
<td>110</td>
<td>-</td>
</tr>
<tr>
<td>Diarrheas*</td>
<td>30-60</td>
<td>20</td>
<td>70</td>
<td>20**</td>
</tr>
<tr>
<td>Gastric aspirate</td>
<td>90</td>
<td>15</td>
<td>110</td>
<td>-</td>
</tr>
</tbody>
</table>

* In diarrhea due to cholera (sodium loss 90-110 mEq/litre)
** Only in severely purging diarrhea
Sodium deficit = (135-observed serum sodium) x wt.in kg x 0.6

Only part of this deficit is corrected rapidly by 3%NaCl to raise serum sodium to 125 mEq/L as rapid correction may lead to central pontine myelinosis. The rest of correction is given slow over 24 hours.

It is recommended to avoid increase in sodium by more than 12 mEq/L/day.

Hyponatremia is fairly frequent in malnourished children. In these children it is mostly asymptomatic and requires no active management. Infact, rapid replacement of sodium in these children may result in excessive fluid retention and even congestive heart failure.

Hypernatremic dehydration (sodium >150 mEq/L): It occurs when there is disproportionately large net loss of water as compared to loss of electrolytes, occurring in formula fed infants or with erroneous ORS therapy. Though uncommon, it is dangerous. The movement of water from intracellular spaces during hypernatremic dehydration protects intravascular volume. Classic signs of dehydration are absent, doughy feel of skin, woody tongue, hyperreflexia, hypertonia and convulsions may be seen.

Management

- Serum electrolytes, ABG should be taken
- To avoid cerebral edema while correcting hypernatremia, the fluid deficit should be corrected slowly.

- The time required to correct sodium concentration is based on initial sodium concentration as serum sodium concentration should not decrease by more than 12 mEq/day. Thus hypernatremia is corrected over 24 hours if serum sodium is between 145-157 mEq/L and in 48 hours if initial serum sodium is 158-170 mEq/L.

- After initial management of shock with ringer lactate (20-30 ml/kg), the remaining deficit is corrected with N/3 saline (sodium content 50 mEq/L)

**Hypokalemia**

Hypokalemia (serum potassium <3.5 mEq/L) occurs in children with diarrhea, especially when they are malnourished. Mostly it is asymptomatic, though it may be associated with pseudo paralysis of different group of skeletal muscle presenting as neck flop and generalised hypotonia.

**Management:** In most cases hypokalemia improves with routine oral or intravenous fluid therapy. Potassium should be added in concentration of 20 mEq/L to the IV fluid after adequate urination is established. If symptomatic hypokalemia is present, this amount can be increased to 30 or even 40 mEq/L. These amounts should not exceed beyond 40 mEq/L (i.e. 4 mEq is equal to 2 ml of 10% potassium chloride per 100 ml of intravenous fluid). Potassium should never be given as rapid correction as this could be rapidly fatal.

**Acidosis**

Metabolic acidosis is quite frequently encountered in diarrheal dehydration. Mostly it is asymptomatic and gets corrected as fluid deficit is corrected and renal function is restored. However in children with symptomatic acidosis,

---

**Table 7. Intravenous therapy in severe dehydration (WHO regimen)**

<table>
<thead>
<tr>
<th></th>
<th>First give 30 ml/kg</th>
<th>Then give 70 ml/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;12 months</td>
<td>1 hour*</td>
<td>5 hours</td>
</tr>
<tr>
<td>Older child</td>
<td>30 min*</td>
<td>2.5 hrs</td>
</tr>
</tbody>
</table>

* repeat again if radial pulse absent
rapid correction of serum bicarbonate may be required. If bicarbonate levels can be obtained, then bicarbonate should be administered in amount calculated to raise plasma bicarbonate levels to 18mEq/L using following formula (18-observed plasma bicarbonate) x weight in Kg. x 0.5

1 ml of 7.5% of sodium bicarbonate contains almost 1mEq/ml of bicarbonate. This is a hypertonic solution and should be given in dilution of 1:5 with distilled water.

If serum bicarbonates levels are not available, and the child is clinically acidotic, then 3-5 ml/Kg of 7.5% sodium bicarbonate solution diluted 1:5 with distilled water can be given intravenously over 1-2 hours period. This will increase plasma bicarbonate levels by 5-8 mEq/L and may be life saving.
Bibliography


BOOK REVIEW

Title: Practical Guidelines on Fluid Therapy
Authors: Dr. Sanjay N. Pandya
Review: Fluid therapy is one of the more important problems but a volatile subject. The author has made it simple by presenting all the chapters in the form of questions and answers which makes understanding of complex problems easy. This book describes the basic physiology and principles of fluid therapy in depth. Approach to dyselectrolytemias is very practical. The author has described many case scenarios in the chapter on acid base disorders, which help in better understanding of the problem. The calculation of rate of fluid infusion for standard and micro IV drip set is sample and will be very useful to practitioners. There is a separate chapter on fluid therapy in children. The print is bold and easy to read. There are a few proofing errors which could have been avoided. Overall, this book can be recommended for practitioners.

Publisher: Dr. Sanjay N Pandya
Samarpan Hospital, Near Lodhawad Police Station, Bhutkhana Chowk, Rajkot – 360 002. Gujarat.

Price: Rs.195/-

ERRATUM

We regret for leaving out the name of Co-author Dr. Sherene Samuel, Pediatrician, C.S.I. Kalyani Multispeciality Hopsital, Chennai 600 004 for the article ‘An unusual cause of recurrent GI bleed’ appeared in Indian J Pract Pediatr 2004; 6(1): 79.
FLUID AND ELECTROLYTE MANAGEMENT IN RENAL DISORDERS

* Vijayakumar M
** Prahlad N
*** Nammalwar BR

Abstract: Disorders of kidney are commonly associated with hypervolemia, hyperkalemia, hyponatremia and metabolic acidosis. Hypovolemia, euvolemia and hypervolemia or fluid overload states are possible in ARF as per the cause and stage of the disease. Hypovolemia per se is an important prerenal factor in renal failure. In renal disorders, oliguric conditions are associated with hyponatremia and polyuric conditions are associated with hypernatremia. Metabolic acidosis is common with renal failure and metabolic alkalosis is common following loop diuretics and in some chronic tubulointerstitial diseases. Decision on fluid and electrolyte is taken as per volume and electrolyte status of the body. Meticulous monitoring of fluid status and serum electrolytes is mandatory in proper management of fluid and electrolyte disorders associated with renal diseases. Dialysis is preferred as a safe and better method to correct multiple biochemical abnormalities with renal disease.

Key words: Fluid balance, dyselectrolytemia, acute renal failure, nephrotic syndrome, acute nephritic syndrome, polyuric states.

In health, the kidney regulates the sodium and water balance and intravascular volume and plays the key role in potassium and acid base homeostasis. Disorders of kidney are associated with hypervolemia, hyperkalemia, hyponatremia and metabolic acidosis. In most children these complications can be managed by adjustment of dietary and oral fluid intake, judicious use of diuretics, cation exchange resins and sodium bicarbonate and uncommonly with intravenous fluids. Dialysis may be required if conservative measures fail to control life threatening fluid, electrolyte and acid base complications. This article is confined to prevention and treatment of disorders of the fluid, electrolytes and acid base balance in acute renal failure (ARF), acute nephritic syndrome (ANS), nephrotic syndrome (NS) and polyuric disorders. Prevention and treatment of metabolic abnormalities are discussed elaborately in ARF and are briefly touched upon in other conditions.

Acute renal failure

Acute renal failure is characterised by inability of the kidney to maintain body homeostasis and is manifested as reduced urine output with increasing blood urea and serum creatinine levels. The onset of renal failure can occur in a few hours to few days. Ten percent of ARF can have good urine output and is called polyuric ARF. It occurs secondary to sepsis, aminoglycoside toxicity, burns and is a feature of multiorgan dysfunction seen in the PICU setting. In ARF, hypervolemia or fluid overload,
hyponatremia, hyperkalemia and metabolic acidosis are common fluid, electrolyte and acid base disturbances.

**Prevention of metabolic abnormalities**

**Fluid:** Maintenance of euvolemia is an important component of renal function and is done by a delicate balance between water and sodium intake or its losses. Disturbances in renal function contribute more commonly to hypervolemia and infrequently to hypovolemia. Fortunately, the majority of ARF acquired in the community are associated with hypervolemia and uncommonly with disturbances of electrolytes and acid base. If at all, they occur, it is usually very minimal. Gross metabolic abnormalities are associated with hospital acquired ARF. Oral administration of fluid, electrolytes and bicarbonate is the ideal route. Alternatively nasogastric administration is the second choice. Parenteral administration of fluid and electrolytes is the third choice and should be preferred only in the situation where there are associated gastrointestinal disorders.

The aim of fluid management in ARF is to maintain euvolemic state. Appropriate management of fluid and salt intake does this. Fluid intake should be restricted to insensible water loss on day one of ARF and subsequently increased to urinary output plus both abnormal and insensible water loss. On day one of the examination, all children are presumed to be anuric and fluid intake is restricted to insensible water loss, namely 30 ml/kg body weight for infant, 20 ml/kg body weight for older children and 10 ml/kg, body weight for adult or 400 ml/m² BSA per day. On day two, fluid intake is increased to the previous days urine output plus insensible and abnormal fluid loss. If adequate fluid has been administered, the daily weight would remain stationary, though it is ideal to have a small loss of around 0.5% of the previous day’s weight. The fluid intake should contain essentially a first class protein and the ideal choice is usually milk or curd, where milk is not tolerated.

In general, child will get fluids as per volume status. In hypovolemic situations maintenance fluid as stated above and extra fluid to correct dehydration is given. In euvolemic states only maintenance fluids are given. But in hypervolemic and edematous situations, fluids are restricted. If parenteral fluid administration is planned a simple maintenance fluid is 5% GDW. If the child is grossly acidotic 5 mEq/100 ml of sodabicarb is added and if child is not acidotic 3 mEq/100 ml of sodabicarb is added to the 5% GDW given as maintenance fluid. If child has hypokalemia needing correction, 4 mEq potassium/100 ml is added to maintenance fluid. If the child has normokalemia or hyperkalemia, no potassium is added to the maintenance fluid. This simple decision on fluid, sodium, bicarbonate and potassium therapy will be a very useful guide in the initial fluid management. Subsequently, daily estimation of serum electrolyte and bicarbonate and their levels will decide the addition or subtraction of the electrolytes to the maintenance fluid. Dehydration correction is essentially done with normal saline or Ringer lactate as per need and not by dextrose solution with sodabicarb. The measure of normal effective circulating blood volume or euvolemia is the assessment of central venous pressure. In clinical practice, features of fluid loss include loss of body weight, poor skin turgor, prolonged capillary refill time, orthostatic hypotension, high urine specific gravity and increased blood urea disproportionate to serum creatinine values. On the other hand fluid overload is suggested by increasing body weight, dependent edema, tachypnea, tachycardia, congestive cardiac failure and cardiomegaly with interstitial edema.

**Sodium:** Children with tubulointerstitial
disorders, post obstructive release diuresis and recovering ATN will need normal sodium on a daily basis. Children with chronic renal failure are given normal sodium requirement, unless there is edema or uncontrollable hypertension.

**Potassium:** Hyperkalemia is almost inevitable in children with persistent oliguria. Serum potassium generally tends to rise by a 0.5 mEq/L/day in anuric children due to accumulation following potassium containing foods and IV solutions and release from damaged tissues as in excessive hemolysis. In the presence of sepsis, trauma, extensive surgery and coexisting metabolic acidosis, the rise in serum potassium can be rapid and reach dangerously high level in a short time. Hyperkalemia can be delayed or prevented by careful assessment of the diet for potassium, avoiding potassium containing diet, discontinuation of ACE inhibitors and potassium sparing diuretics, avoiding of antibiotics containing high potassium salt, rapid control of infection, debridement of necrotic tissues and control of hemolysis. Oliguric children will not get potassium in the daily maintenance. Potassium is added only in the presence of hypokalemia or in a child losing potassium through extra renal losses as in diarrhoea or in the diuretic phase of ATN. The daily serum potassium levels will regulate the intake. Tender coconut water, orange and other fruits containing potassium are initially added. Later if needed potassium is added as oral potassium chloride or rarely as IV potassium.

**Management of metabolic abnormalities**

Hypovolemia, euvolemia and hypervolemia or fluid overload states are possible in ARF as per the cause and stage of the disease. In renal disorders, oliguric conditions are associated with hyponatremia and polyuric conditions are associated with hypernatremia.

**Hypovolemia:** Hypovolemia is potentially dangerous, as it can compromise tissue perfusion and exacerbate renal ischemia and worsen renal injury. Compromise of tissue perfusion can lead to lactic acidosis and further peripheral vasodilatation consequent to tissue ischemia. Renal ischemia can induce acute tubular necrosis secondary to blunted glomerular autoregulatory response in the presence of renal disease. Hypovolemia in renal disease occurs due to indiscriminatory use of diuretics and restriction of salt and fluids. A child with prerenal ARF following diarrhea can present with hypovolemia and a child with prerenal ARF following cardiac failure can present with hypervolemia but with low effective blood volume. Children with oliguric intrinsic renal failure present with hypervolemia whereas non-oliguric ARF present with usually euvolemic status. Children with hypovolemic ARF will get 20 ml/kg of NS over 1 hour and if no improvement should get further 3 similar boluses with CVP monitoring or 2 boluses if CVP monitoring is not available. Care must be taken to avoid over shoot of fluid administration, which can result in dangerous pulmonary edema. Monitoring of the central venous pressure is one of the best ways to prevent the complication. In the absence of these facilities, assessment for increased pulse volume, pulse rate, liver size, onset of hypertension and basal crepitations can avoid this. If euvolemia is achieved but urine output is inadequate, diuretic therapy with 2 mg/kg body weight of IV furosemide is indicated. If no diuresis occurs even after this with an elevated CVP or clinical evidence of hypervolemia child should get restricted fluid or fluid removal with dialysis or continuous renal replacement therapy (CRRT).

Children in diuretic phase or recovery phase of ATN and in polyuric renal failure can present not only with hypovolemia but also have substantial daily fluid and electrolyte losses. The fluid intake in these children should match the urine output plus the insensible water loss. The
fluid loss should be estimated ideally on hourly basis or at least 4th hourly and replaced. The ideal fluid to begin will be 0.45% saline with added daily maintenance of potassium and bicarbonate. Subsequent administration of electrolytes will depend on the 12th hourly biochemical values.

Problem of ‘vascular leak’ should be considered in a critically ill child treated in a PICU. The child presents with volume overload like picture but with decreased effective circulating blood volume due to vascular leak. Fluid leakage from intravascular to interstitial space results in poor renal and tissue perfusion. More fluids are needed to maintain intravascular volume for renal and tissue perfusion in these children but with the problem of further aggravation of clinical picture of fluid overload. Various options are available in this situation. Intense diuretic therapy may help if renal functions are just adequate, in spite of renal failure, to induce diuresis. Continuous furosemide infusion at a rate of 0.04mg/kg/hr is found to be adequate and life saving. If the renal dysfunction is very severe wherein diuresis cannot be achieved, periodic or continuous ultrafiltration by peritoneal dialysis or hemodialysis or by CRRT is needed to remove the fluid to maintain fluid balance and adequate tissue and renal perfusion. As electrolyte losses that accompany ultrafiltration of fluids in the above modalities is isotonic, the electrolyte content of the infused fluid should be adjusted to match the electrolyte content of the ultrafiltrate and should be isotonic in nature.

**Hypervolemia:** Children with oligoanuria present with hypervolemia leading to fluid restriction or fluid removal to get at euvoletic status. In severe edema, intake equals insensible fluid loss. In mild edema, in oliguric and also in non-oliguric ARF, intake equals urine output and insensible fluid loss. Hypervolemia clinically manifests as elevated jugular venous pressure, tachycardia, third heart sound, pleural effusion, pulmonary edema, ascites, dependent edema and increasing body weight. If hypervolemia develops in spite of salt and fluid restriction, an initial dose of bolus IV furosemide (2-4 mg/kg body weight per dose) can be tried. A repeat dose can be tried in the next 2 hours if the urine output does not exceed 1 ml/kg body weight per hour. Continuous infusion of furosemide as stated above has been advocated in certain situations. This is associated with danger of fluid overload if the diuretic response is slow, though it could be sustained. It could be used where fluid overload is not a dangerous situation but there is oliguria and fluid removal can be done over a sustained period of hours and days. Such situation usually arises in intensive care units wherein there is massive shift of fluid from the intravascular to extravascular space such as sepsis, dengue shock syndrome, leptospirosis, burns and hypoalbuminmic conditions. However, IV furosemide is useful in acute glomerulonephritis and not so beneficial in ATN. High dose IV furosemide, as much as 5-10 ml/kg body weight per dose, has been found useful in adults to convert oliguric ATN into polyuric ATN. This helps liberalization in the management of fluid and electrolytes. Mannitol has been advocated to treat hypervolemia. Mannitol is an osmotic diuretic and it can precipitate life threatening pulmonary edema due to expansion of the intravascular volume when there is no resultant diuresis.

**Euvolemia:** Euvolemic status can be maintained by administration of fluid intake to match the urine output with insensible water loss and abnormal losses like loose stools, surgical drains and vomitus as well as excessive sweating when the child is in incubators or in hyperpyrexic conditions and with burns. The replacement fluid can be in the form of dextrose 0.45% saline with appropriate daily requirement of the electrolytes, which should be finally guided by biochemical
values. Replacement of fluid, electrolytes and bicarbonate for abnormal loss will depend on the source of loss. For example the vomitus will be replaced by equal quantity of 0.45% saline.

**Hyponatremia:** Hyponatremia is usually due to presence of excess of free water compared to solutes in plasma. Hyponatremia is commonly due to excess intake of dilute or hypotonic enteral or IV fluids. Hyponatremia is mostly seen in non-oliguric ARF and diuretic phase of ARF as well as in post obstructive release diuresis. If serum sodium is <130 mEq/L, fluid restriction is enough. If serum sodium is < 120 mEq/L, raise S Na to 125 mEq/L which is calculated as deficit X Wt in Kg X 0.6 and given as normal saline (0.9%) over 24 to 48 hrs if the child is asymptomatic. Child may need dialysis, if symptomatic with serum sodium less than 120 mEq/L. Pseudohyponatremia can occur in the presence of hyperglycemia and hypertriglyceridemia. This does not need correction with sodium.

**Hypernatremia:** Hypernatremia is due to reduction in free water compared to solutes in plasma. Hypernatremia complicates renal disease in the presence of large insensible water loss as in hyperpyrexic conditions, use of warmers in NICU, high environmental temperature and gastroenteritis with inadequate replacement of fluids, excessive urine loss as in neonates and in conditions like diabetes insipidus both primary and secondary. In majority of the situations of hypernatremia there is severe dehydration with some degree of acute renal insufficiency. Usually prerenal in nature this situation will respond to restoration of perfusion and early rehydration. The water deficit should be replaced preferably orally via nasogastric tubes. If this is not possible due to disorders of the bowel, replacement with hypotonic or dextrose containing solutions should be administered intravenously. But in children with intrinsic acute renal failure with acute tubular necrosis or acute glomerulonephritis, the renal insufficiency does not respond to volume infusion. This step will aggravate volume overload and hence careful renal replacement therapy (Peritoneal dialysis or CRRT) should be planned.

**Hyperkalemia:** Mild to moderate hyperkalemia (5.5-6.5 mEq/L) can be controlled by oral administration of potassium ion exchange resins such as sodium polystyrene sulphonate (1 g/kg body weight) dissolved in 20% sorbitol or water and given as retention enema for 3 hours. This can be repeated once in every 6 hours. Calcium resonium has the advantage of avoidance of sodium load which can occur with sodium polystyrene sulphonate. In the presence of electrocardiographic manifestation of hyperkalemia, more emergency care is necessary. Dextrose infusion with insulin, IV calcium gluconate and sodium bicarbonate and salbutamol nebulisation are needed. Associated severe renal failure will necessitate dialytic therapy to correct hyperkalemia.

**Hypokalemia:** Hypokalemia rarely occurs in the oliguric renal disease. It is often seen in the diuretic phase of acute renal failure and in polyuric conditions associated with tubulo-interstitial diseases namely, Bartter’s syndrome, primary diabetes insipidus, secondary diabetes insipidus due to nephrocalcinosis, renal tubular acidosis and cystic disease of the medulla. Hypokalemia can be iatrogenic in the presence of indiscriminate use of diuretics, particularly thiazide. It can also occur with the over use of sodium bicarbonate solution, insulin with glucose and with beta agonists. Hypokalemia can produce ventricular arrhythmias and fatal cardiac arrest below 3 mEq/L. Prompt treatment with IV potassium solutions containing as much as 6 mEq/L can be administered with cardiac monitoring in ICU settings.

**Acid base disturbances:** The metabolism of dietary proteins gives about 50-100 mmol of fixed
non-volatile acid. In renal failure the kidney is unable to excrete the H+ ions. Metabolic acidosis occurs in hypercatabolic states. Metabolic acidosis with wide anion gap is seen in oliguric renal disease. In non-oliguric renal disease such as renal tubular acidosis a normal anion gap metabolic acidosis is observed. This is secondary to failure to reabsorb bicarbonate in the proximal tubules or excretion of H+ ions by the distal tubules or failure to produce ammonium ions, which in turn helps in the production of new bicarbonate and removal of H+ ions in the tubular fluids. Lactic acidosis can complicate circulatory collapse, seizures, liver disease or sepsis. These can compound further bicarbonate deficiency. Management is not by replacement of bicarbonate, which can worsen the clinical condition in the presence of lactic acidosis. Correction of the circulatory deficiency, sepsis etc would be safer than administering bicarbonate. After excluding lactic acidosis, metabolic acidosis does not require treatment unless serum bicarbonate and pH fall below 18 mEq/L or 7.35 respectively. Acidosis can be corrected by administration of sodium bicarbonate by mouth or intravenously. Tablet soda-bicarbonate (325 mg) gives about 4 mEq of bicarbonate. The IV bicarbonate solution can be given orally and 1ml gives 1 mEq of bicarbonate. Care must be taken to use fresh solutions, as carbon dioxide will be lost when exposed for a longtime to atmosphere. When pH of blood is <7.2 more active measures are necessary. Rapid correction of acidosis can lead to hypocalcemia and hypokalemia. Prophylactic administration of oral calcium with serial monitoring of serum calcium levels can prevent this. Likewise, monitoring of serum potassium level is needed. Administration of bicarbonate solution should ensure adequate ventilation and is contraindicated in the presence of hypertension, severe edema and anuria.

In ARF, usually metabolic acidosis and dyselectrolytemia are treated by dialysis much more safely than by administration of fluids, electrolytes and bicarbonate. Metabolic alkalosis is a rare complication. If this occurs, it is due to excessive vomiting or administration of alkalis and antacids. Management is essentially by avoiding the precipitating factors.

**Acute nephritic syndrome**

Acute nephritic syndrome is characterised by fluid and salt retention due to failure of glomerular filtration. The retained salt and fluid are present in intravascular compartment leading to fluid overload in both systemic and pulmonary circulation. The capillary leak from accumulated intravascular fluid leads to edema. In acute nephritic syndrome, hypervolemia or fluid overload, hyponatremia, hyperkalemia and mild metabolic acidosis are common fluid, electrolyte and acid base disturbances. Prevention and treatment of these complications are as follows.

**Fluid adjustment:** On day one of the examination, all children are presumed to be anuric and fluid intake is restricted to insensible water loss, namely 30 ml/kg body weight for infant, 20 ml/kg body weight for older children and 10 ml/kg body weight for adult or 400 ml/m² SA per day. On day two, fluid intake is increased to the previous day’s urine output plus insensible water loss. Fortunately acute nephritic syndrome is not associated with massive edema. The oligoanuric period in acute nephritic syndrome usually lasts for about 10-14 days. Hence prolonged restriction of fluids may not be necessary and can be liberalized according to the output in the absence of hypertension. Strict monitoring of body weight is a trusted and simple, reliable evidence of appropriate fluid balance. In the presence of persistent oligoanuria, loop diuretics is indicated.
**Sodium modulation:** Sodium intake is restricted in ANS in the presence of edema and more so in the presence of hypertension. On day one, no added salt is advocated. A typical South Indian diet containing iddly, idiappam, rice puttu, sugar rice, curd rice, lemon rice or chappathi and potatoes or dhal can be taken without salt. With increasing urine output to more than 1 ml/kg body weight per hour and with a normal blood pressure, salt can be added in increasing quantity. One-gram salt/day can be added and increased by 1 gram every 3rd day provided the urine output is adequate with decreasing weight and maintenance of normal blood pressure. Unrestricted salt intake can lead to hypervolemia, hypertension, encephalopathy or cardio-respiratory distress. Unrestricted fluid intake, excessive diuretics and failure to administer salt at an appropriate time can lead to hyponatremia and its consequences.

**Hyperkalemia:** It is rare in ANS and occurs following indiscriminate intake of foods containing high potassium namely tender coconut water, electrolyte solutions and fruit juices. Failure of parents to recognize that the reduced urine output is due to renal disease encourages them to take above fluids and not uncommonly with fatal outcome. ANS like conditions involving hemolytic conditions such as drug-induced hemoglobinurias and HUS can be associated with hyperkalemia. Prevention is essentially by dietary restriction. Hyperkalemic conditions can be managed as mentioned in ARF above.

**Metabolic acidosis:** This is a very rare complication in ANS. It needs to be corrected if the serum bicarbonate falls below 18mEq/L in very small children or 15mEq/L in older children or when pH is < 7.2. Oral bicarbonate tablets can be used. Calcium carbonate can be used with strict monitoring of the serum calcium levels. Use of IV sodium bicarbonate can result in hypervolemia and its consequences.

In all the above situations, where conservative management has failed or worsened the clinical condition or in the presence of multiple fluid, electrolytes and acid base disturbances and persistent anuria > 48 hours, peritoneal dialysis will be indicated.

**Nephrotic syndrome**

Nephrotic syndrome (NS) is characterized by massive urinary protein loss resulting in hypoalbuminemia causing reduction in effective circulating blood volume as a result of extra vascular accumulation of fluids. In NS, gross edema, hyponatremia, hypokalemia and mild metabolic acidosis are common fluid, electrolyte and acid base disturbances. Prevention of these complications is as follows.

**Fluid management:** The edema in NS is characterized by increase in fluids in the extra vascular space with reduction in intravascular volume leading to oliguria. The familiar theory is that these ‘underfill’ nephrotics have accentuated renin angiotensin system causing secondary hyperaldosteronism and further retention of salt and fluids. Recent evidences contradict the above underfill theory. The present ‘overfill theory’ gives importance to primary sodium retention by the kidney rather than following secondary hyperaldosteronism. In both the theories, the common factor is fluid and salt accumulation. In underfill nephrotics plasma infusion followed by diuretics and in overfill nephrotics intense diuretic therapy followed by ACE inhibitors are found useful.

In severe edema, intake of fluid is restricted to insensible water loss. They are also treated with diuretics, either thiazide or loop diuretics or a combination of thiazide plus loop diuretics if there is evidence of resistance to a single diuretic use. In moderate edema, fluid intake equals previous day’s urine output or insensible water loss whichever is smaller. Diuretics should be
used only in the presence of urine output < 1 ml/kg body weight per hour. In mild edema usually fluid intake equals the urine output.

Diuretics are helpful in children in nephrotic relapse, particularly with steroid-resistant disease. Serum potassium levels should be monitored, as well as clinical signs of worsening intravascular volume contraction. If children have moderate intravascular volume contraction, the injudicious use of diuretics could precipitate an episode of hypotension with a greater risk of thrombosis and acute tubular necrosis. The most commonly used diuretic is furosemide; doses of 1-2 mg/kg per dose are given orally or intravenously. Spironolactone is added to the diuretic regimen for its potassium sparing effect. The addition of thiazide (1-2 mg/kg/day) or metalazone (0.5 mg/kg/day) to furosemide is usually very effective in patients who are refractory to furosemide alone. These drugs should precede the administration of loop diuretics by about 2 hours.

The use of intravenous albumin (25%) may be helpful in the child with NS resistant to diuretics. Because albumin infusions are very expensive, and the albumin is excreted very rapidly in the urine, the infusions should be used only to promote edema mobilization in children with marked ascites which compromises the pulmonary function, significant pleural effusions, scrotal or labial edema, and severe peripheral edema with skin breakdown. It is also helpful in protecting the blood pressure and renal perfusion in a septic child with NS. The usual dose is 1 gram/kg up to a maximum of 25 grams, infused over two hours with close monitoring of the blood pressure. The infusion is usually followed by furosemide (1 mg/kg) given intravenously. The albumin infusions may be repeated as frequently as every 6 to 12 hours. If an oligoanuric child fails to increase his or her urine output after the first or second albumin infusion, the child needs to be evaluated immediately for the presence of acute renal failure.

Hypovolemia is the frequent complication in NS and can present as ARF. Nephrotic hypovolemia can present with abdominal pain, hypotension, sluggish circulation, relative polycythemia, acute tubular necrosis and thrombosis. Precipitating factors include severe nephrotic relapse, infections, diuretics, paracentesis and diarrhea. More often it is due to indiscriminate and excessive use of diuretics particularly in children with underfill etiology. Symptomatic hypovolemia is treated with 20 ml/kg plasma or 1 gram/kg 20% albumin infusion followed by diuretics with careful monitoring of the blood pressure and circulatory overload.

**Sodium modulation:** Sodium promotes edema formation and hence the child should be placed on a “no added salt” diet. For children under five, this diet allows 1 to 1.5 grams of sodium, for school-aged children about 2 grams of sodium and for adolescents 3-4 grams of sodium per day. Technically sodium restriction is indicated only during times of relapse, but no extra salt diet at all times helps the entire family to maintain a consistent regimen. Fluid restriction is necessary in addition to the sodium restriction. Failure to restrict fluid intake in the presence of salt restricted diet can lead to hyponatremia and seizures with serious outcome. The management of hyponatremia has been outlined elsewhere. Mild asymptomatic hyponatremia can be managed by strict fluid intake to match insensible water loss and by addition of 1-2 grams of salt in the diet. Loop diuretics can be avoided. Thiazides are relatively safer and can be used when the edema is moderate or severe.

**Hypokalemia:** This is one of the frequent complications seen in NS. Hyperkalemia never probably occurs in NS. The liberal use of
diuretics is the reason. The frequent use of loop diuretics, because of its parenteral dose availability, the rapidity of diuretic onset, failure to monitor serum electrolytes at least twice in a week initially and then once a week subsequently can cause hypokalemia and metabolic alkalosis. Symptoms of hypokalemia such as tiredness, limb weakness, abdominal distension are often over looked. The prevention of hypokalemia involves co-administration of potassium chloride in the absence of renal failure, use of spironolactone or foods containing potassium. The presence of serum potassium less than 3 mEq/L or 3.5 mEq/L with ECG changes warrants parenteral administration of IV potassium as described elsewhere.

**Metabolic alkalosis:** It is more frequent than metabolic acidosis. Hypokalemic metabolic alkalosis is secondary to diuretic therapy and resultant hypovolemia. Prevention is by proper use of diuretics and replacement of salt and fluids in the presence of severe hypovolemia. In clinical practice this condition is never severe enough warranting administration of IV saline and potassium. This can be well treated with oral potassium replacement and acetazolamide.

**Metabolic acidosis:** This condition occurs in NS in the presence of moderate renal failure and sepsis. Management has to be done as already enumerated above.

**Polyuric states**

Polyuric conditions are defined as situations wherein urine output is > 4 ml/kg body weight per hour in children or 5 ml/kg body weight/hour in neonates and infants. Polyuric conditions can be due to non-renal causes such as primary cranial diabetes insipidus or secondary cranial diabetes insipidus or secondary to tumor, trauma or infections of the central nervous system. Renal conditions include primary nephrogenic diabetes insipidus or secondary nephrogenic diabetes insipidus due to renal tubular acidosis, Fanconi’s syndrome, tubulointerstitial diseases such as polycystic kidney disease, medullary cystic disease, nephronophthisis, reflux nephropathy and posterior urethral valve. Transient secondary diabetes insipidus can occur in post obstructive release diuresis and in recovering ATN. The principles of management are outlined below.

**Primary and secondary cranial diabetes insipidus**

Replacement of antidiuretic hormone, desamino-D vasopressin will correct the polyuria. Failure to monitor the fluid intake can cause hyponatremia.

**Primary nephrogenic diabetes insipidus:** Replacement of urinary water losses by adequate supply of fluids is the most important component of therapy. But, infants cannot drink the amount of fluids to match polyuria. So low solute diet, to reduce the renal osmolar load and decrease obligatory water excretion, to reduce polyuria is recommended. Initially to reduce solute load both protein and sodium restriction is attempted but later to avoid protein deficiency, salt restriction alone is continued. Oral fluid intake is encouraged as much as possible. Monitoring the temperature of the child and daily weight will help in judging the appropriate fluid balance. The clinical symptoms of irritability, restlessness, preference for fluids over solids particularly water, preference for cool environmental temperature and inadequate weight gain suggest chronic fluid deficit. In certain situations it may warrant parenteral fluid intake particularly in infants. Preference of fluids can supersede the nutrition intake and cause malnutrition.

To reduce polyuria, the following pharmaceutical interventions are useful. Therapy with hydrochlorothiazide (2-4 mg/kg/24 hours) and reduced salt intake results in reduction of urine output by 25-50% of baseline value.
resultant hypokalemia can reduce renal concentration ability and cause arrhythmias. Hence additional potassium is given. Combination therapy with hydrochlorothiazide and indomethacin (2 mg/kg/24 hours) or hydrochlorothiazide and the potassium sparing diuretic, amiloride (0.3 mg/kg/24 hours) is beneficial. Hydrochlorothiazide in the distal convoluted tubule (DCT) reduces sodium reabsorption by inhibition of NaCl co-transporter. The resultant sodium loss causes ECF contraction and reduction in GFR causing increase in sodium and water reabsorption in proximal convoluted tubule (PCT) thereby reducing sodium and water delivery to distal convoluted tubule and hence reduction in polyuria. Renin - angiotensin - aldosterone axis (RAAAA) is also activated which increases sodium absorption and along with water. In recent times, evidences are made available of a different action of thiazide in polyuric situations. In the absence of vasopressin, luminal thiazide increases osmotic and diffusional water permeabilities causing increase in water reabsorption and hence reduced water excretion.

**Secondary nephrogenic diabetes insipidus:** In this situation unlike the primary nephrogenic diabetes insipidus, child is encouraged normal salt intake with free fluid as per the demands. In certain situations like presence of postural hypotension and chronic dehydration, excessive salt is advised. These children are salt losers and more prone for isotonic dehydration, while children with primary nephrogenic diabetes insipidus are prone for hyponatremia. The use of diuretics is discouraged for it can precipitate an acute renal failure or worsen renal failure in a preexisting situation. Even in the presence of hypertension in these children salt intake is encouraged, as the hypertension is renin-angiotensin dependent, secondary to volume depletion or renal ischemia. Salt and fluid restriction is restricted in these children only in the presence of edema or severe hypertension. Theoretically the salt intake should be guided by the amount of sodium that is lost in a 24 hour urine specimen. In clinical practice normal salt intake is practical.

**Post obstructive release diuresis and diuretic phase of acute tubular necrosis:** In post obstructive release diuresis, child presents with polyuria after release of the obstruction. Catheterisation of the bladder or valve fulguration or vesicostomy are the situations in posterior urethral valve disease causing post obstructive release diuresis. Similarly renal stone disease following release of bilateral obstruction can present with polyuria. The damaged DCT where concentration mechanism is maximal, is not completely repaired following release of obstruction and hence continuing concentration defect. The DCT is insensitive to ADH. The retained osmolar substances like sodium and glucose are excreted with water, which further increases the polyuria. Increase in fluid replacement will also aggravate polyuria. Along with water, sodium is also lost. The distal segment of DCT where final potassium modulation occurs cannot function normally due to rapid delivery of fluid to this segment due to polyuria and results in kaliuresis. The end result is hypovolemia, hyponatremia and hypokalemia. Fluid replacement should include water, sodium and potassium. The calculation of fluid intake is based on the clinical assessment of the dehydration, weight loss and if available biochemical values of serum sodium and potassium and their calculated deficit. Most of the time they may need full maintenance fluid as given for 10% dehydration along with sodium and potassium to maintain fluid status in them. Similar pathophysiological mechanisms operate in diuretic phase of ATN where children may need as much as 200% of maintenance fluid at times with sodium and potassium replacement.
Conclusion

Fluid imbalance, dyselectrolytemia and metabolic acidosis can occur in renal disorders with or without renal failure. In the presence of renal failure the management becomes difficult and some times produce dangerous complications more than the primary defect. Hence dialysis is prefered as a safe method to correct multiple biochemical abnormalities. It also ensures early recovery of renal function.

Bibliography


BOOK REVIEW

Title : Child Intelligence
Authors : Dr. Rajesh Shukla
Review : The author has to be appreciated for having selected this topic of ‘Intelligence in children’. But, unfortunately he has discussed about the main issue in only one part of the book. All other parts, which cover topics such as genetics, embryology, growth and development etc, do not discuss the relevance of them to the intelligence. In the part on intelligence, the author has focused on relatively new area such as emotional intelligence, which does not find a place in day-to-day discussion among the pediatrician. One more novel area is ‘financial intelligence’. Reading about gifted children can be interesting. Throughout the book author has writer the topics in a personnel way adding lot of personal opinions. This makes the reading little easy. This book can be one more companion to basic pediatrics.

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Abstract

Fluid and electrolyte disturbances of endocrine emergencies may be life-threatening but can be highly rewarding if management is carried out promptly and appropriately. A newborn with congenital adrenal hyperplasia presenting as a case of ambiguous genitalia and salt loss needs life-saving treatment promptly. In the absence of ambiguity of genitalia, a high index of suspicion is required to identify this condition. Diabetic ketoacidosis is often the presenting feature of insulin dependent diabetes mellitus at the initial diagnosis. Unless recognized, it may be mistaken for a host of other conditions. Treatment requires careful monitoring. Careful choice of intravenous fluids will be necessary when a child with central diabetes insipidus presents with dehydration.

Key words: Endocrine emergencies, diabetic ketoacidosis, congenital adrenal hyperplasia, adrenal insufficiency

Congenital adrenal hyperplasia

Congenital adrenal hyperplasia (CAH) in the newborn period commonly manifests as a salt-losing crisis, requiring prompt emergency medical care. The clinical features of dehydration, hyponatremia, hyperkalemia, hypoglycemia, hypotension and metabolic acidosis occur rapidly. Soon, if untreated, shock and cardiac arrest will result from deficiency of cortisol and aldosterone. The salt-wasting forms of CAH are

a. Classical form of 21-hydroxylase deficiency – the commonest disorder of CAH (commonly presenting as female pseudohermaphroditism).

b. Lipoid congenital adrenal hyperplasia (male pseudohermaphroditism).

c. 3-β hydroxysteroid dehydrogenase deficiency.

Clinical diagnosis can be suspected in the presence of the following features:

1. Ambiguity of genitalia due to virilisation of a female infant in 21-hydroxylase deficiency.

A fully virilised female infant may sometimes present with normal looking male genitalia. The absence of gonads in the scrotal sac, hyperpigmentation of the body and the presence of uterus on pelvic ultrasonography will serve as useful clues to make a correct diagnosis.

Boys with 21-hydroxylase deficiency have normal male external genitalia and no ambiguity. A high index of suspicion is necessary to make a diagnosis in these cases by the clinical features listed below.
2. Ambiguity of genitalia due to undervirilisation of a male infant (in the case of b and c above).

3. Salt loss occurs in nearly 75% of cases with 21-hydroxylase deficiency. Nearly half the number of cases presents with the first salt-losing adrenal crisis around 6-14 days of age. Serum potassium may be elevated prior to 6 days of age. Salt-losing crisis may occur as late as 6 to 12 weeks of age, often precipitated by a stress.

4. Hyperpigmentation of the nipples, umbilicus, genitalia or very often, the whole body. This finding may require subtle distinction from normal in the case of parent(s) with a dark skin.

5. Unexplained vomiting and dehydration rapidly leading to shock.

6. Convulsions resulting from hypoglycemia, hyponatremia.

7. Previous history of sibling(s) with ambiguity of genitalia or unexplained death.

Fluid management: Prior to commencement of fluids, blood samples are to be quickly obtained for serum electrolytes, cortisol, 17-hydroxyprogesterone (17-OHP), and dehydroepiandrosterone sulphate (DHEAS) and plasma renin activity (PRA).

Restoration of intravascular volume is urgent in the first hour. An intravenous normal saline infusion is given rapidly to correct shock or hypotension at a rate of 20 ml/kg over 20 minutes.

The sodium deficit is calculated using the formula:-

\[
\text{Desired serum sodium level (135 mEq/L) } - \text{ actual serum sodium level in mEq/L } \times 0.6 \times \text{ body weight in kg } = _____ \text{ mEq/L sodium.}
\]

Following the initial therapy, dehydration is to be corrected with 5% dextrose normal saline. Always assume minimum deficit of 10% if the infant is in shock and correct half the deficit within first 8 hours and the rest over 16 hours with isotonic fluids (NS / 5% dextrose ¼ NS). While using 5% dextrose normal saline, one should remember hyperglycemia is likely to occur at times in older children.

If serum sodium is < 120 mEq/L, 4-6 ml/kg of 3% saline infusion may be used, to raise the levels by 1-2 mEq/L/hr or half way towards normal in the first 4-8 hours. Too rapid correction of hyponatremia should be avoided as it may cause central pontine myelinolysis and axonal demyelination leading to irreversible brain damage. These complications are avoided also if one aims for serum sodium level not higher than 135 mEq/L.

Hyperkalemia in general reverts to normokalemia, when serum sodium level rises and fluid volume is replaced. Severe hyperkalemia may be life-threatening, by causing cardiac arrhythmias. If hyperkalemia is persistent or associated with ECG changes, intravenous sodium bicarbonate and calcium and rectal cation exchange resins are useful for rapid correction of serum potassium (protocol given elsewhere). Hypoglycemia should be treated with a bolus of 0.25 g of glucose/kg body weight.

Specific therapy

Hydrocortisone sodium succinate in a dose of 50mg/m² must be given as an intravenous bolus and another 50 to 100 mg/m² should be added to the infusion fluid over the first 24 hours. Fludrocortisone 100 micrograms tablet crushed and given by nasogastric tube will help hyponatremia and hyperkalemia. But if oral feeds or medications cannot be given, hydrocortisone and intravenous fluids may be quite sufficient to
tide over the initial crisis. This is because high
doses of hydrocortisone provide mineralocorticoid action as well (25 mg hydrocortisone = 0.1
mg (100 micrograms) of mineralocorticoid activity).

After a few intravenous doses, hydrocortisone may be changed over to intramuscular route, generally as 15 mg every 6
hourly. Over the next few days, as the infant improves, the dose is tapered rapidly and changed
to the oral route. When the infant is stable, 5 mg
twice daily orally would be adequate.

Fludrocortisone therapy is commenced orally when the infant is ready to feed. 50-100
micrograms per dose Q12H is the usual dose.

Frequent monitoring of serum electrolytes, hydration, body weight and blood pressure are
required for optimizing treatment. Excessive mineralocorticoid dose will cause increased body
weight, hepatomegaly, leg edema, congestive cardiac failure, hypertension and hypertensive encephalopathy. Underdosing with
mineralocorticoid will not correct hyperkalemia and hyponatremia.

**Parent education:** The parents must be given
detailed instructions regarding the nature of the
disease and the need for regular followup and lifelong therapy.

**Points to remember**

**Salt-losing adrenal crisis in the newborn**

1. **Immediate hospitalization essential**
2. **Measure blood pressure**
3. **Collect blood for serum electrolytes and plasma glucose, cortisol, 17-OHP and renin activity**
4. **Start intravenous normal saline infusion immediately**
5. **Inject Hydrocortisone IV 50 mg/m² as a bolus;**
   50 to 100 mg/m² to the infusion; later IM
   15 mg Q6H. Reduce IM dose gradually over the next few days and change to oral when
   infant is ready to feed
6. **Add fludrocortisone 50-100 micrograms bd orally**
7. **When infant is stable, carry out a short ACTH stimulation test**
8. **Parent education essential**
9. **Stress and febrile illness require increased steroid coverage**

**Adrenal insufficiency**

Adrenal insufficiency may be primary,
caused by destruction of adrenal cortex (Addison’s disease), secondary due to deficient
ACTH secretion or tertiary following deficiency of CRH secretion by the hypothalamus.

A high index of suspicion is necessary. Prompt treatment can be highly rewarding. Mortality is high if untreated.

**Helpful clues for diagnosis**

Weakness, fatigue, weight loss, abdominal pain, anorexia, nausea, vomiting, confusion or coma may be the presenting clinical features. Although these are non-specific, these symptoms should alert one to consider adrenal insufficiency, which may be insidious in onset and often go unnoticed unless a crisis occurs during an intercurrent illness or stress situation. Shock may be the initial presentation in a newly diagnosed case of primary adrenal insufficiency. A child previously diagnosed to have adrenal insufficiency, who is not taking stress doses of glucocorticoid during a major illness, accident or surgery may present in the same way. Also when stress doses of hydrocortisone given orally are not retained because of persistent vomiting
or diarrhea the child will show similar clinical presentation.

The other features are
- Hypoglycemic manifestations
- Hyperpigmentation in longstanding cases
- Hypotension
- Adrenal crisis may be sudden following adrenal hemorrhage

**Fluid management:** Adrenal crisis requires prompt life-saving treatment. Treatment of shock and electrolyte abnormalities is urgent. Intravenous normal saline or 5% dextrose saline 20 ml/kg is given as a bolus initially followed with an infusion for rehydration and maintenance. Hyponatremia and hyperkalemia, if severe, will need special attention.

**Specific treatment:** Inj. hydrocortisone 2-4 mg/kg/dose Q3-6H may be given intravenously. Mineralocorticoid therapy is not urgent. The requirement of sodium can be given by intravenous normal saline infusion. In the case of severe hyperkalemia, treatment with intravenous calcium and / or bicarbonate, oral or intrarectal potassium-binding resin, keyexalate or intravenous glucose and insulin infusion will be needed.

The precipitating cause of adrenal crisis needs to be searched for and treated appropriately. When the child’s condition is stable, in new cases, or short synthetic ACTH stimulation test may be carried out to confirm the diagnosis.

Glucocorticoid therapy is tapered over 1 to 5 days to oral maintenance dose. Mineralocorticoid therapy is commenced as oral fludrocortisone 100 to 150 micrograms / day after the saline infusion is stopped.

**Stress dosing:** In children who have already been on physiologic replacement doses, stress dosing involves 2-4 times the daily replacement dose. Aim to gradually decrease to maintenance dose over the next 3-4 days depending on the general condition of the patient and his ability to tolerate orally. Children who cannot retain oral hydrocortisone, may be given intravenous hydrocortisone sodium succinate 50 mg/m²/dose followed by 25 mg/m²/dose intramuscularly every 6-8 hours.

**Physiologic replacement:** Hydrocortisone 10-15 mg/m²/24 hours to be given in two or three divided micrograms twice daily.

**Points to remember**

**Adrenal crisis**

1. *Immediate hospitalization essential; measure blood pressure*
2. *Collect blood for serum electrolytes and plasma glucose, cortisol, renin activity*
3. *Start intravenous normal saline infusion immediately; 20ml/kg as a bolus, followed by maintenance fluid*
4. *Inject Hydrocortisone IV 25-50 mg/m² as a bolus; add a similar dose to the infusion; later change to IM and oral gradually tapered over the next few days to maintenance dose*
5. *Add fludrocortisone 50-100 micrograms bd orally*
6. *Identify and treat the precipitating infection appropriately*
7. *When infant is stable, carry out a short ACTH stimulation test*
8. *Parent education essential*
9. *Stress and febrile illness require increased steroid coverage*

**Diabetic ketoacidosis**

**Definition:** Diabetic ketoacidosis (DKA) is a life-threatening condition in which profound insulin
deficiency combined with counter-regulatory hormone excess leads to hyperglycemia, severe catabolic state with excessive lipolysis and unrestrained fatty acid oxidation producing ketone bodies which leads to metabolic acidosis, ketosis, dehydration, electrolyte depletion and ketonuria. Polyuria, polydipsia, anorexia, nausea, vomiting and abdominal pain are commonly encountered.

**DKA** is said to exist in the presence of

1. Blood glucose $\geq 300$ mg/dl
2. Acidosis (arterial pH $< 7.3$ and serum bicarbonate $< 15$ mEq/L)
3. Marked glycosuria (+++ or ++++)
4. Severe ketonuria (++ to ++++)

Severe DKA: blood pH $< 7.1$, serum bicarbonate $< 5$ mEq/L;

Moderate DKA: pH 7.1 – 7.2, bicarbonate 5 – 10 mEq/L;

Mild DKA: pH >7.2, bicarbonate 10-15 mEq/L.

DKA occurs in 20-40% of children as the presenting manifestation at initial diagnosis and in children with known diabetes who omit insulin doses or who do not appropriately manage an intercurrent illness. Although potentially life-threatening, with proper recognition and appropriate therapy given promptly, it is rarely fatal.

**Pathophysiology of fluid and electrolyte disturbances in DKA**

**Fluids:** Volume contraction is the hallmark of DKA. This is an effect of glycogenolysis, gluconeogenesis and impaired glucose utilization resulting in hyperglycemia causing osmotic diuresis (urine produced is the same osmolarity as $\frac{1}{2}$ normal saline i.e., water deficits are in excess of sodium).

**Potassium:** DKA invariably causes severe potassium loss; so even though serum potassium is normal or even high at admission, there is depletion of total body potassium. Loss of potassium is over the range of 3-10 mEq/kg body weight resulting from

a) shift of potassium to extracellular space in acidosis
b) protein catabolism compounded by hyperaldosteronism
c) osmotic diuresis

With insulin administration, hypokalemia occurs due to intracellular shift of potassium.

**Chloride:** Excessive chloride administration (as NaCl, KCl etc) aggravates acidosis.

**Phosphate:** Phosphate is depleted in acidosis. Phosphate promotes formation of 2,3 DPG which shifts oxygen dissociation curve to the right. Tissue oxygenation improves the metabolic acidosis. Hence phosphate depletion worsens acidosis. When acidosis gets corrected with insulin administration, there is aggravation of phosphate depletion.

**Sodium:** In DKA, there is pseudohyponatremia because of

a) Osmotic effects of glucose. The corrected sodium in mEq/L = measured sodium in mEq/L + 0.016 (measured blood glucose in mg/dl – 100). Ex. If serum sodium measured = 132 mEq/L and blood glucose = 600 mg%, then the corrected serum sodium = 132 + 0.016 × (600 – 100) = 140 mEq/L.

b) Severe hypertriglyceridemia which is common in DKA. Serum sodium concentration decreases by 1.0 mEq/L at serum lipid concentration of 460 mg/dL.
**Bicarbonate:** Serum bicarbonate is always low, but true deficit is not present because ketoacids and lactate are metabolized to bicarbonate during therapy.

**Principles of fluid and electrolyte management in DKA**

1. All patients with DKA are dehydrated. Hence hydration should be assessed and URGENT treatment is commenced to restore normalcy. But at the same time, dehydration and hyperglycemia should be treated with caution as rapid treatment may result in cerebral edema. Mannitol should be readily available

2. Fluids to restore circulation and maintain brisk diuresis

3. Correction of insulin deficiency

4. Correction of electrolyte disturbances

5. Close monitoring of the patient

6. Search for underlying causes for metabolic decompensation

**Initial hydration:** Assess degree of dehydration (usually 10%) and aim to replace this deficit over 24 hours. Fluid status should be reassessed every few hours, since continuing polyuria may lead to excessive losses and require extra fluid replacement intravenously. Start with intravenous normal saline or lactated Ringer’s solution. It may require 15-20 ml/kg in the first hour to expand peripheral circulation. If capillary refill time is > 3 seconds, indicating poor peripheral perfusion, a second bolus may be provided. Rarely, colloid (e.g. albumin) is needed in shock.

As hyperosmolarity along with hyperglycemia is the rule in DKA, the initial hydrating fluid should be normal saline. Rate of correction: half of the calculated deficit over the first 8 hours and the remaining half over and the next 20-30 hours.

If normotensive after first hour of normal saline – change over to ½ normal saline. If hypernatremic or hyperchloremic acidosis occurs, change over to ¼ normal saline.

Glucose replacement is commenced as 5% dextrose normal saline solution, when blood glucose drops to 250 mg/dL. The aim is to maintain serum glucose in the range of 250-300 mg/dL for 24 hours to allow slow equilibration of the osmotically active substances across the cell membranes.

Potassium chloride infusion added to the intravenous fluids is given after the first hour of rehydration. Replacement is usually at the rate of 10-20 mEq/L, but may need up to 40-60 mEq/L if there is protracted vomiting, hypokalemia or persistent acidosis. If patient is oliguric at the end of the first hour of rehydration, give potassium, only if serum potassium is < 4 mEq/L or an ECG recording shows features of hypokalemia. Periodic potassium estimation is required.

Bicarbonate administration (1 mmol/kg/dose over 30 minutes by infusion) is recommended if

1) pH is < 7.0
2) Hemodynamic instability with pH < 7.1, shock or renal failure is present
3) Hyperkalemia with ECG changes is noted.

Sodium bicarbonate should not be given as an intravenous bolus as it may precipitate cardiac arrhythmias.

**Diabetic ketoacidosis treatment protocol**

Fluids and insulin therapy are given as per the protocol (Table 1)

**Monitoring**

DKA management involves close monitoring of the patient’s vital clinical signs,
mental status and lab parameters. The lab parameters include: Initial – Plasma glucose, sodium, potassium, bicarbonate, chloride, urea, creatinine, calcium, magnesium, phosphate, ketones, lactate, CPK, LFT, urine ketones (acetoacetate, acetone, beta-hydroxybutyrate), ECG, CBC, ABG. Subsequently plasma glucose and electrolytes hourly till stabilised for severe Dka and 2-4 hourly for mild to moderate DKA. Serum calcium, magnesium, phosphate are to be monitored closely; blood urea 6-24 hrs, urine ketones on every urine sample passed till negative thrice.

**Flow sheet**

<table>
<thead>
<tr>
<th>Mental status</th>
<th>Vital signs</th>
<th>Insulin dose</th>
<th>Fluids and electrolytes given</th>
<th>Urine output</th>
<th>Lab parameters</th>
</tr>
</thead>
</table>

**Goals**

1) Hemodynamic stability rapidly

2) Correction of ketoacidosis fully in 12-36 hrs

**Insulin therapy**

Insulin infusion is prepared by adding 50 units of short acting insulin (e.g., Actrapid) to 500 ml of normal saline, so that 10 ml of the solution will contain one unit of insulin. Prepare this solution afresh every 24 hours and use in a pediatric microdrip set or infusion pump.

Commence insulin infusion at the rate of 0.1 unit/kg/hr when diagnosis is confirmed and after rehydration has been commenced. The rate of the infusion is varied according to the blood glucose level. If acidosis has not improved within a few hours, insulin rate may be increased by 0.05 unit/kg/hr till improvement occurs.

Aim for a blood glucose fall of 100 mg% per hour. If facilities for insulin infusion are not available, 0.2 unit/kg of soluble insulin may be given as an initial dose and later, 0.1 unit/kg/hr intramuscularly. Subcutaneous injections in a

---

**Table 1. Diabetic ketoacidosis fluids protocol**

<table>
<thead>
<tr>
<th>Time</th>
<th>Fluids</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st hour</td>
<td>10-20 ml/kg IV bolus NS or RL</td>
</tr>
<tr>
<td>2nd hour until DKA resolution</td>
<td>0.45% NaCl: *with 20 mEq/L potassium as KCL 0.45%NaCl in 5% glucose if blood sugar&lt;250 mg/dL</td>
</tr>
</tbody>
</table>

The initial IV bolus is considered part of the total fluid allowed in the first 24 hr and is subtracted before calculating the IV rate.

\[
\text{IV rate} = \frac{85 \text{ ml/kg} + \text{maintenance - bolus}}{23 \text{ hours}}
\]

**Sample calculation for 30 kg child:**

1st hr = 300 ml IV bolus 0.9% NaCl or RL

2nd and subsequent hrs = \( \frac{(85 \text{ ml} \times 30) + 1750 \text{ml} - 300\text{ml}}{23\text{hr}} = 175\text{ml/hr}(0.45\% \text{NaCl with 20mEq/L KCL}) \)

NS – 0.9% sodium chloride; RL – Ringer lactate solution;

*Additional potassium is given as per initial potassium levels and periodically monitored.
dehydrated patient will not be effectively absorbed. Blood glucose level must be checked every 2 hours and repeated regular insulin injections may be necessary. The dose of insulin must be adjusted against the blood glucose level.

**Maintenance fluid therapy**

When the blood sugar level falls > 100 mg/dl/hr or to ~300 mg/dl, a glucose infusion is started at the rate of 3-4 g of glucose/unit of insulin being infused, aiming for a drop of blood sugar 50 mg/dl/hr to ~150 mg/dl at which level it should be maintained throughout the treatment for DKA.

Maintenance fluids are calculated as shown in Table 1. Generally ~20 mEq/L of potassium chloride is required for the first 24 hours of therapy. Insulin therapy shifts potassium intracellularly thereby lowering serum potassium. When serum potassium level is <5.6 mEq/l potassium infusion is commenced. As potassium phosphate and acetate are not easily available, potassium chloride is given.

**Commencing subcutaneous insulin**

If an alert child, normally hydrated and haemodynamically stable, with the blood sugar in the near normal range and acidosis resolved, wishes to take oral fluids and feeds, intravenous insulin may be changed over to subcutaneous insulin at the next meal. This usually takes 12-18 hours.

**Complications**

1) Cerebral edema – common in infants and may be fatal. Treatment is difficult and hence care should be taken to avoid this complication as best as possible. Specific cause is usually not identified. Aggressive hydration with hypotonic fluid may worsen the situation. Cerebral edema may cause respiratory arrest, or severe or fatal brain damage.

2) Adult respiratory distress syndrome

3) Bronchial mucus plugging

4) Arterial and & venous thrombosis

Thus, the principles of fluid and electrolyte therapy and correction of acidosis outlined above form the cornerstone in the treatment of DKA and often result in clinical and biochemical improvement even before the initiation of insulin. Insulin restores the intermediary metabolism.

**Points to remember**

**Diabetic Ketoacidosis**

1. Diagnosis is essential; otherwise may be mistaken for viral encephalitis etc.

2. Severe dehydration is usual; after initial rapid infusion, continue rehydration until hypotension improves and circulation is restored

3. Start intravenous normal saline infusion immediately; 20ml/kg as a bolus, followed by maintenance

4. Continuous insulin infusion 0.1 unit/kg/hr, tailored according to the blood sugar level

5. Severe total body potassium depletion is usual; if oliguric, potassium is given only if serum potassium is <4 mEq/L

6. Pseudohyponatremia is common – for every 100 mg rise in blood glucose add 1.6 mEq/l to the actual serum sodium level obtained

7. Cerebral edema is to be avoided during therapy by lowering the blood sugar 100 mg/hr gradually and not faster

8. Close monitoring is vital

9. Precipitating cause for DKA should be searched for
**Diabetes insipidus**

Diabetes insipidus (DI) is a disorder which manifests clinically with polyuria and polydipsia. The urine produced is hypotonic.

In children, diabetes insipidus may be classified as

1) Central or hypothalamic, where there is inability to secrete vasopressin
2) Nephrogenic where there is inappropriate renal response to vasopressin
3) Primary polydipsia where the primary pathology is ingestion of large quantities of fluid

**Pathophysiology:** Normally hyperosmolarity of serum, hypovolemia and hypotension are detected by osmoreceptors, volume receptors (in the cardiac atria and pulmonary veins) and baroreceptors in the carotid sinus. These stimulate the secretion of vasopressin and also induce thirst. Vasopressin acts on the collecting tubules and the ascending limb of the loop of Henle and periglomerular tubules and causes increased reabsorption of water. Thirst causes increased water ingestion. These two mechanisms help in reducing the hyperosmolarity and hypovolemia.

In DI, because of the inadequate secretion or unresponsiveness to vasopressin, water does not get reabsorbed in the kidneys thereby failing to concentrate the urine.

**Symptoms**

<table>
<thead>
<tr>
<th>Persistent polyuria</th>
<th>Nocturia</th>
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<tbody>
<tr>
<td>Increased thirst and polydipsia</td>
<td>Irritability</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Coma</td>
</tr>
<tr>
<td>Decreased appetite</td>
<td>Hyperthermia</td>
</tr>
<tr>
<td>Weakness, lethargy, dullness</td>
<td>Hypotension</td>
</tr>
</tbody>
</table>

**Diagnosis:** Early morning sample of urine specific gravity <1.005; urine osmolality < 200 mosm / kg; and serum sodium elevated

**Fluid management**

Water intake is advised ad libitum. The aim of the treatment is to decrease thirst and polyuria to an acceptable level to allow the patient to maintain a normal lifestyle. Overtreatment should be avoided and a fine balance should be struck between the relatively benign course of DI and adverse consequences of overtreatment – the most dreaded being hyponatremia.

In an ambulatory patient with free access to water, DI does not usually produce complications of hypernatremia. In a patient who presents with dehydration, correction of dehydration with full strength lactated Ringer’s should be given. Hypernatremia should be corrected with 0.45% saline with an aim not to reduce the serum sodium > 10 mEq/L/24 hrs in order to avoid the risk of cerebral edema.

8-Deamino D-Arginine vasopressin (DDAVP) nasal spray 10 µg once or twice a day can be used in older children as a definite therapeutic agent in central DI. The urine specific gravity can be monitored using a urinometer (a lactometer may be used too) at home before each dose of the nasal spray. Periodic monitoring of serum electrolytes may be done. Water retaining agents like chlorpropamide and indomethacin and natriuretic agents like thiazide diuretics, amiloride and indapamide may be used.

The treatment of congenital nephrogenic DI is difficult and that of secondary nephrogenic DI is to correct the underlying cause. These children should be provided with high calorie diet with minimal osmotic load. Great care must be taken to avoid dehydration. Thiazides or sometimes, high dose DDAVP may bring about some improvement.
Syndrome of inappropriate ADH secretion

This is a clinical condition of euvolemic or hypervolemic hypoosmolarity with hyponatremia.

Pathophysiology: In this condition, the secretion of ADH is not inhibited by either low serum osmolality or expanded intravascular volume. So the plasma level of vasopressin is elevated at a time during which its physiologic secretion should normally be suppressed. The net result is that the child is unable to excrete water leading on to water intoxication and hyponatremia. Along with increase in extracellular fluid volume, intravascular volume also increases and more sodium is excreted in urine in an attempt to normalize the intravascular volume.

Management: The diagnosis is confirmed by hypoosmolality of serum < 275 mosm/kg, serum sodium < 135 mEq/L, inappropriate urine osmolality > 100 mosm/kg (but less than serum osmolality) with normal renal and adrenal function and increase in urine spot sodium more than 20 mEq/L with normal salt and water intake. Hyperglycemia and pseudohyponatremia must be excluded.

Neurological symptoms like seizures appear when there is a rapid fall (within 48 hrs) in serum sodium to < 120 mmol/l. These patients require immediate treatment with infusion of hypertonic (3%) saline or a combination of normal saline and furosemide. The latter combination has the advantage of not rapidly expanding ECF volume in an already volume expanded patient. The goal is to raise the serum sodium level above 125 mmol/L, at a rate of not more than 10 mmol/L in 24 hours.

However, there is a serious risk involved with rapid correction of serum sodium. Patients can develop pontine and extra pontine myelinolysis. Studies in experimental animals have shown that chronic hypoosmolality predisposes to opening of the blood brain barrier after rapid correction of sodium. Opening the blood brain barrier can lead to subsequent myelinolysis through an influx of complement which is toxic to the oligodendrocytes that manufactures and maintains the myelin sheath of neurons. The factors that predispose to the development of myelinolysis are

1) Severity and duration of pre-existing hyponatremia – the more severe and prolonged the hyponatremia the more the changes of developing myelinolysis. It is known that shorter duration of hyponatremia does not cause sufficient brain volume regulation.

2) Nutritional status of the patients – malnutrition predisposes to myelinolysis.

In short, the correction of hyponatremia should carefully be weighed against the risk of development of myelinolysis. In acute symptomatic hyponatremia, the risk of neurological complications due to hyponatremia per se exceeds the risk of development of myelinolysis and therefore serum sodium level should be brought up rapidly.

In chronic asymptomatic hyponatremia, neurological symptoms are minimal. However these patients can develop demyelination after rapid correction of sodium because of greater degree of brain volume regulation through electrolyte and osmolyte losses. These patients should be treated with fluid restriction (previous day’s urine output + insensible fluid loss).

In chronic symptomatic hyponatremia controlled and limited increase in hypoosmolality should be the aim. Serum sodium correction should not exceed 10 mEq/L in the first 24 hrs and 18 mEq/L in the first 48 hours of treatment.
The end points of treatment regardless of the initial rate of correction would be 1) control of symptoms, 2) serum sodium > 120 mEq/L, 3) total serum sodium correction > 20 mEq/L.

When any of these end points are reached, slower acting therapy like fluid restriction and unrestricted salt intake should be advised. Close monitoring of serum sodium is essential in the management of SIADH.

References


FLUID GUIDELINES FOR SURGICAL PROBLEMS IN CHILDREN

* Shanbhogue HR
** Madhu R
** Balagopal S

Abstract: Fluid guidelines in children are very important for pediatricians, pediatric surgeons and people in the casualty who deal with pediatric emergencies. It is a fallacy to think that children are ‘mini-adult’ and give - half the required fluids as for an adult, as fluid requirements in children are totally different. In this article we dwell upon fluid guidelines for children in general and then take up specific conditions like burns, trauma, preoperative patients, pyloric stenosis and intestinal obstruction and provide the guidelines for fluid requirements. The fluid requirements for these surgical conditions are specific so as to correct the metabolic and electrolyte imbalances in these conditions.

Key words: Burns, pyloric stenosis, paradoxical aciduria, transepithelial water loss, intestinal obstruction, trauma.

Fluid therapy in surgical conditions differs from other medical disorders. Fluid therapy during surgery is influenced by complex interaction between anesthetic drugs, abnormal losses and stress response which includes autonomic, hormonal and inflammatory responses. Apart from surgery there are some special clinical situations where accurate assessment of fluid and electrolyte status in mandatory for a successful outcome. Fluid and electrolyte status is assessed by clinical parameters such as weight, dryness of mucous membrane, skin turgor, heart rate, pulse volume and blood pressure. Monitoring urine output and maintaining strict fluid balance chart, estimation of haematocrit, electrolytes, renal parameters and measuring central venous pressure will supplement the clinical assessment.

1. Perioperative fluid management

Water and electrolyte deficit caused by vomiting or stasis due to intestinal obstruction should be corrected prior to surgery. Uncorrected hypovolemia might have been compensated by increase in heart rate and vascular resistance prior to surgery. When baroreceptor reflexes are abolished by anesthesia, severe hypotension may develop. Hence preexisting deficit should be corrected by Ringer Lactate or normal saline. When there is no deficit, intravenous maintenance fluid is started.

Intraoperative fluid management

This includes the following

1. Current maintenance fluid
2. Replacement of third space and evaporative losses
3. Replacement of extraordinary losses such as hemorrhage

Maintenance fluid during surgery
Body weight | Fluid rate |
--- | --- |
1 – 10 kg | 4 ml/kg/hour |
11 – 20 kg | 40 ml + (2 ml/kg for every kg above 10) per hour |
> 21 kg | 60 ml + (1 ml/kg for every kg above 20) per hour |

Example: 22 kg child requires

\[(4 \times 10) + (2 \times 10) + (1 \times 2) = 62 \text{ ml/hour}\]

Fluid used – D5W with 0.25 NS or electrolyte maintenance solution

**Replacement of third space and evaporative loss**

- **Major surgery**
  - (Thoracic, abdominal) surgery: 10 ml/kg/hour of surgery
- **Surgery like herniorraphy**
  - 5 ml/kg/hour of surgery
- **Surgery involving extremities**
  - 2 ml/kg/hour of surgery

Fluid used: Isotonic crystalloids such as ringer lactate

**Replacement of extraordinary losses**

This can occur because of relative hypovolemia caused by anesthetic drugs and blood loss which is recognized by appearance of hypotension and tachycardia

Fluid therapy during surgery should consider the following points

1. Avoid excessive administration of fluid
2. Only electrolyte solution should be used and 5% Dextrose solution should not be used in isolation
3. Fluid used should contain no potassium or little potassium because of danger of hyperkalemia due to release of intracellular potassium caused by tissue trauma.

**Postoperative fluid therapy**

Fluid therapy should not exceed 85% of maintenance because of possible SIADH after surgery. After 24 hours, usually maintenance therapy is gradually resumed.

**Fluid management in a trauma victim**

In a trauma victim shock is commonly due to hemorrhage in addition to other problems like hypoxia. Following facts should be considered in fluid management in a trauma victim.

1. For a vascular access, cannulate larger veins using largest possible catheter. Ideal to start a second intravenous line within first few minutes of resuscitation.
2. If intravenous access is difficult, intraosseous cannulation using intraosseous needle should be done within 90 seconds or after three attempts at intravenous access, whichever is earlier. All medications, infusions and blood products can be administered through intraosseous route. Common site preferred is upper end of tibia on the medial surface, but avoid fractured bones.
3. Generally by every 1ml of blood lost can be replaced by 3 ml of crystalloids like Ringer lactate or normal saline. IV bolus is given at the rate of 20ml/kg body weight as fast as possible, in decompensated shock (shock associated with hypotension) and 20ml/kg over 20 minutes in compensated shock (shock with normal BP). When shock is not corrected in 2 or 3 boluses blood transfusion should be the next option. Blood transfusion should be the first option in pelvic fractures, splenic trauma, femoral fractures and major thoracic injuries. Where a major vessel bleed is anticipated blood transfusion is mandatory in addition to arresting the bleed by surgical intervention. Hence blood grouping should
be done and arrangements should be done for cross matching even when first Ringer lactate bolus is infused.

3. Fluid guidelines in pyloric stenosis

Pyloric stenosis classically occurs in a first born male child, between 3rd to 6th week of life. The triad of IHPS is nonbilious, projectile vomiting, mass, visible gastric peristalsis. It is one of the common pediatric surgical problems.

Due to prolonged vomiting, child usually presents with

1. Dehydration (of varying degrees)
2. Hypochloremic, hypokalemic metabolic alkalosis
3. Paradoxical aciduria during late stages
4. Rarely presents with hypocalcemic tetanic seizures

Reasons for this electrolyte imbalance:

Child predominantly vomits gastric contents (HCl) with sodium. So child develops hypochloremic alkalosis. To conserve Na⁺, kidney tends to absorb Na⁺ at the expense of K⁺ ions initially and then H⁺ ions (due to aldosterone surge). So H⁺ ions are excreted in the urine in extreme cases producing the classical paradoxical aciduria.

Now it must be clear that pyloric stenosis is initially a medical emergency and the metabolic abnormalities have to be treated first before taking the child for surgery.

Management

(a) Fluid resuscitation

Ideal fluid of choice depends on degree of dehydration

Mild dehydration (5%) of body weight loss  
- 0.33 Normal saline

Moderate dehydration (5 – 10%) body wt. loss  
- 0.45 Normal Saline

Severe dehydration (>10%) body wt. loss  
- 0.9 Normal Saline

Generally the fluid of choice would be 0.45% Normal saline in dextrose. Fluid is given at 1½ times the maintenance requirement during the first 12 hours. Hydrate the child for 24 hours, check serum electrolytes every 24 hours and then take up for surgery once electrolyte imbalance is corrected.

(b) Electrolyte imbalance correction

0.45 NS with dextrose corrects hyponatremia. For hypokalemia, potassium supplementation (2 mEq/kg) to the maximum of 20 mEq can be added over 24 hours. In the average case presenting in clinical practice, potassium supplement is not necessary. Alkalosis is taken as corrected if HCO₃ level is less than 30 mEq. Rehydration with IV fluids corrects paradoxical aciduria.

4. Fluid guidelines in burns

Burns is a catastrophe to the skin covering whatever the degree. In the first 48 hours since epithelium is lost and the cell wall integrity is damaged, there is massive exudate of fluid. This fluid loss has to be replaced to the child to prevent hypovolemic shock.

Various fluid calculations available

1. Brooks formula  - 2 ml/kg x % of burns  
   + Maintenance fluid

2. Parkland formula - 4 ml / kg x % of burns  
   + Maintenance fluid

3. Shirner Burns Institute formula - 5000 ml / m² BSA of burns + Maintenance fluid  
   2000ml/m² BSA (BSA – Body Surface Area)

Parkland formula is the one most frequently followed.
The rule of 9 of Wallace to calculate the percentage of burns may not be applicable to children. So a modified formula is given age wise. In a child more important than the weight is the body surface area.

Example for one-year-old child – wt. of 10 kg, with 20% burns, the fluid requirement would be

\[
\text{Parkland formula} = 4 \text{ ml} \times 10 \text{ kg} \times 20 \text{\% of burns} + \text{maintenance}
\]

\[
\text{Burns requirement} = 800 \text{ ml}
\]

\[
\text{Maintenance fluid 100 ml} \times 10 \text{ kg} = 1000 \text{ ml}.
\]

\[
\text{Total requirement} = 1800 \text{ ml}.
\]

**Choice of fluid:** The ideal fluid is Ringer lactate. 50% of fluid calculated is given in the first 8 hours from the time of burns. Remaining 50% fluid is given over the next 16 hours in equal amounts. This massive fluid replacement is because of excessive TEWL (trans epithelial water loss). Colloids are not recommended in the first 24 hours.

So, as per the example 900 ml RL is given in the 1st 8 hours 450 ml RL is given in the 2nd 8 hours 450 ml RL is given in the 3rd 8 hours.

**Monitoring for adequacy of fluid replacement:**

(a) Urine output in a child with burns < 4 year is 1 ml / kg / hr while urine output in a child > 4 years is about 30 – 50 ml / hr.

(b) Blood Pressure > 100 mm Hg, pulse rate < 100 per minute.

Other signs for monitoring for fluid balance are level of sensorium, central venous pressure, peripheral circulation and electrolytes.

**Next 24 hours**

Once the initial critical 24 hours are over the fluid losses by TEWL, become lesser. Nutritional aspects become important. The ideal fluid of choice would be 5% dextrose with 0.45 Normal Saline. 5% albumin may be needed if indicated.

Beyond 48 – 72 hours, fluid requirements become secondary as child starts taking oral feeds well. The nutrition and infection are to be specifically attended after this period.

5. **Fluid guidelines in intestinal obstruction**

Fluid losses related to gastric drainage are replaced in equal volumes with Ringer Lactate solution if the drainage is bilious and 5% dextrose in 0.45% normal saline if the gastric drainage is clear. 7

- Post operative patients require less fluid because of ADH and cortisol release.
- Stomal effluent losses to be replaced (volume by volume) by Ringer Lactate solution eg. jejunostomy, ileostomy.
- 20ml/kg of Ringer Lactate solution is given as a bolus in 30 minutes if there is a suggestion of 3rd space interstitial fluid shift, which is reflected, by poor urinary output and high urine specific gravity.

Body weight, skin turgor, urine osmolality and urine specific gravity are reasonable means to assess fluid requirements.

- The blood loss occurring in intussusception and Meckel’s diverticulitis, necrotising enteritis has to be replaced volume by volume by fresh blood.
- 20 ml of blood per kg is a safe transfusion in the child.
- Potassium and sodium requirements are 2 to 3 mEq per kilogram per day.
- Insensible loss through skin is approximately 20ml/kg/day and should be increased if there is fever or child is undergoing radiant warmer therapy.
The child requiring fluid therapy in general should be subjected to:

a) Clinical signs like weight, skin turgor, BP, pulse rate.
b) Periodic electrolyte estimations.
c) Central venous pressure.
d) Urine volume and specific gravity.

**Bibliography**


**NEWS AND NOTES**

**IAP BANGALORE CME 2004**

IAP Bangalore(BPS) is organizing the annual CMEP on June 12th and 13th 2004 at Bangalore. The theme of this year’s CME is “PEDIATRICS FOR TODAY AND TOMORROW.” CME will consist of symposia, case discussions and lectures by distinguished faculty from various parts of the country and abroad. Registration is limited and will be done first come first served basis. The registration fee is Rs 400/- and Rs 250/- for PG students. The fee should be sent as demand draft in in favour of CMEP-2004 payable at Bangalore before 15th may 2004.

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**National Conference**

**IAP Chapter on**

**Growth and Development & Behavioral Pediatrics**

**September 11th and 12th, 2004, Guwahati, Assam**

**Registration Fee**

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## COMPOSITION OF COMMERCIALLY AVAILABLE INTRA VENOUS FLUIDS

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<th>Type</th>
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* Different commercial preparations may have slight variation in the electrolyte composition (Refer label for exact composition)
Commercially available electrolyte and dextrose stock solution

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<thead>
<tr>
<th>Solution</th>
<th>Concentration</th>
<th>Available form</th>
<th>Equivalents</th>
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<td>7.5%</td>
<td>10 ml ampoule</td>
<td>1 ml = 1 mEq of HCO₃⁻ + 1 mEq of sodium</td>
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<tr>
<td>2. Potassium chloride</td>
<td>15% w/v</td>
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<td>1 ml = 2 mEq of potassium</td>
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<td>3. Calcium gluconate</td>
<td>10% w/v</td>
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<td>1 ml = 9.3 mg of calcium</td>
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<td>4. Magnesium sulphate</td>
<td>50% and 25%</td>
<td>2 ml ampoule</td>
<td>If 25% Magnesium 4.15 mOsm/dL</td>
</tr>
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<td>5. Sodium chloride</td>
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<td>1 ml = 0.5 mEq of sodium</td>
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<td>6. 25% Dextrose</td>
<td>25 w/v 25G/100ml</td>
<td>10 ml ampoule and 25 ml ampoule</td>
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<td>50 w/v 50G/100ml</td>
<td>25 ml ampoule</td>
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</table>

Compiled by Dr. Malathi Sathyasekaran, Dr. T.L. Ratna Kumari and Dr. S. Shanthi

**NEWS & NOTES**

**IV National Conference of Adolescent Chapter of IAP**

**&**

**First National Conference on tribal child health**

IAP Raipur invites you on 11th & 12th of September 2004 for Twin National Conferences, being organised for first time in the history of IAP-Adolescent Conference will be devoted to the them of ‘Adolescent sexuality’, speakers of National repute will be the faculties. Registration fees for twin conference will be 800/-. First fifty delegates will enjoy free a/c room facilities by the organising committee. Scenic beauty of Baster is awaiting your arrival. For details contact: Dr.Anoop Verma, Swapnil Nursing Home & Research Center, Civil Lines, Raipur, CG, Email: anoopve@yahoo.com

**XXIII Annual State Conference of Indian Academy of Pediatrics, WB Branch**

**Organised by:** IAP Tamralipta Branch

For further information please contact:

Dr.Sutapa Ganguly, Secretary, IAP WB, Oriental Apartments, 15-C, Canal Street, Flat H1, Kolkata 700 014

Dr. Arup Roy, President, IAP Tamralipta Branch, Doctor’s Quarters, Type II/3/2, Haldia SD Hospital, PO.Khanjanchak 721 602, Purba, Mendipur.
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Pioneers in
Cephalosporins
ABDOMINAL MASSES (CONT.)

* Vijayalakshmi G  
** Natarajan B  
*** Ramalingam A

This time we will see benign masses and how to make a diagnosis and plan treatment without unnecessary, harassing and time consuming tests. Benign masses do not produce worrying symptoms and so present after they have grown to a considerable size and become clinically obvious. When the mother or the pediatrician makes out a mass, the first imaging modality is ultrasound as you all would be aware. Broadly speaking, if ultrasound picks up a cystic lesion it is a benign pathology. The choledochal cyst, mesenteric cyst and the omental cyst are some examples. These can be tackled by the pediatric surgeon without further investigations.

Fig. 1 shows a black or cystic, rounded lesion. The location is sub hepatic, just anterior to the portal vein. Therefore this is a cystic dilation of the common bile duct or a choledochal cyst. It can be accompanied by a variable involvement of the intrahepatic biliary duct system and the extent of this will help you to classify the type. The condition can present with pain or recurrent jaundice with fever. US will not only reveal the location, the wall and the content, but also the presence of any debris or stasis calculi. Remember you should not mistake a distended gall bladder for a choledochal cyst. Always make sure that the gall bladder is visualized and for this US is useful because it can scan in multiple planes. If you cannot see the gallbladder, rescan on a fat-free diet. If you still cannot see the gall bladder the next step is to do an isotope scan. Fig. 2 is a CT picture of a choledochal cyst showing the same features as US. It has not revealed any additional information. Ultrasound studies are sufficient to confirm the diagnosis and delineate the anatomical type of choledochal malformation. Radionuclide excretion studies are useful in situations where the gall bladder is not seen or when there is a doubt between a distended gall bladder and a choledochal cyst.

In the previous issue we saw, that the presence of bowel loops with peristalsis coursing through a hypo echoic mass means that they are enlarged mesenteric nodes. Similarly bowel loops passing through or around a cystic lesion mean it is a mesenteric cyst. (Fig. 3). Mesenteric cysts are peritoneal. Therefore they will be surrounded by bowel loops.

Fig. 4 shows a large, well-defined cyst in a two-year-old child. This was diagnosed as ascites and the child also received a full course of anti-tuberculous treatment. Since the cyst kept increasing in size the child was brought to the Institute of Child Health and Hospital for Children. The US shows a large, very anteriorly placed cyst. All the air containing bowel loops are displaced posteriorly. This is an omental cyst.
Fig 1  Choledochal cyst. The GB (g) is also seen.

Fig 2 Choledochal cyst. Sub-hepatic location.

Fig 3 Mesenteric cyst. (c) Look at the septations.

Fig 4 Omental cyst.

Unlike the mesenteric cyst, which has a number of criss-cross septations, the omental cyst is devoid of septae or has only one or two. A CT of this condition will show a large, well-defined cyst with bowel loops characteristically clumped up together posteriorly and superiorly near the spine.

The child had a distended abdomen and there were no other disturbing clinical symptoms.
Therefore clinically it can be mistaken for ascites. But ultrasound will help in differentiating between the two. The omental cyst has a definite wall. But in ascites, as in Fig. 5, there is no wall and bowel loops will be found floating within. This is akin to the area of resonance around the umbilicus that you can elicit on percussion in a supine position.

In the next issue we will see some more benign masses that we can analyse based on location and content.

**Fig 5** Ascites. Floating bowel.(B)

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**NEWS AND NOTES**

**PALS COURSE**

**Date:** October 16th and 17th 2004  
**Venue:** Ludhiana (IAP-ICC, Punjab State Branch)  
**Course Coordinator:** Dr. Daljeet Singh  
**Contact:** Dr. Rakesh Gupta or Dr. Sukhmeet Singh, Garg Nursing and Maternity Home, Tehsil Road, Jagraon. Ph: 01624-222900, 9815622900 and 9814011170. Email: dranitarakesh@rediffmail.com, sssukhmeet@rediffmail.com

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**IAP State Branch Rajasthan**  
**Rajpedicon – 2004 – 32nd Annual State Conference**

**Date:** 23rd and 24th October 2004  
**Venue:** Umaid Hospital, Jodhpur.

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<td>PG Student</td>
<td>250/-</td>
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<td>350/-</td>
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<tr>
<td>PG Student’s Spouse</td>
<td>100/-</td>
<td>150/-</td>
<td>150/-</td>
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</tbody>
</table>

Accompanying children are free.  
**Contact:** Dr. Pramod Sharma, Organising Secretary, PCG Complex, Mahamandir, Jodhpur, Ph: 2547387 ©, 2555387 ®, 3547387 (M), Email: drpramodsh@hotmail.com, rajpedicon2004@yahoo.com
PEDIATRIC SURGERY

MANAGEMENT OF MULTIPLE INJURIES IN CHILDREN

* Chowdhary SK
* Prema Menon
** Rao KLN

Abstract: Children sustaining traumatic multiple injuries are increasingly being seen in most hospitals. An efficient and knowledgeable trauma team with adequate facilities for primary resuscitation and secondary care should be available in all centers dealing with this problem. In every child, the priority of management should always be airway, breathing and circulation in that order. Operative intervention, whenever indicated, should be carried out only after all the systems have been thoroughly examined and assessed by the concerned specialists. Awareness of special physiological attributes of a child also helps us in understanding and managing a trauma victim better.

Key words: Trauma, Childhood injuries

In the United States, traumatic injuries remain the leading cause of emergency room presentation in children, second only to infections. The epidemiological data on trauma in children is available from the National Pediatric Trauma Registry (NPTR)\(^1\). In our country also, trauma is a common problem although no such national data is available.

Since most preventable deaths occur in the first hour after injury, trauma care in the developed world has seen the evolution of an efficient ambulance service with well-trained paramedical staff and development of regional pediatric trauma care centers with identified trauma teams. The protocol for primary management has been standardized by the American College of Surgeons into a simple, reproducible, easily taught module called the Advanced Trauma Life Support Course (ATLS), which is being followed worldwide\(^2\).

The concept of “golden hour” is probably far more relevant in the context of a child. Two large catastrophes in recent times, the September 11, 2001 attack on the World Trade Centre towers and the Gujarat earthquake prove the point. In both these tragedies, hardly any child with multiple injuries survived due to their limited reserve as compared to adults. Several pediatric trauma-scoring systems have been devised mainly to decide who should be transported to the regional centre with the highest level of facility. A simple pediatric trauma score (PTS) is given (Table 1)\(^3\) with scores ranging from a maximum of +12 (no or minor injury) to -6 which is fatal. A PTS of 8 is taken as a critical triage point.

Awareness of the special physiological attributes of a child helps us in managing a trauma victim better. A proportionately larger tongue characterizes the airway, the epiglottis is less stiff and may block the glottis, the larynx is situated cephalad and anteriorly with a narrow cricoid and the trachea is short in length. The normal blood
volume in a child is approximately 80ml/Kg or 7-8% of total body weight. The babies are able to maintain a relatively normal blood pressure with compensatory tachycardia till almost 40% blood loss, followed by rapid severe hypotension. This occurs as a result of compensatory constriction of medium and small arteries increasing the peripheral resistance till a very late hypovolemic stage. Because of the large body surface to mass ratio, they are at a greater risk of dehydration from burns and hypothermia. The head of a child is large relative to the body, making the centre of gravity higher. This leads to a higher risk of acceleration or deceleration injury resulting in higher incidence of cerebral concussion. The skeletal framework is more pliable and elastic leading to higher incidence of soft tissue injury without associated fracture.

The management of a child with multiple injuries must take place in a centre with certain basic minimum infrastructure, if the best possible outcome has to be achieved. In the developed world, the regional trauma centers are designated based on regional and national level planning. In our country, similar purpose is served by the tertiary level institutions. A child with multiple injuries needs a team of physicians and surgeons to work together under a team leader to bring out the best possible outcome. The basic infrastructure includes round the clock well equipped emergency department, blood bank, intensive care with ventilatory facility, radiology services including X ray, ultrasound and CT scan, operating rooms and availability of all trauma team personnel within half an hour of an accident. Adequate facility should be available for rapid communication. The pediatric surgeon holds the primary responsibility for a child with multiple injuries in our institute unless the abdomen or chest is not involved. In isolated specific injuries like burns, neurotrauma and limb trauma, the concerned specialities take over this responsibility. This is an area, which should be clearly discussed and guidelines made available in all centre taking on children with multiple injuries to avoid confusion in the face of an emergency.

Primary assessment and management

A member of the trauma team does the initial assessment of an injured child after arrival into a hospital. The priorities of management include Airway, Breathing, Circulation, Disability and Exposure (ABCDE) based on the well established guidelines of resuscitation. Decompression of closed visceral cavities like a tension pneumothorax may be a part of this procedure. Management of pain is not well emphasized in the standard guidelines on trauma but is an important aspect of practical management of a child with multiple injuries.

Table 1. Pediatric trauma score

<table>
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<th>Component</th>
<th>+2</th>
<th>+1</th>
<th>-1</th>
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<tbody>
<tr>
<td>Size (weight,age)</td>
<td>&gt;20kg</td>
<td>10-20 kg</td>
<td>&lt;10 kg Unmaintainable</td>
</tr>
<tr>
<td>Airway status</td>
<td>Normal</td>
<td>Maintainable</td>
<td>Comatose</td>
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<tr>
<td>CNS status</td>
<td>Awake</td>
<td>Obtunded</td>
<td>&lt;50 Major/Penetrating</td>
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<td>Systolic blood pressure</td>
<td>&gt;90</td>
<td>50-90</td>
<td>Open or multiple fractures</td>
</tr>
<tr>
<td>Open wounds</td>
<td>None</td>
<td>Minor</td>
<td></td>
</tr>
<tr>
<td>Fractures</td>
<td>None</td>
<td>Single closed fracture</td>
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The management of airway is the first in order of priority. Every child should be assumed to have cervical spine injury unless proven otherwise and the airway cleared of vomitus, blood etc. without hyperextension or hyperflexion of the neck. A laryngoscopic examination should be done to ensure that the airway is clear. In a violent child this may be difficult and help must be sought from other members of the trauma team. The jaw thrust manoeuvre to keep the airway patent involves placing two fingers behind the angle of each mandible and lifting anteriorly. In an unconscious child, an oropharyngeal airway or endotracheal (ET) intubation may be required. ET intubation is difficult in a violent child and carries the risk of aggravating cervical spine injury. Therefore, preoxygenation with bag and mask, atropine (0.1mg/Kg intravenous) and midazolam (0.1mg/Kg, intravenous, not to exceed 5mg) can be safely administered. If this does not help, succinylcholine or thiopentone sodium may be used. ET tubes of different sizes should be available in the emergency. Above 1 year, the size of the tube can be calculated by the formula: Age (in years) + 18.0/4.0 or Height (cm) / 20. However in the emergency, the tip of the little finger of the patient or the external diameter of the external nares can be taken as the appropriate size of an uncuffed ET tube. The tube should not be pushed in too far and the position must be checked by bilateral auscultation in the axilla before ensuring adequate fixation. A chest X-ray after intubation provides further proof of proper tube placement.

While the assessment of the airway and breathing is going on, the nurse should start oxygen support. This is usually done by facemask, unless the child is intubated. During the same period, the patient gets connected to electrocardiograph (ECG), oxygen saturation, temperature and non-invasive blood pressure (NIBP) monitors. If easy vascular access is possible, a nurse or doctor assisting resuscitation simultaneously inserts a cannula, collecting samples for blood investigation and cross-match. A request is also sent for essential X-rays. Regardless of the injury, any child who has been involved in trauma of a serious nature should have at least three X-rays (skull, cervical spine and chest) done straight away. Before shifting the injured child, the neck should be securely stabilised either on a splint or between two sandbags.

Evaluation of breathing and appropriate intervention is the next priority. Thoracic trauma is infrequent in children but carries a high morbidity. The thoracic cage is less protective in a child and there can be serious pulmonary contusion even without rib fracture. Tension pneumothorax and massive hemothorax should be diagnosed on clinical examination in the presence of respiratory distress with diminished breath sounds and hyperresonant or dull note on percussion and time should not be wasted waiting for a chest X-ray. Prompt aspiration of the chest in the fourth intercostal space in the midclavicular line confirms the diagnosis. There should be no delay in introducing intercostal tube drainage if either of these conditions is diagnosed. Pericardial tamponade is rare and is best treated by aspiration under imaging control only by a person with appropriate experience. Despite drainage of the thoracic cavity, a child with severe flail chest or associated pulmonary contusion may need ventilatory support and is best shifted to an intensive care unit.

Vascular access is the next priority. This can sometimes be very difficult in hypovolemia. Circulatory insufficiency may manifest with weak, thready, rapid pulse, lethargy and confusion, cool clammy skin and decreased urinary output. Delay in capillary refill of more than 2 sec is a very good indicator of circulatory insufficiency. Quite often, because of the better
elasticity of the child’s medium and small sized vessels, compensatory vasoconstriction can maintain the cardiac output till a very late stage followed by a precipitous fall of blood pressure. Therefore, the assessment of circulatory status must be based on a combination of symptoms and signs and proactive measures should be taken. Vascular access is usually obtained by cannulating a large vein like saphenous or basilic/cephalic vein. If access is not available easily, a venous cut down at the ankle is the safest. External jugular cannulation is safe, if the mode of trauma clearly excludes the risk of cervical injury. It is rare that venous access cannot be obtained by any of these measures in practice. At our institute, basilic vein cutdown in traumatized adults was in practice for years and has remained the standard mode of access for a traumatized child too. This is the cheapest form of central venous access and allows us to monitor the jugular venous pressure. It does not carry the risk of any large vessel injury in the neck/chest although there is a small but definite risk of brachial artery injury. If vascular access still remains an issue, insertion of a central venous line into the subclavian/ internal jugular vein or even intraosseous infusion are the other options. An intraosseous infusion is administered through a bone marrow needle (or a 16/18 G hypodermic needle) inserted into the anterior surface of the tibia 2 cms below the tibial tuberosity. Isotonic fluids and drugs can be administered through this route.

The next aim is to resuscitate the child for any circulatory insufficiency. This requires rapid infusion over a few minutes of fluid boluses of 20 ml/Kg. Our fluid of choice for initial resuscitation is normal saline which is a better isotonic fluid (308 mosm/L) compared to Ringer lactate (RL) (273 mosm/L). Moreover, normal saline is better than potassium containing RL till urine output is established. While this infusion is going on, the child is always catheterized. Urine output monitoring has remained the simplest and quite reliable index of the outcome of resuscitation. It is not uncommon for a child with multiple injuries to require two to three such boluses. Depending on the exact nature of injury, specific fluid therapy is subsequently planned. In a child who is hypovolemic due to blood loss, blood transfusion is administered as soon as available. In case of non-availability of appropriate group blood, O negative blood is acceptable as a life saving transfusion.

While the resuscitation for circulatory insufficiency is going on, a quick neurological assessment is made. The level of consciousness is assessed based on an easy mnemonic AVPU: ‘A’ alert and oriented, ‘V’ responds to verbal stimuli, ‘P’ responds to painful stimuli, ‘U’ unresponsive. Even if the child has a head injury, the initial focus must remain on resuscitation, as the eventual outcome can be significantly improved with well-perfused and oxygenated blood 6. This completes the ABCDE of the primary assessment and management of a child with multiple injuries.

Secondary survey and management

Once the initial resuscitative steps are in order, the child is now fully exposed with due care to the ambient temperature, so that a thorough assessment can be done of all the parts of the body from the top of the head to the feet and no injury missed out. While examining the child, a brief history is also elicited from the parents regarding the mode of trauma.

The examination of the head includes examination for laceration, hematoma, bleed from ear, nose or throat, cerebrospinal fluid leak and evaluation of both eyes including the pupils. Maxillofacial injury including mandibular fracture is rare in children, but can easily be missed. Neck examination includes any external signs of trauma, tenderness and radiological
abnormality seen on cervical spine x-ray. It is important to be aware that spinal cord injury is not always associated with visible abnormality of the cervical spine on X-ray. In the examination of the chest, due care must be taken to exclude fracture clavicle and ribs. The child should be log rolled, so that the entire back can be examined at once. Abdominal examination is best done after nasogastric decompression. Examination of the groin, genitals, perineum, and rectum should not be ignored. Any obvious sign of injury in the form of blood discharge from any orifice should be specifically recorded. Any tenderness of the pelvic bone should be elicited. All four limbs are examined with respect to laceration, joint dislocations and fracture. The pulses should be specifically examined distal to the fractured limb. At this stage before completing the evaluation, a second detailed neurological evaluation is done to establish the status of the child based on the Glasgow Coma Scale (GCS).

A list of relevant investigations including ultrasound and CT scan in case of head injury is organized. All members of the trauma team who need to see the child are informed while the child is being assessed. At this stage, it is also ensured that necessary documentation and medico legal papers are completed. At our institute we use ultrasound for every child with abdominal trauma, CT scan being used only when some specific information is sought. A rapid abdominal ultrasound scan in an injured child is called a ‘Focussed Abdominal Sonogram Test’ (FAST). This policy is based on our own logistics. However, in most pediatric trauma centers, CT scan is used extensively. There are certain other investigations, which are specifically used in case of trauma, e.g.: retrograde urethrogram in case of any difficulty with catheterization or blood at the urethral meatus; angiography in case of suspected vascular injury etc. These are usually done only after all the specialist members of the trauma team have examined the child.

The aim of this stage of evaluation is to assess the entire effect of trauma on the child, evaluation by the respective specialities, organization of specific investigations and planning of intervention in the correct order. This stage usually takes a few hours in our hospital. In dedicated trauma units, this may be quicker. But as long as the child has recovered from the shock and is breathing well, undue haste is unnecessary. Appropriate attention must be paid to pain relief during this stage of trauma management while different specialists are assessing the patient.

Principles of management of specific injuries in a child

Head Injury: Head injury is one of the commonest problems in a child with multiple injuries and is associated with falls from height, deceleration injuries, and bicycle injuries. Majority of deaths after multiple injuries is related to head injury. It is important to evaluate a child in detail in the context of GCS, which evaluates the best motor, verbal and eye response. The most direct correlation in the long term has been with the best motor response. Children with total scores less than 8 are at the greatest risk of developing life threatening intracranial hypertension and should be electively ventilated. Pulse rate, pupil size and reaction to light, and GCS should be monitored continuously.

The presence of asymmetric clinical signs like pupillary constriction, dilatation or neurological signs of lateralisation increases the possibility of intracranial mass lesion. Every child with altered sensorium or any of the above signs should have a CT scan of the head at the earliest. In general, presence of a mass lesion and depressed fracture of the skull are indications for surgery.

Majority of the children are found to have diffuse cerebral edema caused by the acute
trauma and secondary hypoxia, hypotension, hypercarbia, raised intracranial tension and seizures. Apart from oxygen therapy, anti-epileptic medication and supportive care of an unconscious patient, treatment of raised intracranial pressure includes controlled ventilation maintaining the pCO2 between 25-30mmHg.

**Spinal injury:** The cervical spine in a child is prone to deceleration injuries due to increased flexibility of supporting ligaments, joint capsules and horizontal facet joints that are prone to displacement. Older children are more prone to lower cervical and thoracolumbar spinal injury, while the younger ones sustain injury between the occiput and third cervical segment. The lateral, anteroposterior and odontoid radiographic views are used to examine cervical spine injury. Spinal cord injury without radiological abnormality (SCIWORA) may present with motor and sensory symptoms without a radiological abnormality on conventional radiology. The long-term outcome in this kind of spinal cord injury is dependent on the neurological status on admission.

The treatment for majority of the spinal injury remains supportive. It represents high velocity trauma and is often associated with hollow viscus perforation. Rarely, if the spinal segment is displaced and unstable it may require operative fixation. In our center, craniospinal trauma in a child is managed by the neurosurgeons. However in many other centers, unless specific surgical intervention is required, pediatric surgeons manage this injury.

**Thoracic trauma:** Pulmonary contusion is the commonest form of thoracic trauma in children. As a result of direct trauma to chest and chest wall compression, the lung parenchyma undergoes disruption and hemorrhage with edema. Plain x-ray of the chest misses or underestimates pulmonary contusion in 40% cases and it is better detected on a CT of the chest. Rib fractures are uncommon. However in severe injury of the chest, it may be present including flail chest. The fracture of first and second ribs may be associated with great vessel injury. Upper limb pulses must be examined in such a child. This may even present later with chronic ischemic changes of the upper limb.

Pneumothorax is not uncommon and requires urgent attention. Intercostal tube drainage is the immediate treatment and in most cases, a few days of drainage is enough. Rarely, if the pneumothorax persists, bronchial injury must be suspected. This is a definite indication for CT scan and bronchoscopy, followed by surgical intervention. Hemothorax is also common but is usually transient and self-limiting. However, the collected blood must be drained to improve breathing to prevent delayed fibrothorax and also to confirm that active hemorrhage has ceased.

**Abdominal trauma:** Abdominal trauma can be broadly classified into penetrating and blunt injuries. Majority of the penetrating trauma will require exploration. In blunt abdominal trauma, the abdomen should be examined for bruise, contusion, abrasion, fracture ribs/pelvis, abdominal distension, tenderness and guarding. This is supported by radiological evaluations like plain X-ray, ultrasonography and CT scan. In most cases unless there is a definite sign of hollow viscus perforation with features of peritonitis and pneumoperitoneum, the initial management is conservative. The abdomen should be evaluated at regular intervals for resolution of abdominal signs and return of bowel activity.

Most solid organ injuries (liver, spleen and kidney) can be managed conservatively with bed rest, blood transfusion and supportive care unless the patient is not hemodynamically stable despite
transfusion of 40ml/Kg. However, some rare injuries like duodenal injury, pancreatic injury, transection of bile duct and pelviureteric junction can have a delayed presentation. If the abdomen is not settling in terms of symptoms and signs, specific investigations have to be aimed towards these injuries like upper gastrointestinal contrast study, isotope scan for bile duct or ureteric injury, contrast enhanced CT/MRI for pancreatic injury etc.

**Genitourinary trauma:** Majority of renal injuries in children are secondary to blunt trauma. Evaluation of this injury is based on urine examination for hematuria, ultrasonography and subsequent isotope renography and/or intravenous pyelography in selected cases. Contrast CT scan is the best modality for accurate initial evaluation of renal parenchymal injury and staging, but must be used selectively. Management is based on ongoing hemorrhage, associated extrarenal injury, the condition of the contralateral kidney and the physiological condition of the child. Bed rest is curative in most instances. In any case, the renal injury alone is rarely an indication for emergency exploration. Delayed renal scans may pick up scars, which may be responsible for hypertension in later life.

Pelvic injury is frequently associated with fracture pelvis, bladder and urethral injury. This should always be anticipated and in case of any doubt a contrast study including retrograde urethrogram and stress cystogram should be done. Unless extensive experience is available in the definitive management of these conditions, the simplest operative intervention is a suprapubic urinary diversion. Definitive management of urethral injuries can be undertaken in a specialist unit.

**Musculoskeletal trauma:** Musculoskeletal trauma is one of the commonest components of a child with multiple injuries and includes fall from a height, bicycle injury, motor vehicle injury etc. The limb needs evaluation with respect to soft tissue, fracture of bones as well as associated vascular and nerve injury. Hemorrhage from an injured limb should be treated by compression dressings and limb elevation. Control of bleeding by using artery forceps should not be attempted in the emergency room due to the high risk of nerve damage. The priorities in the initial management are appropriate diagnosis, relief of pain and stabilization of the fracture. The limb requires a detailed examination after initial resuscitation to determine the need for operative intervention.

An absent pulse with signs of ischaemia is a definite indication for angiography and exploration.

**Summary**

The management of a child with multiple injuries requires an excellent ambulance system, which can rapidly evacuate children safely to a regional pediatric trauma center. The initial focus must be on adequate resuscitation by the team in the emergency room based on a standardized protocol. There are very few indications in pediatric trauma practice to rush to emergency surgery. Operative intervention should be carried out in a child whenever indicated, only after all the concerned specialists have evaluated the child and a plan of management has been developed. The pediatric surgeon usually coordinates this exercise. Such a team approach provides the best chance for recovery of a child with multiple injuries with a good long-term outcome.

**Points to remember**

1. **The priorities of primary management of a child with trauma include airway, breathing, circulation and disability (neurological assessment).**
2. A thorough secondary survey should be performed from head to toe for other injuries after initial resuscitation has been completed and the child stabilized.

3. A child should be taken to the operation theater only after all relevant investigations have been performed and all concerned specialties have completed their assessment.

References


NEWS AND NOTES

**PEDIATRIC EMERGENCY MEDICINE COURSE**

**Date:** June 5th and 6th 2004  
**Venue:** Kanchi Kamakoti CHILDS Trust Hospital  
The course fee is Rs.1000/- for Post Graduates (to produce student certificate from heads of institution) and Rs.1200/- for practitioners. Preference will be given to PALS certified personnel. For details contact the course coordinators  
Dr.Indumathy Santhanam  
A/3, Sreshta, 473,  
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12-A, Nageswara Road, Nungambakkam,  
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Email: jayramo2@rediffmail.com

**“MECHANICAL VENTILATION 2004 - BASICS AND BEYOND ”**

**Date:** July 17th & 18th 2004  
**Organized by:** KR Hospital, Bangalore

**Contact:** Dr. Girish H.C, Organizing Secretary – MV 2004, KR Hospital, 979, 25th Main Road, Hanumanth Nagar, 50 ft Road, BSK 1st Stage, Bangalore 560076, Phone: 26755800/ 402 / 901, Mobile: 9845272933, Email: mvkrh2004@hotmail.com
EVALUATION OF THE LIVER - LABORATORY TESTS

* Malathi Sathiyasekaran
** Shivbalan So

“Liver function tests are no more infallible than the people who use them” – A.M. SNELL

The liver performs a variety of important biochemical, synthetic and excretory functions. The term “liver function tests” (LFTs) is a misnomer as the majority of these tests assess liver cell damage rather than function. Hence it can be called as “laboratory investigations of the liver”. These tests are used in the evaluation and management of patients with hepatic dysfunction. They help in screening for the presence of liver disease, recognizing the type of disease, estimating severity, assessing prognosis and monitoring therapy.

These tests lack sensitivity and specificity. No single biochemical test is capable of providing an accurate global assessment of hepatic function. To increase both the sensitivity and specificity of these laboratory tests it is essential to use them as a “battery” rather than as a single test. When one or more of these tests are abnormal or persistently abnormal on serial testing, the possibility of detecting liver disease is high. When all the tests are normal, the probability of missing occult liver disease is low.

The tests are usually grouped into the following categories:

I. Tests that reflect injury to hepatocytes
II. Tests that indicate cholestasis
III. Tests that measure the capacity of the liver to transport organic anions
IV. Tests that measure hepatic synthetic functions
V. Tests that measure the capacity of the liver to metabolize drugs.
VI. Tests that detect chronic inflammation or altered immune response
VII. Markers of liver fibrosis and fibrolysis
VIII. Miscellaneous

Tests that reflect injury to hepatocytes

Aminotransferases

The aminotransferases namely the aspartate aminotransferase (AST) or SGOT and alanine aminotransferase (ALT) or SGPT are relatively good indicators of liver cell membrane damage. ALT is primarily from liver cytosol, whereas AST is distributed in a wide variety of tissues such as liver (cytosol and mitochondria), cardiac muscle, kidneys, brain, pancreas, lung, WBC and RBC. Several factors other than liver cell injury modify AST level. In practice therefore, the ALT is used as a marker for liver cell injury. The aminotransferases are probably the earliest enzyme to be elevated in acute hepatitis. In a clinical setting they are elevated even prior to...
the rise in serum bilirubin. There is no direct correlation between the transaminase level and the severity of liver disease. For example, in cirrhosis the transaminases may be normal. Marked elevation more than 20 fold is seen in circulatory shock, toxic and viral hepatitis. Moderate elevation (3 to 20 fold) is observed in chronic hepatitis, alcoholic hepatitis, acute biliary obstruction, hepatitis due to non-viral agents such as salmonella and leptospirosis. Mild elevation (2 to 3 fold) is seen in fatty liver, non-alcoholic steatohepatitis (NASH), myositis and hepatic neoplasms. The levels may be low in patients on long-term dialysis. Falling levels are seen in a patient recovering from acute hepatitis, but a rapid fall may indicate poor prognosis and suggest impending fulminant hepatic failure (FHF). Fluctuating levels are at times observed in chronic hepatitis type C and this phenomenon is called “yoyo” effect.

AST/ALT Ratio: The ratio of AST and ALT is significant in certain liver diseases. In ethanol injury it is more than 2:1, here the mitochondrial AST is elevated more than the cytosolic AST. There is also a reduction in hepatic ALT due to deficiency of cofactor pyridoxine-5-phosphate. The ratio may be more than 2 in Reye’s syndrome and drug induced hepatitis, however it is <1 in viral hepatitis.

The limitations of transaminases are that 6% of healthy asymptomatic people have abnormal liver enzymes. Transaminases may increase due to non-hepatic causes (myocardial infarction, myositis). It may not help in identifying the aetiology of the liver disease or correlate to the severity of liver injury. It cannot also predict outcome. The transaminases are used in screening for liver cell injury and monitoring therapy in chronic hepatitis.

Lactate Dehydrogenase: This cytoplasmic enzyme has wide distribution and elevations are seen in acute and chronic liver disease apart from skeletal and myocardial injury, haemolysis and renal infarction. In ischaemic hepatitis it is high but transient. A persistent elevation with high serum alkaline phosphatase (SAP) indicates malignant infiltration. The ALT to LDH ratio of > 1.5 helps to differentiate between acute viral hepatitis and ischaemic hepatitis. This ratio is also used to differentiate viral from typhoid hepatitis, which is >4 in the former and <4 in the latter.

Tests that indicate cholestasis

Serum Alkaline Phosphatase (SAP): This family of immunologically distinct isoenzymes is distributed in abundance in placenta, ileal mucosa, kidney, bone and liver. In the liver it is present in the canalicular membrane and bile duct epithelium. Cholestasis stimulates synthesis and bile salts facilitate release from cell membranes.

An increased level of alkaline phosphatase is seen in childhood, adolescence (due to rapid bone growth), pregnancy, extra hepatic and intra hepatic cholestatic disorders, biliary cirrhosis, abscess and infiltrative liver disease. Since the half-life of alkaline phosphatase is seven days, the levels in obstructive jaundice may be increased for almost a week after relief of obstruction. Low levels of SAP are seen in Wilson’s disease with FHF and hemolysis, congenital hypophosphatasia, hypothyroidism, pernicious anemia, zinc deficiency and malnutrition. The limitations of increased SAP are that it does not differentiate between hepatic and non-hepatic sources, extra and intra hepatic obstruction.

Gamma Glutamyl Transpeptidase (GGTP): This enzyme is located in the entire hepatobiliary tree and levels parallel SAP estimation. Within the hepatocyte it is a microsomal enzyme and induced by alcohol and drugs. The values vary with age and sex. SAP is high but GGTP is
normal or low in metabolic liver diseases such as inborn error of bile acid metabolism and progressive familial intrahepatic cholestasis (PFIC 1 and 2)

5’ Nucleotidase: This enzyme is found in the liver in association with canalicular and sinusoidal plasma membranes. The increase in 5’ nucleotidase parallels SAP and elevation of both confirms cholestasis. It helps to differentiate increased SAP seen in normal children and pregnant women from those with liver disease and also for screening in hepatic metastasis.

**Tests that measure capacity to transport organic anions.**

Serum Bilirubin: The estimation of total bilirubin and its fractions is a reflection of the hepatocyte’s capacity for the uptake, conjugation and excretion of bilirubin. In normal individuals the serum bilirubin is mostly unconjugated (0.1 to 1mg/dl). The method used in analysis may at times overestimate the conjugated fraction but normally the level of conjugated should be only 4-5% of total bilirubin. In hemolytic jaundice, congestive cardiac failure and Gilbert’s syndrome the jaundice is predominantly unconjugated. Conjugated hyperbilirubinemia is seen in biliary obstruction. In congenital hyperbilirubinemia such as Dubin Johnson syndrome and Rotor syndrome the direct bilirubin is increased without rise in SAP.

Urine bilirubin: Only direct reacting conjugated bilirubin is detected in the urine. The test is useful in early detection of acute viral hepatitis. The absence of bile pigments in a patient with jaundice could indicate recovery, unconjugated hyperbilirubinemia or anicteric hepatitis.

Urine urobilinogen: The levels are increased in hemolysis and slightly elevated in hepatocellular dysfunction. The urobilinogen is usually absent in extra hepatic bile duct obstruction.

**Serum Bile acids:** Serum bile acids are the most sensitive indicator of cholestasis. However this test is not readily available in India. They are useful in diagnosing inherited disorders of bile acid metabolism.

**Serum Ammonia:** The liver primarily regulates the concentration of blood ammonia. It is cleared mainly by urea synthesis. The serum ammonia is elevated in hepatic encephalopathy and is useful in monitoring these patients. The levels increase before the onset of coma and return to normal 48 to 72 hours before neurological improvement. The measurement of arterial ammonia is more reliable than venous. High concentrations are seen in urea cycle disorders and Reye syndrome.

**Tests that measure hepatic synthetic function.**

The liver synthesizes several proteins including albumin, coagulation factors and lipoprotein.

**Serum Albumin:** This is an important plasma protein and is synthesized exclusively by the liver. Normal serum values range from 3.5 to 4.5gms/dl. Albumin has a long half-life of 21 days. The level depends on rate of synthesis, degradation and volume of distribution. Decreased albumin is also seen in protein energy malnutrition (PEM), protein losing enteropathy, chronic infections and nephrotic syndrome. In liver disease decreased albumin < 3gms with elevated gammaglobulin should raise the suspicion of chronic liver disease.

**Prothrombin Time (PT):** This is a useful test to measure the synthesis of some of the coagulation factors I, II, V, VII and X. The normal value is 9-11secs. It is abnormal if prolonged 2secs more than control. If the prothrombin time returns to normal or improves by at least 30% within 24 hours after a single dose of vitamin K it may be surmised that the liver parenchymal function is good.
International Normal Ratio (INR) has been introduced to standardize the reporting of prothrombin ratio obtained with various thromboplastin reagents. In patients with liver disease PT rather than INR is used, however this does not standardize results between laboratories. As these proteins have a shorter half-line than serum albumin, PT may be used as an early indicator of injury. Prolonged PT is an ominous prognostic sign in both acute and chronic hepatobiliary injury.

**Activated Partial Thromboplastin Time (APTT):** An abnormal APTT indicates deficiency of factors VIII or IX, but does not replace PT in the evaluation liver disease.

**Lipoprotein:** Liver is the major source of plasma lipoprotein except chylomicrons, which is synthesized in intestine. In cholestasis, serum cholesterol, phospholipids and lipoproteinX are increased.

**Tests that measure the capacity of the liver to metabolize drugs.**

**Lidocaine Metabolite Formation:** Lidocaine is metabolized to monoethylglycine xylidide (MEGX) within the hepatic cytochrome P450 system. Lidocaine is given intravenously (1mg/kg) and 15 mts later the serum is tested for MEGX. Cirrhotics with higher MEGX fare better than those with lower MEGX levels indicating functioning liver cells. This estimation has better prognostic value than CHILD-PUGH score. It has been used in identifying suitable donors for liver transplantation.

**Tests that detect chronic inflammation or altered immune response**

Serum immunoglobulins are produced by stimulated β lymphocytes and thus do not test liver function directly. Their elevation in many patients with chronic liver disease is believed to be due to impaired function of reticulo-endothelial cells in hepatic sinusoids or due to shunting of portal venous blood from the liver. In chronic liver disease there is a diffuse polyclonal increase in immunoglobulin. Hypergammaglobulininemia with or without hypoalbuminemia is seen in chronic liver disease and autoimmune hepatitis. In primary biliary cirrhosis IgM is increased and IgG is elevated in autoimmune hepatitis.

**Markers of liver fibrosis and fibrolysis**

Liver biopsy being an invasive procedure has its own complications, however it may be necessary to evaluate the extent and type of liver injury (hepatocellular necrosis or fibrosis). Certain blood tests have now been introduced which reflects the degree of hepatic fibrosis and may replace liver biopsy in the near future. They are

- **P III NP:** Immunoreactive aminoterminal propeptide of procollagen III. This is a marker of fibrosis especially useful in chronic progressive liver disease. Decreasing PIII NP levels have shown to correlate with response to therapy.

- **Collagen VI:** This is a marker of fibrolysis particularly useful for the detection of hepatic collagen turn over in children as its levels are independent of body growth.

- **Collagen XIV:** This is also known as undulin and increased 3 to 5 fold in early stages of alcoholic liver disease, schistomiasis and PBC when markers such as PIII NP are within the normal range.

Other markers that have been studied are hyaluronan, β 1 integrins, α 2 and α 5 integrins.

**Miscellaneous tests:**

The laboratory tests discussed above suggest but seldom make an aetiologial diagnosis. Serological markers for viral hepatitis,
autoantibodies, serum ceruloplasmin, serum ferritin, alfa-l-antitrypsin, alfa-fetoprotein, ultrasonography, liver biopsy, cholangiography and computerized tomography along with the selective laboratory tests are useful in making a more specific diagnosis.

Thus laboratory tests of the liver have a definite role in the evaluation of a liver disease and the choice of the test will depend on the nature of the liver disease and the indication for the investigation.

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ANNOUNCEMENT

The Respiratory Chapter of IAP has prepared an interactive module on Asthma Training. This consists of a set of slides, based on the IAP National Guidelines on Asthma Management, contained in the book ‘Asthma By Consensus’. This module has been successfully conducted in six centers of the country. Any branch / individual member of IAP interested in organizing ‘Asthma Training’ workshop in their city may contact office bearers of IAP Respiratory Chapter through post, phone, or email. The chapter will assign two master trainers from that region to conduct the 4 hrs workshop. Interested organizer(s) would have to arrange a sponsor for the workshop. Contact persons are - Raju Khubchandani – 23865522 / 98210 95552 / dr_rajukay@hotmail.com; Sailesh Gupta – 28089744 / 98910 42213 / gupta_sailesh@hotmail.com; Ajit Gajendragadkar – 98200 32947 / suprajit@vsnl.com.

CONTRIBUTORS TO CORPUS FUND OF IJPP

Rs.1000/-
1. Dr.K.Ravi Raj Rao, Byndoor, Karnataka.
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PEDICON 2005: KOLKATA

Theme: Towards Vision 2020: Our Mission - Total Health Care from 0 to 18
(Dedication - APJ Abdul Kalam, President of India)

Dates:
1. Preconference workshops : 5 January 2005
2. Preconference CMEs : 6 January 2005
   Three CMEs:
   i. Practical Pediatrics
   ii. Advanced Pediatrics
   iii. Pediatric Dermatology
3. Inauguration : 6 January 2005, 6 pm
4. Conference : 7, 8, 9 January 2005
5. Valedictory : 9 January 2005

Venues:
Main venue : Science City, Kolkata
2nd venue : ITC Sonar Bangla Sheraton and Towers (7 star hotel)

Registration:

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* Above 5 years of age, everybody should register
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ASPHYXIATING THORACIC DYSTROPHY

* Seenivasan V  
** Venkataraman P  
** Ravisekar C.V  
*** Vasanthamallika T.K  
**** Ramadevi P

Asphyxiating thoracic dystrophy is a rare autosomal recessive chondrodysplasia that often leads to death in infancy because of severely constricted thoracic cage and respiratory insufficiency. The diagnosis is mainly based on radiological features. We present an infant who developed progressive asphyxia with typical radiological features.

Case report

A 50 days old male child, third born to second degree consanguineous marriage has been repeatedly admitted in our hospital for difficulty in breathing which was persistent from birth. The first born of the parents was a stillborn female baby. The second born female child died at the age of six months because of an unexplained respiratory problem.

On clinical examination, there was obvious thoracic deformity with a narrow thorax and short limbs without any polydactyly. The child had respiratory distress and was not cyanosed. The weight of the child was 3.1 kg. The ratio of upper segment to lower segment was 1.9. The chest circumference at the nipple level was 25 cm, 10 cm less than the head circumference. Auscultation of the chest revealed basal rales bilaterally.

The chest x-ray showed a normal cardiac silhouette. Thorax was narrow with short horizontal ribs and the clavicles were highly placed. The anterior ends of the ribs were enlarged and irregular with this clinical picture and chest x-ray findings, the diagnosis of asphyxiating thoracic dystrophy was made which was confirmed by features of pelvic x-ray. The x-ray pelvis showed small pelvis with hypoplasia of iliac wings, more horizontal acetabular roof and spur like projections from the lower margin of sciatic notches. Echocardiogram revealed a small closing ventricular septal defect. Ultrasonogram of the abdomen revealed no visceral abnormality. Liver and renal function tests were within normal limits. Fundus examination showed normal retina.

The treatment was mainly supportive.

Discussion

Asphyxiating thoracic dystrophy (ATD) or Juene syndrome is a multisystem autosomal recessive disorder associated with a characteristic skeletal dysplasia and variable renal, hepatic, pancreatic and retinal abnormalities. The reported incidence is 1/100000 – 1/130000 worldwide.

The growth is usually stunted with various skeletal abnormalities. Chest x-ray usually
reveals short horizontal ribs with bulbous, irregular anterior ends and highly placed clavicles giving the appearance of handle bars. Pelvis is usually small with hypoplastic iliac wings. The roof of acetabulum is more horizontal and a spur like projections are seen from the lower margins of sciatic notches. Early ossification of capital femoral epiphysis is a consistent feature. Radiological features in limbs, hands and feet are gradually discernable beyond the period of infancy. The long bones have irregular metaphysis and epiphysis. Ulna and fibula may be relatively shorter than other long bones. Cone shaped epiphysis are seen in hands and feet. Because of early fusion of metaphysis and epiphysis, phalanges are usually short. Polydactyly is an inconstant feature of ATD and when present, usually affects both the hands and feet.

The predominant visceral involvement is lung hypoplasia secondary to small thoracic cage. Cystic tubular dysplasia and glomerular sclerosis, fibrotic and cystic changes involving liver and pancreas and peripheral retinopathy are other visceral abnormalities with variable incidence.

Medical therapy is mainly supportive in the form of controlling recurrent pulmonary infections and taking care of the feeding difficulties. Ursodeoxycholic acid appears to control the progression of hepatic dysfunction. Genetic counseling should be given to the parents of child with ATD, as there is 25% risk of recurrence in the offspring.

The progressive asphyxia, recurrent pulmonary infections and failure to thrive are indications for surgical intervention. The idea of the surgery is to increase the thoracic volume. Sternotomy and lateral thoracic expansion are the surgical techniques currently practiced.

70% of infants die because of asphyxia with or without pneumonia. Renal insufficiency may appear by 2 years of age. Hepatic dysfunction may complicate the course. Because visceral involvement is variable, survival had occurred up to the 4th decade.

Though ATD locus has been mapped to the long arm of fifteenth chromosome (15q 13), the defective gene is still not known. Antenatal diagnosis is not currently possible by genetic methods.
ATD is a rare skeletal dysplasia and without intervention mostly lethal in infancy. Aggressive medical therapy during infancy and appropriate thoracic expansion procedure at later stages can bring down the mortality considerably. Substantial longevity can be expected, if visceral involvement is minimal.

References:


CONGRATULATIONS

Dr.S.Sushambai, had been awarded Dr.P.A.Alexander Oration Award 2003 by the IMA AMS Kerala Chapter in recognition of her exemplary contribution to the field of modern medicine for the year 2003.

The Journal of Postgraduate Medicine, the official publication of the Staff Society of Seth GS Medical College and King Edward Memorial Hospital was started in the year 1955. In 2004, the journal has the distinction of completing 50 years of publication. A Golden Jubilee Conference (JPGM GOLDCON) is being organised on the 23rd, 24th, 25th and 26th September 2004 in Mumbai to commemorate this event.
**QUESTIONS AND ANSWERS**

**Q. No. 1:** Following vaccination, which one is preferable - Hot water sponging or ice cube sponging? Why?

**Dr.K.Thinakaran**  
Attur, Tamilnadu.

**A. No. 1:** Available references on vaccine administration do not recommend either hot water or ice fomentation. Just a gentle pressure for a few seconds will suffice for vaccines administered by intramuscular route (Aluminium salt adjuvanted vaccines).

**Dr. A.Parthasarathy,**  
Retd. Sr. Clinical Professor of Pediatrics,  
Madras Medical College, Chennai.

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**Q. No. 2:** Do all hepatitis B vaccinated children who get acute viral hepatitis subsequently need evaluation of Australia antigen routinely? What is the transition period when the efficacy of hepatitis B Vaccine starts waning? In other words what is the exact time to give hepatitis B booster?

**Dr. Uma Kathiresan**  
Tenkasi, Tamilnadu.

**A. No. 2:** The WHO position paper 2001 categorically reiterates the following facts regarding hepatitis B routine infant immunization:

1. If the infant responds to three doses of HB vaccine, provided a minimum of 4 weeks is maintained between the second and third doses, no booster dose is required. (2) Even if virus exposure occurs in an immunized child, due to an anamnestic response the antibody titre automatically goes up above the protective level of 10mIU/L, which acts as a booster.

2. (3) IAP currently recommends hepatitis B vaccines:-

   a. At birth, 6 weeks and 14 weeks for those babies born to HBs Ag positive mother along with HBIG within 24 hrs after birth. If HBIG is not available a 4th dose may be given at 9-12 months.

   b. At 6,10,14 weeks for those babies born to HBs Ag negative mothers and

   c. At 0,1,6 months (‘0’ being elected date for older children, adolescents and when combined Hep A – Hep vaccine formulation is used.)

**Dr. A.Parthasarathy,**  
Retd. Sr. Clinical Professor of Pediatrics,  
Madras Medical College, Chennai.

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**Q. No. 4:** How to treat a four months old infant with pinworm infestation?

**Dr.K.Thinakaran**  
Attur, Tamilnadu.

**A. No. 4:** Enterobius vermicularis the human pinworm is very difficult to eradicate. The adult worm lives in the rectum. At night hours it migrates to the anus and in the perianal region it lays eggs and dies. Infection can occur mostly by scratching the perianal region; the eggs are transmitted through the nail into the mouth. Swallowed eggs release larvae in the small intestine and the larvae migrate to the large intestine. The retrograde infection can return to
the rectum. The eggs are also light and are known to be transmitted through air. It can also migrate to the vagina and can cause itching. The diagnosis can be easily made by inspecting the perianal region by observing the live thread worms. The time taken for swallowing the egg and development to the adult worm is roughly 14 days. Treatment consists of a single administration of pyrantel pamoate 11mg/kg or a single dose of mebendazole 100 mg irrespective of age. But what is important is the treatment to be repeated after 2 weeks to kill the worms that may have hatched from eggs swallowed during the initial treatment.

Dr. C. V. Ravisekar  
Asst. Prof. of Pediatrics  
ICH & HC., Chennai.

Q.No.3: As per ARI programme, there is no role of Dexamethasone and Deriphyllin in LRI – (Bronchiolitis / Br Pneumonia – WA LRI – but to my surprise many practitioners still use it, as an anti inflammatory and bronchodilator in rural areas, when O2 and nebulisation, SaO2 are not available. What is the harm these two drugs can cause?

Dr. C. M. Chhajer,  
Agartala, Tirupura

A.No.3: As the pneumonia in children below five years old in India is largely caused by bacterial pathogen(s), an early assessment of the severity and administration of appropriate antibiotics remain the key factors to reduce the morbidity and the mortality from the disease. Several other pathogens such as chlamydia trachomatis, ureaplasma, mycoplasma pneumoniae, RSV, influenza and adenovirus also cause respiratory tract infection in 20 to 30% children. Infants and young children who have non bacterial pneumonia can also present with cough accompanied with an evidence of airway narrowing such as prolonged expiration and wheeze on physical examination.

Viral bronchiolitis is the one of the commonest causes of lower respiratory tract illness in young infants and the condition is usually self limiting with support needed for infants born prematurely, those with pre-existing cardiac or respiratory disease. On current evidence, inhaled and systemic corticosteroids, and bronchodilators do not have a role in the routine management of infants with bronchiolitis. Administration of these agents in such illnesses is unlikely to show an improvement in oxygenation or in the duration of hospital stay. However, the use of inhaled beta-2 agonists may be useful in a selected/ severely affected children. Additionally the use of deriphylline/dexamethsone instead of oxygen administration in a child with severe pneumonia is irrational and can worsen the child’s hypoxic condition contributing to further morbidity.

Reference


Dr. Niranjan Shendurnikar  
Associate Professor of Pediatrics  
Medical College Baroda 390001.
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The Respiratory Chapter of IAP has prepared an interactive module on Asthma Training. This consists of a set of slides, based on the IAP National Guidelines on Asthma Management, contained in the book ‘Asthma By Consensus’. This module has been successfully conducted in six centres of the country. Any branch / individual member of IAP interested in organizing ‘Asthma Training’ workshop in their city may contact office bearers of IAP Respiratory Chapter through post, phone, or email. The chapter will assign two master trainers from that region to conduct the 4 hrs workshop. Interested organiser(s) would have to arrange a sponsor for the workshop.

Contact persons are: Raju Khubchandani - 23865522 / 9821095552 / dr_rajukay@hotmail.com;
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