Nutrition

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- Infant and Young Child Feeding
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- Breastfeeding
  JP Dadhich
- Malnutrition
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- Water-soluble Vitamins
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- Trace Elements
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- Human Milk Banking
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4.1 Infant and Young Child Feeding

We know that shamefully large numbers of children in both wealthy and poverty-stricken regions suffer malnutrition: the malnutrition of excess amounts of inappropriate foods and the malnutrition of insufficient nutritious foods. As adults, we should feel embarrassed that so many small children are so poorly fed. We can change this situation, if we want to and many people are already working to this end. Good nutrition for children harmonizes with good nutrition for adults...and it would be good for the world if we worked to this end.

—Gabrielle Palmer

INTRODUCTION

While adequate nutrition is important throughout childhood, it is crucial during the first 5 years of a child’s life particularly so, in the first 2–3 years when rapid growth occurs and when the child is entirely dependent on the mother and the family for food.

OPTIMAL INFANT AND YOUNG CHILD FEEDING

Optimal infant and young child feeding (IYCF) is an evidence-based measure for improving child nutrition and child survival. The “World Health Organization (WHO)/United Nations Children’s Fund (UNICEF) Global Strategy for Infant and Young Child Feeding and the National Guidelines on Infant and Young Child Feeding 2010” recommended by the IYCF subspecialty chapter of the Indian Academy of Pediatrics (IAP) stress that for proper growth and development, infants should be exclusively breastfed with no other food or drink—not even water in the first 6 months of life (see Chapter 4.2). This must be followed by sequential addition of nutritionally adequate, preferably home-made semisolid and solid foods to complement (not to replace) breast milk, till the child is gradually able to eat normal family food after 1 year while breastfeeding is continued up to 24 months of age or beyond (Fig. 4.1.1). Adequate nutrition for adolescent girls and pregnant and lactating mothers is also important for child nutrition.

The period after 6 months, when other foods are added is also referred to as weaning. Some wrongly interpret it as weaning the baby away from the breast. Complementary feeding is a better term than weaning.

COMPLEMENTARY FEEDING

It is the process of giving a child other food while continuing breastfeeding, when her or his nutritional demands can no longer be fulfilled by breastfeeding alone. Appropriate complementary feeding should be timely, culturally acceptable, nutritionally adequate, safe and responsive.

Timely Feeding

It is recommended that all infants must be exclusively breastfed for 6 months and adequate complementary foods should be added after that. Complementary feeding indicators

<table>
<thead>
<tr>
<th>Global consensus on optimal infant feeding</th>
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<tbody>
<tr>
<td>Age 24 months</td>
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<tr>
<td>In the second year, more and more family foods are given, yet breastfeeding continues</td>
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<tr>
<td>Age 12 months</td>
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<tr>
<td>Breast milk plus gradual introduction of complementary foods after 6 months</td>
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<tr>
<td>Age 6 months</td>
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<tr>
<td>Exclusive breastfeeding from birth to 6 months</td>
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<td>Age 0 month</td>
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Figure 4.1.1 Optimal infant and young child feeding
in India are far from satisfactory (Fig. 4.1.2). According to the National Family Health Survey 3 (NFHS-3), introduction of complementary feeding along with continued breastfeeding in children of 6–8 months is only about 55%.

Addition of anything other than breast milk before 6 months is fraught with danger for the following reasons:

- Addition of foods and other liquids (including water, soup, juice, rice-water, dal-water, etc.) interfere with optimal breastfeeding. They may fill up the child’s stomach and quench the thirst and consequently, may lead to less suckling at the breast with reduced milk production.
- Increased risk of allergic disorders due to allergens passing through the not yet fully mature gut of the infant. It takes about 6 months after birth for the intestine to become reasonably mature. Enzymes needed to digest foods other than breast milk are also produced around 6 months.
- The tongue-thrust reflex is active before 6 months. Infants tend to push out with the tongue anything other than liquids.
- Foods other than breast milk may result in more gastrointestinal and other infections and malnutrition. They may put unnecessary load on the kidney and lead to obesity, hypertension and coronary artery disease later in life.
- Less frequent suckling also increases the possibility of the mother becoming pregnant again.

**Nutritionally Adequate Complementary Feeding**

To be nutritionally adequate, the complementary foods should contain all food groups—the staple, proteins, vitamins and minerals (Fig. 4.1.3, Tables 4.1.1 and 4.1.2).

After 6 months, add home-made porridge or a fruit like ripe banana. Porridge can be made with the staple cereal used by the family like whole wheat flour (atta), rice, semolina (suji or rava), broken wheat, ragi (nachni) or millet. Breast milk or any other milk can be used to make the porridge. Pieces of *chapatti* could also be soaked in milk, mashed properly and passed through a sieve to provide a soft semisolid food for the infant. Sugar and cream (malai) can be added to make it energy-dense.

Boiled or well-cooked mashed vegetables (pumpkin, peas, cauliflower, carrots, leafy vegetables, sweet and other potato, beet, tomato) should be added to provide vitamins and iron. Also, offer other seasonal fruits. Gradually, introduce *khichdi* with ghee or butter or oil, *seera* or halwa, *upma*, *poha*,

![Figure 4.1.2] Complementary feeding indicators in India (National Family Health Survey-3)

![Figure 4.1.3] Food square for the young child

dhokla, idli, dosa, pongal, missi roti (paratha made with a batter of wheat flour, gram flour, spices and dal). If foods of animal origin are acceptable to the family, flesh foods should be encouraged.

Start with only one food at a time. Wait for a week before introducing another food so that we know whether or not the child is tolerating it. Children who do not eat at a time should be offered food, fruits or milk-feed every 2–3 hours. The child should be encouraged but not forced to eat. Some children may choke a little while learning to eat. Parents may be told not get scared but to be with the child to ward off any trouble. When children are helped to use a spoon, let it be dipped into a preparation like shrikhand or phirni. A bit will stick to the spoon and give children the joy of getting something into their mouth on their own.

A child eating well around 7 months or so may suddenly become disinterested in eating. The parents should be told not to panic but to try some new preparation. In any case, children should never be forced to eat more than what they want. Even if half a spoon is left in the bowl and the child is not interested to eat any more, the parents should respect the child’s appetite. Water can be given, once the child starts taking foods other than breast milk and fruits. It should be offered from an ordinary glass. Children learn to sip from the glass within a few days.

Few general guidelines about the nutritive value of foods are:
- To retain the nutrients, vegetables including potatoes should be scraped instead of peeling them. The water in which rice or vegetables have been boiled for cooking

<table>
<thead>
<tr>
<th>Table 4.1.1: Foods rich in iron</th>
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<tbody>
<tr>
<td><strong>Foods rich in iron (mg/100 g of edible portion)</strong></td>
</tr>
<tr>
<td><strong>Vegetables</strong></td>
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<tr>
<td>Cauliflower leaves</td>
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<tr>
<td>Chaulai</td>
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<tr>
<td>Muli leaves</td>
</tr>
<tr>
<td>Suva ni bhaji</td>
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<tr>
<td>Pudina</td>
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<tr>
<td>Arvi pan green</td>
</tr>
<tr>
<td>Carrot leaves</td>
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<tr>
<td>Green onion</td>
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<tr>
<td>Kothmir</td>
</tr>
<tr>
<td>Spinach</td>
</tr>
<tr>
<td>Arvi pan black</td>
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<tr>
<td>Sargava pan</td>
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<tr>
<td><strong>Milk and its products</strong></td>
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<tr>
<td>Mawa-khoa</td>
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<tr>
<td>Cheese</td>
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<tr>
<td>Sugars</td>
</tr>
<tr>
<td>Jaggery</td>
</tr>
<tr>
<td>Sago</td>
</tr>
<tr>
<td><strong>Meat</strong></td>
</tr>
<tr>
<td>Liver (Sheep)</td>
</tr>
<tr>
<td>Egg (Hen)</td>
</tr>
<tr>
<td><strong>Cereals</strong></td>
</tr>
<tr>
<td>Rice flakes (Poha)</td>
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<tr>
<td>Pearl millet (Bajra)</td>
</tr>
<tr>
<td>Rice puffed (Mamra)</td>
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<tr>
<td>Wheat flour</td>
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<tr>
<td>Sorghum bicolor (Jowar)</td>
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<td>Vermicelli-sev (Wheat sev)</td>
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should be used and not thrown away. The vegetables should not be overcooked. Children should be encouraged to get used to the taste of properly washed raw vegetables from an early age. In general, some amounts of spices are good for health. Most children can tolerate moderate amount of spices used for cooking in most homes.

- Dry fruits are good, but they must also be rationed, partly because they are expensive and more so because some of these, like dates, figs and raisins can remain stuck between the teeth leading to caries. Foods that may lead to choking in children should be avoided in those below 3 years. Examples of these foods are—raw carrots, roasted grams, peanuts, other nuts, popcorn, hard candies, berries and whole grapes.
- Foods like idli and parathas made from whole-wheat flour should be preferred to white bread.

**Avoidable Foods**

Foods that may be avoided in the first year of life are discussed below:

In families with a strong history of allergy, peanuts and other nuts should be avoided. If any member of the family is known to react adversely to a particular food, avoid that as well. Also fried foods, foods containing too much sugar, artificial sweeteners, monosodium glutamate (MSG, ajinomoto) and high intake of salt should be avoided.

As recommended by the National Institute of Nutrition (NIN), the following points need to be kept in mind:

- The recipes for complementary foods should be based on locally available food stuffs.
- The cooking methods must be simple.
- The cost should be minimal.
- The recipes should be acceptable in taste and consistency.

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**Table 4.1.2: Foods rich in calcium and zinc**

<table>
<thead>
<tr>
<th>Foods rich in calcium (mg/100 g of edible portion)</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Milk products and dry fruits</td>
<td>Gingelly seeds</td>
</tr>
<tr>
<td>Sesame seeds</td>
<td>Bengal gram (Desi)</td>
</tr>
<tr>
<td>Cheese</td>
<td>Cashew nut</td>
</tr>
<tr>
<td>Khoya</td>
<td>Safflower seeds</td>
</tr>
<tr>
<td>Coconut (Dry)</td>
<td>Mustard seeds</td>
</tr>
<tr>
<td>Black sesame seeds</td>
<td>Cowpea</td>
</tr>
<tr>
<td>Almond</td>
<td>Omum seeds</td>
</tr>
<tr>
<td>Milk (Buffalo)</td>
<td>Red kidneybean (Rajma)</td>
</tr>
<tr>
<td>Curd</td>
<td>Soybean black</td>
</tr>
<tr>
<td>Pistachios</td>
<td>Poppy seeds</td>
</tr>
<tr>
<td>Milk (Cow)</td>
<td>Groundnut</td>
</tr>
<tr>
<td>Watermelon seeds</td>
<td>Samai almond</td>
</tr>
<tr>
<td>Walnut</td>
<td>Betel leaves</td>
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<tr>
<td>Cereal and pulses</td>
<td>Soybean white</td>
</tr>
<tr>
<td>Finger millet (Ragi)</td>
<td>Black gram (Whole)</td>
</tr>
<tr>
<td>Red kidney beans (Rajma)</td>
<td>Coriander seeds</td>
</tr>
<tr>
<td>Soybean</td>
<td>Sorghum bicolor (Bajra)</td>
</tr>
<tr>
<td>Turkish gram (Moth)</td>
<td>Lentil dal</td>
</tr>
<tr>
<td>Chickpeas</td>
<td>Red gram (Whole)</td>
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<tr>
<td>Black gram (Urad dal)</td>
<td>Fenugreek seeds</td>
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<tr>
<td>Vlung</td>
<td>Black gram (Dal)</td>
</tr>
<tr>
<td>Pigeon peas (Tuvar dal)</td>
<td>Green gram (Whole)</td>
</tr>
<tr>
<td>Bengal gram (Channa dal)</td>
<td>Sanwa millet</td>
</tr>
<tr>
<td>Cashew nuts</td>
<td>Garbanzo beans (Kabuli)</td>
</tr>
<tr>
<td>Poppy seeds</td>
<td>Cardamom</td>
</tr>
<tr>
<td>Almond</td>
<td>Maize (Dry)</td>
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<tr>
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<td>Maize (Dry)</td>
</tr>
</tbody>
</table>
• Gradually, the child should be introduced to healthy foods eaten by the rest of the family.

**Consistency of Complementary Foods (Fig. 4.1.4)**

- To provide more calories from smaller volumes, food must be thick in consistency—thick enough to stay on the spoon without running off when the spoon is tilted.
- Foods, such as nuts, which can pose choking hazard, should be avoided. Introduce lumpy or granular foods and new tastes by about 9–10 months. Missing this age may lead to fussy eating later. Avoid using mixers to make the food too smooth.
- 3–4 teaspoons of roasted groundnut powder can be added to the daily diet of the infant. The meal can also be made energy dense by adding ghee, butter, oil and sugar or jaggery.

### Table 4.1.3: Amount of food to be offered

<table>
<thead>
<tr>
<th>Age</th>
<th>Texture Description</th>
<th>Frequency</th>
<th>Average amount of each meal</th>
</tr>
</thead>
<tbody>
<tr>
<td>6–8 months</td>
<td>Start with thick porridge, well-mashed foods</td>
<td>2–3 meals per day plus frequent breastfeeding</td>
<td>Start with 2–3 tablespoon full</td>
</tr>
<tr>
<td>9–11 months</td>
<td>Finely chopped or mashed foods and foods that baby can pick up</td>
<td>3–4 meals along with breastfeeding. Depending on appetite offer 1–2 snacks</td>
<td>1/2 of a 250 mL cup or bowl</td>
</tr>
<tr>
<td>12–23 months</td>
<td>Family-food chopped or mashed, if necessary. As per appetite offer 1–2 snacks</td>
<td>3–4 meals plus breastfeeding. Depending on appetite offer 1–2 snacks</td>
<td>3/4 to one 250 mL cup or bowl</td>
</tr>
</tbody>
</table>

If baby is not breastfed, give in addition: 1–2 cups of milk per day and 1–2 extra meals per day.

The amounts of food included in the table are recommended when the energy density of the meals is about 0.8–1.0 kcal/g. If the energy density of the meals is about 0.6 kcal/g, recommend increasing the energy density of the meal (adding special foods) or increase the amount of food per meal. Find out what the energy content of complementary foods is in your setting and adapt the table accordingly.

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### Ready-to-use Infant Weaning foods

The following recipes can be prepared in bulk and kept ready at hand for feeding infants.

<table>
<thead>
<tr>
<th><strong>Pearl millet (Bajra)</strong></th>
<th><strong>Infant Food</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearl millet (Bajra), (dehusked, roasted)</td>
<td>3 tablespoons</td>
</tr>
<tr>
<td>Roasted green gram dal (Or any other dal)</td>
<td>1 1/2 tablespoons</td>
</tr>
<tr>
<td>Roasted groundnut</td>
<td>Three-fourths tablespoon</td>
</tr>
<tr>
<td>Roasted decorticated sesame (gingelly) seeds</td>
<td>1 tablespoon</td>
</tr>
<tr>
<td>Sugar</td>
<td>2 tablespoons</td>
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</table>

Powder all the roasted ingredients individually; mix them in the proportions suggested, and store in air-light containers.

**Finger Millet (Ragi, Nachni) Infant Food**

Use 45 grams of finger millet (ragi) prepared as given here instead of bajra in the earlier formula. Soak finger millet (ragi) in water overnight. Drain the water, spread the grains on a plate and allow them to germinate by covering with a damp cloth for one day. Dry the germinated finger millet (ragi) in sun and roast till it develops a malted flavor. Powder and store in an air-tight tin.

**Methods of Feeding**

When required, take suitable amounts (say 3 tablespoons) of any one of the above ready to use infant weaning food and mix with a small amount of hot water. Add more sugar or jaggery, if required, before feeding.

**Amount and Frequency of Food to be Offered (Table 4.1.3)**

This depends on the capacity or the size of the child’s stomach, which is usually 30 mL/kg of the child’s body weight. A child
who weighs 8 kg will have a stomach capacity of 240 mL, about one large full cup and cannot be expected to eat more than that at one meal. Parents may not realize that a child of 1 year needs about 1,000 calories each day—almost half of what an adult may take. So some guidance to parents may be given in this respect. After that, children should be left to decide how much they want to consume. A good guide that children were having optimum quantity of food is their level of activity and weight gain.

**Safe Complementary Feeding**

All utensils used for feeding must be washed thoroughly. There is no need to sterilize the utensils. Eating by hand need not be discouraged. Finger foods which the child can hold and chew may be given. However, the hands of the caregiver and the child must be washed thoroughly with soap and water before and after eating. Microbial contamination of complementary foods is more in hot weather. It is slower if the food is refrigerated. When that is not possible, the food should be eaten within 2 hours of its preparation. Even if the food is kept in the refrigerator, it should be consumed within a day or two.

**Responsive Feeding (Fig. 4.1.5)**

While feeding young children, the caregivers should provide psychosocial stimulation to the child through age appropriate play and praise. Children sitting on the lap of a caregiver or eating with loved ones learn to enjoy eating. Self-feeding must be encouraged even if the child makes a mess (Fig. 4.1.6). Forced feeding, threatening and punishment interfere with development of proper feeding habits. Distractions during meals and feeding in front of the television should be avoided.

**Feeding during and after Illness**

The appetite during an illness may go down. However, even sick babies continue to breastfeed quite often. They should be encouraged to take enough liquids and small quantities of nutrient rich food that they like to eat. After the illness, the nutrient intake can be increased by adding one or two extra meals in the daily diet for about a month by offering nutritious snacks between meals by giving extra amount at each meal and by continuing breastfeeding.

**Junk and Commercial Nutrition Supplements**

Commercial readymade cereals, though convenient to use are not preferred over homemade foods. Besides high cost, the smooth consistency of such products may make the children get so used to them that they may not accept homemade foods. Families must be equipped with means and knowledge to feed their children without the need for processed foods. All effort must be made to ensure that government policies protect public health before private profits.

Tinned food juices, cold-drinks, packaged drinks and packaged wafers, health drinks, nutrition supplements promoted for “picky eaters”, bakery products, drinks with low nutrient value such as tea, coffee and sugary drinks should be avoided.

**Picky Eaters**

Of late, a nutritional supplement is being aggressively promoted for so called “picky eaters” for better growth and height. Before the parents start using such a product, they should be given the following information:

- An infant grows rapidly in the early months of life. In the second year, the growth will be slower, the appetite may decrease and vary from day to day. Between 15 months and 3 years, the child often passes through a phase of negativism and does the opposite of what the parents
want. If the child is unwell, the appetite may suffer even more though the mother’s milk is often not refused.

- Keeping the earlier point in mind, the child should not be forced to eat. Of course, various healthy food options must be offered at frequent intervals. Allow children to eat with their hands even if it turns out to be a messy affair. Then let them learn to use the spoon. The parents may fill the spoon off and on or may offer the child some food with a separate spoon, while gradually encouraging the child to eat independently.
- Parents need not get upset if the child does not eat “nourishing” foods for a few days. Children have their moods; for some days, they may eat less of certain foods, but if left to themselves, they may start eating the same again after a gap of few days.
- Children, who are small at birth, may not weigh as much as their peers. The parents should be told that so long the child follows the growth curve, they should be happy. In fact, if these children are given food or products too high in calories and become obese, they become potential candidates for developing diabetes and cardiovascular diseases.
- Make sure the child is not anemic and does not have urinary or any other infection responsible for anorexia.
- Convey to the parents that the product promoted for “picky eaters” is expensive, not wholesome and comes in the way of the child developing healthy food habits. Also, one may get a false sense of security while the underlying causes for fussy eating mentioned earlier are missed.

**KEY MESSAGES FOR OPTIMUM INFANT AND YOUNG CHILD FEEDING**

- Initiate breastfeeding as early as possible after birth, preferably within 1 hour.
- With the exception of any essential medicine, practice exclusive breastfeeding from birth to 6 months of age and introduce complementary foods at 6 months (180 days).
- Continue frequent on-demand breastfeeding until 2 years of age or beyond.
- Start complementary foods at 6 months of age with small amount. Increase the quantity and frequency as the child gets older, while maintaining frequent breastfeeding.

- Gradually, increase food consistency and ensure that all nutrient needs of the child are met.
- Practice responsive feeding by applying the principles of psychosocial care.
- Practice good hygiene by hand-washing with soap and water before preparing food, before feeding the child and after using the toilet.
- Increase fluid intake during illness including more frequent breastfeeding, and encourage the child to eat soft favorite foods. After illness, encourage the child to eat more often.
- Ensure adequate nutrition including control of anemia in infants, young children, adolescent girls and pregnant and lactating mothers. Vitamin and mineral supplements must be given if required.
- Support the implementation and monitoring of Infant Milk Substitutes (IMS) Act.

**BIBLIOGRAPHY**

4.2 Breastfeeding

The provision has been made for infants to be feed upon their mother’s milk. They find their food and their mother at the same time. It is complete nourishment for them, body and soul. It is their first introduction to the great truth that man’s true relationship with the world is that of personal love and not that of the mechanical law of causation.

—Rabindranath Tagore

INTRODUCTION

Optimal infant and young child feeding (IYCF) practices are critical for child nutrition and survival. Breastfeeding is a vital component of IYCF. The "global strategy for IYCF" states that "breastfeeding is an unequalled way of providing ideal food for the healthy growth and development of infants; it is also an integral part of the reproductive process with important implications for the health of mothers. As a global public health recommendation, infants should be exclusively breastfed for the first 6 months of life to achieve optimal growth, development and health. Thereafter, to meet their evolving nutritional requirements, infants should receive nutritionally adequate and safe complementary foods while breastfeeding continues for up to 2 years of age or beyond." Presently, when global efforts are on to achieve better nutritional status and survival for children enshrined in the millennium development goals (MDGs), breastfeeding emerges as a very effective intervention to achieve these targets.

DEFINITIONS RELATED TO BREASTFEEDING

Various definitions used in context of breastfeeding are summarized in the Table 4.2.1.

Recommendations for Breastfeeding

Child health and nutrition programs all across the world (including India) conform to these guidelines based on the global recommendations. These recommendations are based on the available scientific evidence, some of which are defined as follows:

Initiation of Breastfeeding Immediately after Birth, Preferably within 1 Hour

Early initiation has been documented to improve neonatal survival, and protective against the infection specific mortality among newborn infants. Early initiation of breastfeeding helps to develop a bond between a mother and her baby. Early initiation is extremely important to establish successful and sustained lactation. It stimulates contractions and expulsion of placenta. The practice of delaying breastfeeding after birth and giving something else, i.e. prelacteal feeds expose the infant to infections and also lead to problems in establishing a successful lactation. Scientific evidence suggests that early is the initiation of breastfeeding, more are the chances of survival of neonate.

After cesarean section, some delay in initiation of breastfeeding may be unavoidable due to the condition of the mother or infant. After cesarean section with spinal anesthesia, breastfeeding can often be initiated immediately. With general anesthesia, breastfeeding can be initiated within a few hours as soon as the mother regains consciousness.

Exclusive Breastfeeding for the First 6 Months

Exclusive breastfeeding is recommended as breast milk contains all the necessary nutrients which are sufficient to sustain appropriate growth and development of a healthy term infant for the first 6 months of life. There is sufficient evidence that a significant number of under-5 month deaths in resource poor countries could be prevented through achievement of 90% coverage with exclusive breastfeeding for 6 months. Any supplementation during the first 6 months
will expose infant to infections and also decrease breast milk output.

**Appropriate and Adequate Complementary Feeding after 6 Months of Age while Continuing Breastfeeding**

Additional foods are needed at this stage to complement the breast milk to sustain the growth and development of the infant. Along with the breastfeeding, children age 6–24 months should be fed from three or more different food groups; two to three times a day (see more details in Chapter 4.1).

**Continued Breastfeeding up to the Age of 2 Years or Beyond**

Breastfeeding along with other foods remains an important and safe source of high-quality protein, energy and other nutrients like vitamin A and vitamin C between 6 months and 24 months of life. It is, therefore, crucial in preventing undernutrition and morbidities. It can provide about one-third of energy needs, half of protein and 75% of the vitamin A requirements of a child of this age (Fig. 4.2.1). Thus, breast milk helps a child to get enough energy and high quality nutrients from breastfeeding during the second year of life. These nutrients may not be easily available from the family diet. Continuing to breastfeed during the second year can help to prevent malnutrition and vitamin deficiencies.

**STATUS OF BREASTFEEDING PRACTICES IN INDIA**

The status of breastfeeding and complementary feeding practices is very dismal in India. According to the National Family Health Survey-3 (NFHS-3), only 24.5% of children are breastfed within the first hour of birth and about 55% initiate breastfeeding within first day of life. More than half of newborn infants receive prelacteal feeds, like milk other than breast milk, honey, sugar or glucose water, and plain water. The exclusive breastfeeding rate up to the age of 6 months is only 46.4%. Exclusive breastfeeding rapidly declines from 1st month to 6th months, and only about 20% children continue it by 6 months.

Possible reasons for suboptimal breastfeeding are primarily due to lack of proper information to mothers, inadequate healthcare support, inability of the healthcare providers to help mothers experiencing breastfeeding difficulty, aggressive promotion of baby foods by the commercial industry and lack of proper support structures at the community and at workplace which includes maternity entitlements and crèches. Cultural beliefs also appear to be important, e.g. breastfeeding initiation is delayed because of the belief that mother’s milk does not “come” at the time of childbirth but flows 2–3 days later.

**NUTRITIONAL COMPOSITION OF BREAST MILK**

The breast milk contains all the macronutrients (carbohydrates, proteins and fats), micronutrients such as vitamins and minerals, and adequate water to meet the requirements of a healthy term infant for the first 6 months of life. Apart from the nutrients, breast milk provides a variety of bioactive factors which protect the infant against infection, and also modulate the composition of the indigenous intestinal microbiota. Breast milk also contains some factors to help in digestion and absorption of nutrients.

**Fats**

The mature human breast milk contains 3.2–3.8 g/dL of fats. Fats provide 50% of the total energy content of the breast milk. Breast milk fat in immediate postpartum period contains fat needed for gray matter development and in later months, fat which is needed for myelination. Breast milk fat has steady higher level of cholesterol than animal milks and formula. Breastfed babies have significantly higher total cholesterol and low-density lipoprotein (LDL) cholesterol compared to mixed fed babies in the first 6 months of life with improving high-density lipoprotein (HDL) cholesterol/LDL cholesterol ratio at 6 months. High cholesterol intake in infancy may have a beneficial long-term programming effect on synthesis of cholesterol by down regulation of hepatic enzymes. Human milk contains essential fatty acids and n-3 fatty acids (docosahexaenoic acid and eicosapentaenoic acid), which are needed for a baby’s growing brain and eyes and for healthy blood vessels. Human milk contains the enzyme lipase which helps to digest fat. Thus the fat in breast milk is more completely digested and more efficiently used by a
Nutrition

baby’s body than the fat in cow’s milk or formula. The lipase in breast milk is called bile salt stimulated lipase because it starts working in the intestine in the presence of bile salts. The lipase is not active in the breast, or in the stomach before the milk mixes with bile.

Carbohydrates

Lactose is the main carbohydrate in human breast milk and provides about 50% of its energy content. Breast milk also contains oligosaccharides such as glucose, galactose, N-acetylglucosamine and sialic acid. These oligosaccharides attach to the epithelial cell surface in the intestines and prevent adhesion of microorganisms thereby preventing their growth.

Proteins

Proteins in breast milk provide amino acids for growth and anti-infective factors. Mature breast milk contains 0.9 g/dL of protein while colostrum contains 2.3 g/dL. The breast milk protein contains more whey protein and less casein. Due to high whey to casein ratio, the breast milk forms softer curds which are easier to digest.

In human milk, much of the whey protein consists of anti-infective proteins, which help to protect a baby against infection. The anti-infective proteins in human milk include lactoferrin (which binds iron and prevents the growth of bacteria which need iron) and lysozyme (which kills bacteria) as well as antibodies (immunoglobulin, mostly IgA). Animal milk and formula may lack the amino acid cystine, and formula may lack taurine which newborns need especially for brain growth. Human infant is ill equipped to handle phenylalanine and tyrosine, two amino acids which are in high concentration in the animal milk in comparison to breast milk.

DYNAMIC COMPOSITION OF BREAST MILK

The composition of breast milk is not always the same. It varies according to the age of the baby, and from the beginning to the end of a feed. It also varies between feeds, and may be different at different times of the day.

Colostrum

The milk produced during the first few days after the delivery is known as colostrum, which is a special, thick, sticky, bright lemony yellowish fluid. It is secreted in small quantities for first 3–4 days of life. Although it is in small quantities, it is sufficient to meet the needs of the newborn baby. Colostrum contains more protein than later milk. Colostrum is considered the first immunization for newborn as it is rich in the anti-infective factors that helps protect the baby against diarrhea, respiratory and other infections. Colostrum contains more epidermal growth factors in comparison to mature breast milk, which help a baby’s immature intestine to develop after birth. This helps to prevent the baby from developing allergies and intolerance to other foods. Colostrum helps to clean baby’s intestine which is important to prevent jaundice in the newborn. Colostrum is also rich in vitamin A.

Transitional Milk

During the transition from colostrum to the mature milk, the amount of immunoglobulin, proteins, vitamin A and vitamin E decreases, and amount of lactose, fats, energy and water-soluble vitamins increases.

Mature Milk

After a few days, colostrum changes into mature milk. Mature milk is in large amounts and the breasts feel full, hard and heavy. Some people call this as the milk “coming in”. Foremilk is the bluish milk that is produced early in a feed. Foremilk is produced in larger amounts, and it provides plenty of protein, lactose and other nutrients. Because a baby gets large amounts of foremilk, he or she gets all the water that he or she needs from it. Hindmilk is the whiter milk that is produced later in a feed. It contains more fat than foremilk. This fat provides much of the energy of a breastfeed. This is why it is important not to take a baby off a breast too quickly, not until he or she leaves the breast on her/his own.

BENEFITS OF BREASTFEEDING

The benefits of breastfeeding for infant, mother and community include:

• Breastfeeding provides all the nutrients a baby needs for the first 6 months of life, after which it continues to provide a major portion of the infant's nutrition along with appropriate family foods. It provides almost half of the nutritional requirements between 6 months and 12 months of age, and upto one-third between 12 months and 24 months of age
• Breast milk is easily digested by the baby
• Breast milk contains antibodies and other factors which protect the baby against diarrhea and other infections
• Breast milk contains enough water which is sufficient even for very dry and hot climates
• Breast milk is clean, safe and cheap
• Breastfeeding provides a perfect opportunity for building a close bond between mother and baby
• It helps the mother by reducing the postdelivery bleeding and thus preventing anemia
• Breastfed babies are less prone to have diabetes, heart disease, eczema, asthma, rheumatoid arthritis and other allergic disorders later on in life
• Breastfeeding enhances brain development, visual development and visual acuity leading to learning readiness
Breastfeeding has contraceptive effect for the mother, if she exclusively breastfeeds her infant for first 6 months.

- Mothers have a lower risk of breast and ovarian cancers.
- Breastfeeding costs less in terms of healthcare expenses as breastfed infants get ill less often.
- Breastfeeding protects the environment.

### Risks of Formula Feeding

Infant formula, which is generally used as an artificial substitute for human breast milk, is time consuming, less nutritious and expensive. It is also fraught with innumerable risks for the infants and children in comparison with the breastfeeding. Some of these risks are depicted in the Table 4.2.2.

<table>
<thead>
<tr>
<th>Risks of Formula Feeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased risk for infection from inherent and subsequent contamination of formula with</td>
</tr>
<tr>
<td>microbes like <em>Enterobacter sakazakii</em> and <em>Salmonella</em></td>
</tr>
<tr>
<td>Increased risk of acute respiratory infections, diarrhea, otitis media and ear infections</td>
</tr>
<tr>
<td>Increased risk of necrotizing enterocolitis</td>
</tr>
<tr>
<td>Increased risk of asthma and other allergies</td>
</tr>
<tr>
<td>Reduced cognitive development</td>
</tr>
<tr>
<td>Increased risk of chronic diseases like type 1 diabetes, ulcerative colitis and Crohn's</td>
</tr>
<tr>
<td>disease</td>
</tr>
<tr>
<td>Increased risk of cardiovascular disease, increased blood pressure, obesity, altered blood</td>
</tr>
<tr>
<td>cholesterol levels and atherosclerosis in later adulthood</td>
</tr>
<tr>
<td>Increased risk of side effects of environmental contaminants and harmful chemicals like</td>
</tr>
<tr>
<td>melamine and bisphenol A (BPA)</td>
</tr>
</tbody>
</table>

### Science of Milk Transfer

Understanding the structure of breast and the process of breast milk production and transfer to the infant is useful to provide effective skilled help to the lactating mother.

### Anatomy of the Breast

The human breast consists of the nipple and areola, mammary tissue, the soft tissue including supporting connective tissue and fat, blood and lymphatic vessels and nerves (Fig. 4.2.2). The nipple is the area from which the milk comes out of the breast through multiple small openings. This area of breast is very richly supplied with nerves. The nerve endings in the nipple are important to provide stimulus for the hormonal reflexes important for production and release of the milk from breast. The areola is the dark skin surrounding the nipple. The milk ducts beneath the areola are filled with milk and become wider during a feed. Areal is an important anatomical landmark as it is important to ensure that majority of areola is in baby's mouth during the feed to achieve an effective sucking. The mammary tissue is composed of alveoli, which are small sacs, made up of millions of milk secreting cells.

### Physiology of Lactation

**Production of Breast Milk**

Production of the breast milk is controlled by the hormone prolactin. When a baby suckles at the breast, sensory impulses go from the nipple to the brain. In response, the anterior part of the pituitary gland at the base of the brain secretes prolactin. Prolactin goes in the blood to the breast and makes the milk secreting cells produce milk. This process is known as the prolactin reflex (Fig. 4.2.3). From this, it is evident that milk production is dependent on the suckling stimulus. If the baby suckles more, the breast will produce more milk. For the same reason, if a mother has two babies, breast milk production increases accordingly. Prolactin is present in the blood for about 30 minutes after the baby finishes the feed. It makes the breast produce milk for the next feed. More prolactin is produced at night due to the inhibition of dopaminergic drive during sleep so breastfeeding at night is especially helpful for keeping up the milk supply. Prolactin suppresses ovulation so breastfeeding can help to delay a new pregnancy.

**Flow of Breast Milk**

When a baby suckles, sensory impulses go from the nipple to the brain. In response, the posterior part of the pituitary...
gland at the base of the brain secretes the hormone oxytocin. Oxytocin goes in the blood to the breast and makes the muscle cells around the alveoli contract. This makes the milk which has collected in the alveoli flow along the ducts toward nipple. It makes the milk in the breast flow for this feed (Fig. 4.2.4). Sometimes the milk is ejected in fine streams. This is the oxytocin reflex or the milk ejection reflex. Oxytocin can start working before a baby suckles, when a mother expects a feed. The oxytocin reflex is positively affected by mother’s sensations and feelings like thinking lovingly about the baby; touching, smelling or seeing the baby; or hearing the baby cry. If the oxytocin reflex does not work well, the baby may have difficulty in getting the milk. This may happen, if the mother is emotionally disturbed or experiencing pain and discomfort. In such a condition, mother needs support to make her physically and/or emotionally comfortable to make the oxytocin reflex work again and let the milk flow.

Signs of an active oxytocin reflex are a tingling sensation in the breast before or during a feed, milk flowing from breasts when mother thinks of the baby or hears him/her crying, milk flowing from the other breast when the baby is suckling, milk flowing from the breast in streams, if suckling is interrupted, and uterine pain or a flow of blood from the uterus during the feed. However, the absence of these signs does not indicate an inadequate oxytocin reflex.

Breast Milk Inhibitor

Breast milk production is also controlled within the breast itself. Sometimes one breast stops making milk, while the other breast continues to make milk although oxytocin and prolactin go equally to both breasts. There is a substance in breast milk which can reduce or inhibit milk production. If a lot of milk is left in a breast, the inhibitor stops the cells from secreting any more. If breast milk is removed by suckling or expression, the inhibitor is also removed and the breast makes more milk.

Positioning and Attaching the Baby at the Breast

For effective milk transfer from mother to the infant, good breastfeeding skills including proper positioning of the baby and good attachment at the breast are required.

Positioning

A woman can feed her baby in any comfortable position such as sitting, lying or even standing. If the baby suckles properly from the breast, he or she will get sufficient milk. However, for a good attachment on breast, some basic principle need to be observed for relative positioning of the baby while breastfeeding. These are:

- Baby turned towards mother and his or her ears, shoulder and buttock are in a straight line
• His face should face the breast with nose opposite the nipple
• Mother should hold the baby close to her
• In a newborn, she should support his bottom with hand and not just his head and shoulders.

The mother should be explained how to support the breast with her hand while offering it to the baby:
• With her fingers and palm placed on her chest wall below the breast so that her first finger forms a support at the base of the breast
• With her thumb placed on the top of the breast so that it is easier for her baby to attach well.
• She should not hold her breast too near to the nipple.

The mother should be explained how to bring the baby to the breast:
• Touch baby’s lips with her nipple
• Wait until baby’s mouth is wide open
• Move the baby quickly onto the breast from below.

Attachment

This is important how baby’s mouth is attached to mother’s breast for a successful suckling (Figs 4.2.5 and 4.2.6). In good suckling position, baby is suckling with nipple and a larger breast tissue having in his or her mouth. In poor suckling position, baby is suckling with nipple only or nipple with a small breast tissue in his or her mouth.

Signs of good suckling attachment are:
• The baby’s chin touches the breast
• His mouth is wide open
• His lower lip is turned outwards
• One can see more of the areola above his or her mouth and less below. This shows that he or she is reaching with his tongue under the lactiferous sinuses to press out the milk.

Poor attachment may lead to pain and damage to mother’s nipple and she may develop sores or fissures in nipple. It may also lead to engorgement of the breast due to improper milk

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Figures 4.2.5A and B  Good and poor attachment. External signs. (Reproduced with permission from World Health Organization (WHO). Infant and young child feeding (IYCF); model chapter for textbooks for medical students and allied health professionals. Geneva: WHO press; 2009)

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Figures 4.2.6A and B  (A) Good attachment and; (B) poor attachment inside the infant’s mouth. (Reproduced with permission from World Health Organization (WHO). Infant and young child feeding (IYCF); model chapter for textbooks for medical students and allied health professionals. Geneva: WHO press; 2009)
removal. The baby remains hungry and frustrated that leads to refusal to suck. Ultimately, it leads to production of less milk in the breast; baby is not able to feed properly, leading to weight loss. Common causes of poor attachment are use of feeding bottle, inexperience of mother and lack of skilled support.

**Practices for Successful Breastfeeding**

To ensure adequate milk production and flow for 6 months of exclusive breastfeeding and thereafter continued breastfeeding, certain practices are very important.

- The infant should be fed as frequently and for as long as he or she wants to, during both day and night. The sucking should be allowed until the infant spontaneously releases the nipple. This is called demand feeding. Restricting length of the breastfeeding session may result in the baby getting less of the energy rich hindmilk. The 24 hour average intake of milk is about 800 mL per day during the first 6 months.
- At the time of delivery, before breastfeeding is initiated, no prelacteal feed should be given to the infant. Apart from having the harmful effects on the infant like risk of infection, such a practice may interfere in the establishment of breastfeeding. Later on, in the first 6 months of life, no supplementary feed, like other milks, should be given to the infant. This may lead to a decreased supply of breast milk.
- Sometimes, mother may have the perception that her milk is not sufficient for her infant. Adequacy of breastfeeding may be ascertained by documenting, if the infant has regained the birth weight by 2 weeks of age, and the cumulative weight gain is more than 500 g in a month and the infant is passing adequate urine at least six times a day, while on exclusive breastfeeding.

**Hospital Practices and Breastfeeding**

Maternity homes and healthcare practices should support exclusive breastfeeding during the first 6 months of life and continued breastfeeding along with appropriate complementary feeds thereafter. To ensure successful breastfeeding, the World Health Organization (WHO) and the United Nations Children’s Fund (UNICEF) launched the baby-friendly hospital initiative (BFHI) in 1991. The initiative is a global effort for improving the role of maternity services to enable mothers to breastfeed babies for the best start in life. It aims at improving the care of pregnant women, mothers and newborns at health facilities that provide maternity services. The initiative has measurable and proven impact, increasing the likelihood of babies being exclusively breastfed for the first 6 months.

**Components of Baby-friendly Hospital Initiative**

A maternity facility can be designated “baby-friendly” when it has implemented ten steps given in the Table 4.2.3 to support successful breastfeeding.

**Table 4.2.3: Ten steps of baby-friendly hospital initiative**

<table>
<thead>
<tr>
<th>Step</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Have a written breastfeeding policy that is routinely communicated to all healthcare staff</td>
</tr>
<tr>
<td>2.</td>
<td>Train all healthcare staff in skills necessary to implement this policy</td>
</tr>
<tr>
<td>3.</td>
<td>Inform all pregnant women about the benefits and management of breastfeeding</td>
</tr>
<tr>
<td>4.</td>
<td>Help mothers initiate breastfeeding within a half-hour of birth</td>
</tr>
<tr>
<td>5.</td>
<td>Show mothers how to breastfeed and maintain lactation, even if they should be separated from their infants</td>
</tr>
<tr>
<td>6.</td>
<td>Give newborn infants no food or drink other than breast milk unless medically indicated</td>
</tr>
<tr>
<td>7.</td>
<td>Practice “rooming in”—allow mothers and infants to remain together 24 hours a day</td>
</tr>
<tr>
<td>8.</td>
<td>Encourage breastfeeding on demand</td>
</tr>
<tr>
<td>9.</td>
<td>Give no artificial teats or pacifiers (also called dummies or soothers) to breastfeeding infants</td>
</tr>
<tr>
<td>10.</td>
<td>Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from the hospital or clinic</td>
</tr>
</tbody>
</table>

**Breastfeeding the Preterm Babies**

The nutritional management plays a large role in the immediate survival and subsequent growth, and development of the preterm infants. The optimal diet for premature infants should support growth at intrauterine rates without imposing stress on the infant’s immature metabolic and excretory functions and ensures healthy short-term and long-term outcomes. Breast milk produced during early postpartum period offers nutritional advantage because of its higher protein and electrolyte concentrations. Preterm infant fed preterm milk demonstrate increase in weight, length and head circumference as well as retention rates of various nutrients comparable to those for the fetus of similar postconception age. Fat absorption in preterm babies fed their own mother’s milk is significantly higher in comparison to infants fed cow’s milk formula. Long-chain polysaturated fatty acids (LCPUFAs), which are important for mental and visual development, are also higher in human milk.

Preterm infants fed breast milk have lesser incidence of necrotizing enterocolitis in comparison to feeding with formula milk. Even if the disease occurs in infants fed with breast milk, the course of disease is less severe and the prevalence of intestinal perforation is lower. This is due to various protective factors in breast milk like immunoglobulin,
erythropoietin, interleukin-10 (IL-10), epidermal growth factor, platelet activating factor, acetylhydrolase and oligosaccharides which are in greater quantity than in term milk. These factors may prevent intestinal attachment of enteropathogens by acting as receptor homologues resulting in the suppression of enteral colonization with harmful microorganisms. Breast milk also prevents a host of neonatal infections, a leading cause of neonatal mortality across the globe. The use of human milk can be adopted as an important healthcare intervention in neonatal units.

**Breast Conditions and Difficulties in Breastfeeding**

There are several common breast conditions which sometimes cause difficulties with breastfeeding. Management of these conditions is important both to relieve the mother and to enable successful breastfeeding. The difficulties in breastfeeding can be overcome by careful guidance, reassurance and encouragement to the mother during antenatal period to prepare for breastfeeding and by providing skilled counseling after birth.

**Flat Nipple**

Many a times, mother becomes apprehensive that a flat nipple is a hindrance in successful breastfeeding. However, in a good suckling attachment, the infant takes the nipple and the breast tissue underlying the areola into his mouth to form a “teat”. The anatomical nipple only forms about one-third of the “teat” of breast tissue in the baby’s mouth. This is therefore evident that shape of the nipple is immaterial for successful suckling. The nipple is just a guide to show where the baby has to take the breast. A woman with flat nipples should be reassured that she has normal nipples, even if they look short provided her nipples protract easily.

**Inverted Nipple**

Sometimes a nipple does not protract and on attempting to pull out the nipple, it goes deeper into the breast. The condition is known as inverted nipple (Fig. 4.2.7). The mother needs support in such a situation. She should be reassured that with some help she will be able to breastfeed her infant successfully. Help is most important soon after delivery when the baby starts breastfeeding.

A mother with the inverted nipple may be helped with the syringe method as follows (Fig. 4.2.8):

- Cut the nozzle end of a disposable syringe (10–20 mL).
- Introduce the piston from the ragged cut end side.
- Ask the mother to apply the smooth side of the syringe on the nipple and gently pull out the piston and let her wait for a minute.
- Nipple would then protrude into the syringe. Ask the mother to slowly release the suction and put the baby to breast; at this time it helps the nipple to erect out and baby is able to suckle in the proper position.
- After feeding the nipple may retract back, but doing it each time before feeding over a period of few days will help to solve the problem.

**Engorgement of Breasts**

If breasts are not emptied, the milk gets collected in the breast leading to engorgement. The engorged breast is tight, shiny
(because of edema) and painful. Also, the milk may stop flowing. The factors which cause engorgement of breasts are:

- Giving prelacteal feeds to the baby
- Delayed initiation of breastfeeds
- Long intervals between feeds
- Early removal of the baby from the breast
- Bottle-feeding and any other restrictions on breastfeeding.

Engorgement of the breast can be prevented by avoiding factors mentioned earlier. If the baby is able to suckle, he or she should feed frequently. If pain and tightness of the breast does not allow suckling, expressed milk may be given to the infant with cup/spoon. Once the mother feels comfortable, she should be advised again to breastfeed the infant on demand. Edema of the breasts may be reduced by applying cold compress. Engorged breasts may cause mild fever, which subsides spontaneously within a day or two.

**Mastitis and Abscess**

Mastitis is an inflammation of the breast which becomes red, hot, tender and swollen. The mother feels sick, has fever and severe pain in breast. Mastitis usually affects a part of the breast and usually unilateral. Mastitis may develop in an engorged breast, or it may follow a condition called blocked duct. Mastitis must be treated promptly and adequately. If treatment is delayed or incomplete, there is an increased risk of developing breast abscess. An abscess is when a collection of pus forms in part of the breast. The most important part of treatment is supportive counseling and improved drainage of milk from the affected part of the breast. The mother needs clear information and guidance about all measures needed for treatment, how to continue breastfeeding or expressing milk from the affected breast. This is important to help the mother to improve infant’s attachment at the breast with frequent unrestricted breastfeeding. If necessary express breast milk by hand or with a pump until suckling is resumed. Antibiotic should be given, if laboratory tests indicate infection, symptoms are severe, or symptoms do not improve after 12-24 hours of improved milk removal. Pain should be treated with an analgesic and warm packs to the breast. Incision and drainage should be done, if abscess develops.

**Sore and Cracked Nipples**

The most common cause of sore nipples is poor attachment in which the infant pulls the nipple in and out as he or she sucks and rubs the skin of the breast against his or her mouth. If the baby continues to suckle in this way, it damages the nipple skin and causes a crack or fissure. Oral thrush in the infant’s mouth is another important cause of sore nipple but it usually develops when a baby is few weeks old. The situation is very painful for the mother. If a mother has sore or cracked nipples, improving infant’s attachment to the breast relieves the pain. Medicated creams are best avoided as they may worsen the soreness. Hindmilk, which is rich in fat, should be applied on the nipple after feeding. For oral thrush 1% gentian violet should be applied over the nipple as well as inside the baby’s mouth.

**Breastfeeding and Maternal Illness**

Maternal illnesses can have adverse effects on lactation. A sick woman may perceive that her milk supply has gone down because of illness. She may also believe that her milk will make the baby ill. These factors may lead to discontinuation of breastfeeding. Minor illnesses such as cold and other mild viral infection, which are self-limiting, should not prevent a mother to continue breastfeeding. However, major illness requires a more careful approach. The potential role of breastfeeding in the transmission of infections must also be acknowledged and appropriate precautions should be taken. If the mother has tuberculosis, the mother-infant dyad should be treated together and breastfeeding should be continued. Similarly, in case of hepatitis (A, B and C) breastfeeding can continue normally as the risk of transmission by breastfeeding is very low. In human immunodeficiency virus (HIV) positive mother, mother should be provided with counseling and support for appropriate infant feeding practice. With adequate and appropriate antiretroviral drugs to mother and infant, exclusive breastfeeding for first 6 months of life is now the preferred recommendation in India.

Certain maternal drugs may affect the breastfed infant adversely as they are secreted in the breast milk. Breastfeeding should be avoided, if mother is consuming cytotoxic drugs, like cyclophosphamide, methotrexate and doxorubicin, radioactive compounds like gallium 67 (67Ga), indium 111 (111In), iodine 131 (131I) and technetium 99m (99mTc).

**Infant Feeding during Emergencies**

In disasters and emergencies like earthquakes, floods, typhoons and tsunami, breastfeeding is the safest, often the only reliable choice for infants and young children. It provides adequate and appropriate nutrition to the affected infants in a situation where child survival is a key issue. In disasters, infants are more likely to become ill and die from malnutrition. Uncontrolled distribution of breastmilk substitutes during disasters may lead to early and unnecessary cessation of breastfeeding. For the vast majority of infants, emphasis should be on protecting, promoting and supporting breastfeeding and ensuring timely, safe and appropriate complementary feeding.

**Protecting Breastfeeding from Commercial Influence**

During last many decades, extensive promotion by the infant food manufacturing companies through advertisements, free samples, gifts to mothers and health workers has led to convince them that formula feeding is as good as
IAP Textbook of Pediatrics

Malnutrition

INTRODUCTION

"Underlying every other condition is malnutrition, due to both calorie and protein deficiency. Though poverty is the main contributing cause, it is greatly aggravated by lack of proper dietary knowledge".

Malnutrition is the gravis single threat to the world’s public health. Protein energy malnutrition (PEM) is one of

breastfeeding. This has also made a dent in the confidence of lactating women in her capacity to optimally breastfeed and has contributed to the decline of breastfeeding rates. Recognizing this trend, the Indian parliament enacted the “infant milk substitutes (IMS), feeding bottles and infant foods (regulation of production, supply and distribution) Act 1992 IMS Act”. The IMS Act was further amended in the year 2003. The IMS Act controls marketing and promotion of infant milk substitutes, infant foods and feeding bottles. Some salient features of the IMS Act include:

- It bans any kind of promotion or advertisement of infant milk substitutes, infant foods and feeding bottles to the public including electronic and print media.
- It prohibits providing free samples of infant milk substitute, infant foods and feeding bottles and gifts to any one including pregnant women, mothers of infants and members of the families.
- It prohibits donation of free or subsidized supplies of infant milk substitute, infant foods and feeding bottles for healthcare institutions except donations to the orphanages.
- It prohibits display of posters of infant milk substitutes, infant foods and feeding bottles at healthcare facilities, hospitals and health centers.
- It prescribes rules for information on the containers and labels of infant milk substitutes and infant foods including a specific statement in English and local languages that “mother’s milk is best for the baby” in capital letters.
- It prohibits having pictures of infants or women or phrases designed to increase the sale of the product on the labels of the products.
- It prohibits any contact of employers manufacturing and distributing company with pregnant women even for providing educational material to them.
- It prohibits direct or indirect financial inducement or gift to health worker or to any members of his family by the producer, supplier or distributor of the infant milk substitute, infant foods and feeding bottles.
- The IMS Act also prohibits offering or giving any contribution or pecuniary benefit to a health worker or any association thereof including funding of seminar, meeting, conference, educational course, contests, fellowship, research work or sponsorship, etc. by the manufacturers, supplier or distributors of the products mentioned earlier.
- It prescribes standards for the infant milk substitute, infant foods and feeding bottles.

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3. Breastfeeding Promotion Network of India. Infant and young child feeding counseling—a training course, the “4 in 1 course” (integrated course on breastfeeding, complementary feeding and infant feeding and HIV). New Delhi: Breastfeeding Promotion Network of India; 2015.
the most widely spread health and nutritional problems of the developing countries. Annually, undernutrition kills or disables millions of children. It often causes disease and disability in the survivors and prevents millions from reaching their full potential. Under-nutrition as of 2010 was the cause of 1.4% of all disability adjusted life years (DALYs). WHO estimates that malnutrition is the biggest contributor to child mortality in under five children, accounting for 54% child deaths worldwide, translating to an unnecessary loss of about 3 million young lives every year (Fig. 4.3.1).

**MAGNITUDE OF THE PROBLEM**

Of the 146 million underweight children (<5 years old) from the developing world, 73% or 106 million live in just 10 countries. India is home to 57 million of this. In 2013 as per the global estimates of under five children, one in four of world’s children was chronically malnourished, 161 million were stunted and 51 were wasted with 17 million severely wasted. Approximately, half of these stunted and two-thirds of all wasted children lived in Asia and almost one-third of both stunted and wasted lived in Africa (Figs 4.3.2 to 4.3.4).

Despite India’s 50% increase in GDP since 1991, progress with largest food and nutrition programs, more than one-third of world’s malnourished children belong to India. Among these one-half of them under 3 years are underweight and one-third of the wealthiest children are overnutriented. These alarming statistics have dire consequences for morbidity, mortality, productivity and economic growth. The prevalence of malnutrition in India varies statewise with highest in Madhya Pradesh, Jharkhand and Bihar and lowest in Mizoram, Sikkim, Manipur, Kerala, Punjab and Goa, although the rate is still considerably higher than that of developed nations. It is more in rural areas and generally in those who are poor.

The PEM has higher incidence in nutritionally vulnerable groups, young children between 6 months to 2 years and women during pregnancy and lactation and the elderly age group as the nutritional requirements are larger relative to their body size than in older children and adults. The damage caused by malnutrition in the intrauterine life or in the first 2 years of life may be due to impairment in the developing brain.

**DEFINITIONS AND CLASSIFICATIONS**

The World Health Organization (WHO) defines PEM as range of pathological conditions arising from coincidental lack in varying proportions of proteins and calories, occurring most frequently in infants and young children, and commonly associated with infection. The extent of weight loss and growth rate varies with severity of PEM—in early stages, there is failure to maintain weight or growth rate but as it becomes progressive, there is loss of weight associated with loss of subcutaneous fat and muscle mass with dysfunction of many vital organs, which leads to a variety of clinical features. With increasing severity, there is increasing failure in the homeostatic mechanisms of the body and damage to the immune defenses which may result in infections, shock and death.

Protein-energy malnutrition is a generalized syndrome complex and it is very difficult to classify it using a single parameter. A large number of classifications using anthropometric, clinical and biochemical parameters have been proposed. Nutritional anthropometry is a valuable index of assessment of nutritional status of children and mothers. Among the most studied are weight, length or height, arm circumference, skinfold thickness and head circumference.
Undernutrition contributes to half of all deaths in children under 5 years and is widespread in Asia and Africa.

**Figure 4.3.2** Percentage of children under 5 years, who are underweight by region (1990–2013)

*Abbreviations: CEE, Central and Eastern Europe; CIS, Commonwealth Independent States*

*Source: UNICEF/WHO/World Bank Joint Child Malnutrition Estimates, 2014*

**Figure 4.3.3** Percentage of under 5 years children, who are stunted; 2008–2013

*Source: UNICEF/WHO/World Bank Joint Child Malnutrition Estimates, 2014*
Since Gomez first proposed classification based on weight for age, standard weight for age measurement used was Harvard growth standard, 50th centile being 100%, many classifications have been suggested (Table 4.3.1). In 2009, WHO recommended new growth standards replacing the earlier National Centre for Health Statistics (NCHS) reference charts.

Table 4.3.2 presents the diagnostic criteria for severe acute malnutrition (SAM) based on WHO growth standards. Moderate malnutrition is defined when the weight for length or height is between –2SD and –3SD, or when the midarm circumference is between 11.5 cm and 12.5 cm.

**ACUTE VERSUS CHRONIC DEFICIENCY**

Weight and arm circumference are affected within a short duration of inadequate nutrient intake and ill-health, while height and head circumference do not change so rapidly. A slowing in the rate of growth indicated by poor gain in height would take at least 6 months to manifest itself, while a slowing of weight gain or loss can be demonstrated within a month. A child can lose weight but not height.

Anthropometric parameters can be classified into two main groups—(1) the age dependent and (2) age independent criteria if child’s age is not known.

**Age Dependent Criteria**
- Weight for age (WFA) expressed as percentage of the median value, Z (SD) score or as percentiles.
- Height for age compares child’s height with the expected height or length for a healthy child of the same age.

**Age Independent Criteria**
- **Mid-upper arm circumference (MUAC) or mid-arm circumference (MAC):** Between 1 year and 5 years, the MUAC is relatively constant between 16.5 cm and 17.5 cm. Any child in this group whose MUAC is less than 12.5 cm is classified as undernourished. MUAC is a useful method of screening large number of children during nutritional emergencies but is less useful in long-term growth monitoring programs.
- **Weight for height (WFH):** The degree of wasting is assessed by comparing the child’s weight with the expected weight of a healthy child of same height. Combinations of these measurements have been suggested sometimes to distinguish types of malnutrition. For example, Waterlow proposed that weight or height allows one to distinguish between children who have suffered malnutrition in the past from those who are currently experiencing malnutrition. In chronic malnutrition, the child is stunted, i.e. her or his weight for age and height for age are low. In acute malnutrition, however, her or his height for age is appropriate, but she or he is wasted (low weight for height and age). Thus, weight and height measurements together are useful to understand the dynamics of malnutrition, distinguishing between current malnutrition and long-term or chronic malnutrition.
- **Quack stick:** Quacker’s midarm circumference measuring stick is a height measuring rod calibrated in MUAC rather than height, values of 80% of expected MUAC for height are marked on the stick at corresponding heights levels and the child is made to stand in front of this stick.
Table 4.3.1: Classification of malnutrition

<table>
<thead>
<tr>
<th>Classification</th>
<th>Definition</th>
<th>Grading</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gomez</td>
<td>Weight below percentage of median value WFA</td>
<td>Mild (Grade 1) 75–90% WFA</td>
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<tr>
<td></td>
<td></td>
<td>Moderate (Grade 2) 60–74% WFA</td>
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<tr>
<td></td>
<td></td>
<td>Severe (Grade 3) &lt;60% WFA</td>
</tr>
<tr>
<td>Waterlow</td>
<td>Z-scores (SD) below median WFH</td>
<td>Mild 80–90% WFH</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate 70–80% WFH</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe &lt;70% WFH</td>
</tr>
<tr>
<td>WHO (wasting)</td>
<td>Z-scores (SD) below median WFH</td>
<td>Moderate Z-score &lt; –2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Z-score &lt; –3</td>
</tr>
<tr>
<td>WHO (stunting)</td>
<td>Z-scores (SD) below median WFA</td>
<td>Moderate Z-score &lt; –2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe Z-score &lt; –3</td>
</tr>
<tr>
<td>Kanawati</td>
<td>MUAC divided by occipitofrontal head circumference</td>
<td>Mild &lt;0.31</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moderate &lt;0.28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe &lt;0.25</td>
</tr>
<tr>
<td>Cole</td>
<td>Z-scores of BMI for age</td>
<td>Grade 1 BMI for age Z-score &lt; –1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grade 2 BMI for age Z-score &lt; –2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grade 3 BMI for age Z-score &lt; –3</td>
</tr>
<tr>
<td>Jelliffe</td>
<td>Percentage of standard weight for age (50th centile of Harvard standard)</td>
<td>Normal &gt;90%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grade 1 80–90%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grade 2 70–79%</td>
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<tr>
<td></td>
<td></td>
<td>Grade 3 60–69%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grade 4 &lt;60%</td>
</tr>
<tr>
<td>Arnold</td>
<td>Midarm circumference</td>
<td>Normal &gt;13.5 cm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mild-moderate 12.5–13.4 cm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe PEM &lt;12.5 cm</td>
</tr>
<tr>
<td>Welcome</td>
<td>Presence or absence of edema</td>
<td>&lt;80% Edema absent (underweight)</td>
</tr>
<tr>
<td></td>
<td>Weight or age percentage of expected</td>
<td>60–80% Edema present (Kwashiorkor)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt;60% Edema absent (Marasmus)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt;60% Edema present (Marasmic Kwashiorkor)</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index; HFA, height for age; MUAC, mid and upper arm circumference; SD, standard deviation; WFA, weight for age; WFH, weight for height; WHO, World Health Organization.

His nutritional status is easily read as 50%, 60%, 70% or 80% of the standard. If a child is taller than his circumference level on the stick, he is considered malnourished (Fig. 4.3.5).
- **Midarm circumference to head circumference ratio**: A ratio of 0.280–0.314 indicates mild malnutrition, 0.250–0.279 indicates moderate PEM and less than 0.249 indicates severe PEM.
- **Midarm/height ratio**: Less than 0.29 indicates gross malnutrition (normal 0.32–0.33).
- **Chest/head circumference ratio**: Chest circumference becomes equal to head circumference at 1 year, and after 2 years it becomes more than head circumference. In PEM, it is still smaller than head circumference beyond 2 years of age.

Table 4.3.2: Diagnostic criteria for severe acute malnutrition (SAM) in children of age 6–60 months

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Measure</th>
<th>Cut-off</th>
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</thead>
<tbody>
<tr>
<td>Severe wasting</td>
<td>Weight for height</td>
<td>&lt; –3 SD</td>
</tr>
<tr>
<td>Severe stunting</td>
<td>Height or age</td>
<td>&lt; –3 SD</td>
</tr>
<tr>
<td>Severe under-nutrition</td>
<td>Weight or age</td>
<td>&lt; –3 SD</td>
</tr>
<tr>
<td>Severe wasting</td>
<td>Midarm circumference</td>
<td>&lt; 115 mm</td>
</tr>
<tr>
<td>Bilateral edema</td>
<td>Clinical signs</td>
<td></td>
</tr>
</tbody>
</table>

PEM, it is still smaller than head circumference beyond 2 years of age.
• **Skinfold thickness**: It is an indicator of availability of caloric stores in the form of subcutaneous fat. Sites for measurements are usually the triceps and subscapular region. Its normal value is present in Tanner’s chart and measurements below 90% of the standard are considered subnormal (80–90% mild, 60–80% moderate and less than 60% severe malnutrition).

• **Mid-thigh and calf circumference**: Standards for mid-thigh and calf circumference have been developed.

**THE GROWTH CHART**

While the reliability of a single anthropometric measurement may be suspect and difficult to interpret in terms of a child’s...
past growth and cannot give predictive value of future growth, measurements at regular intervals and recording on a growth chart permit systematic assessment of child’s growth. The idea of monitoring the growth of the individual which would be useful in provision of child health care gave rise to the concept of “growth chart” pioneered by Morley in 1959. Since then various growth charts have been used (Figs 4.3.6A and B).

ECOLOGY AND ETIOLOGY OF MALNUTRITION

Protein energy malnutrition is the result of a complex interplay of interacting and related factors in the individual, family and community. Inadequate dietary intake and diseases are immediate determinants of PEM. Disease may affect PEM by various mechanisms. Conversely, PEM may increase susceptibility to and severity of infections. The causes in individual are anorexia, increased losses from intestine, malabsorption and micronutrient deficiency disease, infectious diseases, inadequate intake of breast milk, early weaning from breast, late introduction of complementary feeding and inadequate access to food. The familial causes are maternal illiteracy, poor knowledge and practices of child rearing, maternal malnutrition, overcrowding, poverty, poor living and sanitary conditions, unemployment, alcoholism or debt. The community causes include national poverty, poor educational

Figure 4.3.6A
status, inadequate medical facilities, poor access to health services, cultural practices and beliefs, marginalizing of girls and women, natural and man-made disasters, poor rainfall or excess rain, or poor facilities for storage and transport, and hoarding and black marketing. The community and national causes have direct impact on the family and individual child. Thus, PEM is an end result of many ecological problems (Flow chart 4.3.1, Figs 4.3.7 to 4.3.10).

The UNICEF has suggested a conceptual framework of the causes of malnutrition (positive and negative) and three main causes: (1) basic, (2) underlying and (3) immediate. While the basic causes remain the same in either group, the lack or deficiency of resources start appearing in the underlying and immediate causes finally manifesting as malnutrition and death (Flow chart 4.3.2) in the negative conceptual frame work.

Although malnutrition is associated with lack of food and poverty, it is also seen in economically advantaged families probably because of lack of awareness in the mothers about proper infant feeding and child nutrition. In the developed
nations like USA, PEM has been reported in families who use unusual and inadequate foods to feed infants, whom the parents believe to be at risk for milk allergies, and also in families who believe in fad diets. In addition, PEM has been noted in chronically ill patients in neonatal or pediatric intensive care units as well as among patients with burns, HIV, cystic fibrosis, failure to thrive, chronic diarrhea syndromes, malignancies, bone marrow transplantation and inborn errors of metabolism.

**Flow chart 4.3.1 Ecology of protein energy malnutrition (PEM)**

**Figure 4.3.7 Socioeconomical and biological determinants of protein energy malnutrition (PEM)**

**Figure 4.3.8 Mortality related to bottle feeding**

**PATHOPHYSIOLOGY OF PROTEIN ENERGY MALNUTRITION**

Many of the manifestations of PEM represent adaptive responses to inadequate energy and/or protein intakes, resulting in decreased activity and energy expenditure. To meet the energy requirement, initially fat stores are mobilized followed by protein catabolism for maintaining basal metabolism. Furthermore, micronutrients are essential
in many metabolic functions as components, cofactors in enzymatic processes and immune response. In the etiopathogenesis of PEM, why and what is it that among children destined to become malnourished; some develop kwashiorkor while others develop marasmus. Amongst various theories postulated was Gopalan’s theory on adaptation/dysadaptation, Srikantia’s on antidiuretic effect of ferritin, loss of edema without change in serum albumin, noxious insults producing reactive oxidative free radicals, decreased Na, K, ATPase activity, depressed cellular protein synthesis, etc. The latest theory postulated by Golden suggests deficiency of type I (functional nutrients), like zinc, and type II nutrients, like phosphorus, magnesium, manganese, copper and vitamins D and C, in the diets of malnourished children due to decrease in appetite. No amount of additional energy as lipids or carbohydrates would enhance convalescence of PEM, unless these specific nutrients are supplied in the balanced form.

**CLINICAL MANIFESTATIONS**

The clinical manifestations of malnutrition depend on the severity and duration of nutritional deprivation, the age of the undernourished subject, relative lack of different proximate principles of food and micronutrients and the associated infection. Nutritional marasmus and kwashiorkor are two different extreme forms of a continuous process of malnutrition. Nutritional marasmus results from predominant energy deficiency whereas kwashiorkor is due to predominant protein deficiency though some energy deficiency may coexist.

Occasionally, patients who are initially marasmic may develop edema due to protein loss when the individual is known as marasmic kwashiorkor. In clinical practice, such extremes account only for a small proportion of cases of malnutrition. A majority has mild to moderate deficiency with varied clinical manifestations, and this range is known as PEM. Malnutrition can be compared to an iceberg, while only the tips of the iceberg, i.e. the severe forms are seen by the health workers (Fig. 4.3.11). Those hidden in the community constitute a vast majority of children suffering from mild and moderate forms of PEM. They are not brought for any medical attention and are at a high-risk of deterioration and progress to severe forms if uncared for prolonged period. PEM impairs resistance to infection and may present with its varied manifestations. Mild degrees of PEM lead to growth retardation, frank malnutrition if prolonged may cause mental retardation.

Initial response to nutritional deprivation are of two types: (1) dynamic children, who remain active but fail to gain weight and later length, and (2) sedentary children, who maintain their growth initially by limiting their activities but ultimately fail to grow. Two-thirds of malnourished children do not present with any clinical signs, and are diagnosed by anthropometry.

**Marasmus**

Marasmus can develop in the first few months of life, commonly from birth to 2 years. It results if the baby is fed with diluted milk from buffalo, cow, goat or even tin milk without offering breast milk or any other food. Marasmus is characterized by failure to gain weight and irritability, followed by weight loss
The “iceberg” of malnutrition

The dangerous 9/10th are hidden

The “Iceberg” of malnutrition has only 1/10th visible

Flow chart 4.3.2 The United Nations Children’s Fund conceptual framework

Positive conceptual framework

Child survival, development and protection

Adequate maternal and child care, household food security, sufficient services and healthy environment, education and information

Adequate dietary intake, psychosocial healthcare

Resources and controls: human, economic and organizational

Political and ideological superstructure

Economic structure

Existing and potential resources

Immediate causes

Negative conceptual framework

Malnutrition and death

Inadequate dietary intake, psychosocial stress, trauma diseases

Inadequate maternal and child care, poor household food security, insufficient and unhealthy environment, lack of education and information

Resources and controls: human, economic and organizational

Political and ideological superstructure

Economic structure

Existing and potential resources

Underlying causes

Basic causes

and listlessness until emaciation results. It is diagnosed by gross loss of subcutaneous fat and the infant seems to have only skin and bones, ribs become visible and costochondral junctions look prominent. There is conspicuous absence of edema. Growth retardation is severe and obvious. The head appears disproportionately large with very little hair. Weight is less than 60% of expected weight, muscles may be atrophied leading to hypotonia. The child is conscious, alert but apathetic and in extreme cases, is also disinterested in surroundings and sits listless for long hours. The facial pads of the fat are last to go; when the child looks like a wizened old man. Anemia is moderate and may be associated with vitamin deficiencies, infections and infestations, and electrolyte imbalance. In the
early stages, the child’s appetite is good and he readily accepts what is offered. However, in advanced stages, there is loss of appetite and it requires a lot of tact and patience to coax the child to eat. Infants are often constipated but may have starvation diarrhea with frequent small mucoid stools. The abdomen may be distended or flat with the intestinal pattern readily visible. As the condition progresses, the temperature usually becomes subnormal and pulse slows (Fig. 4.3.12).

**Kwashiorkor**

Kwashiorkor is an African word was suggested by Cicely Williams in early 1930’s, meaning “the disease that occurs when the child is displaced from the breast by another child”. The age incidence is later than that of marasmus and this condition is uncommon under the age of 1 year. Kwashiorkor may initially present with vague manifestations that include lethargy, apathy and/or irritability. When advanced, there is lack of growth, lack of stamina, loss of muscle tissue, increased susceptibility to infections, vomiting, diarrhea, anorexia, flabby subcutaneous tissues and edema. The edema usually develops early and may mask the failure to gain weight. It is often present in internal organs before it is recognized in the face and limbs.

Edema is characteristically pitting. It usually occurs first around the eyes, then above the ankles and above dorsum of the feet. In the later stages, the whole face, hands and body may be edematous, but ascites is rarely due to kwashiorkor alone. Edema is mainly due to tissue wasting, together with low plasma osmotic pressure caused by low serum albumin levels. The child is listless, lethargic, apathetic and miserable, her/his moaning cry is characteristic. The hair changes are variable. Hair may be thin, dry, brittle and lusterless. These become straight and hypopigmented (grayish-white or reddish-brown). During recovery, the growing part of the hair gets approximately pigmented, and gives appearance of “flag”. The skin changes are not constant and manifestations are known as dermatosis (Figs 4.3.13A and B). Skin becomes darkened in irritated areas but in contrast to pellagra, it does not occur in areas exposed to sunlight. Depigmentation may occur after desquamation in these areas, or it may be generalized. The skin lesions appear as large areas of erythema, followed by hyperkeratosis. The epidermis peels off in large scales, exposing a raw area underneath which is prone to infection. It resembles old paint flaking off the surface of the wood hence called “flaky paint dermatosis”. The lesions are moist and common on areas exposed to continuous pressure and irritation. In severe cases, petechiae or ecchymoses may appear. Alternate areas of hypopigmentation and hyperpigmentation give a resemblance to pavement, and this is known as “pavement dermatosis” or when skin changes are seen in a particular mosaic form, as “mosaic dermatosis”. Liver may be enlarged and fatty. There may be associated infections in the form of diarrhea, respiratory infections, urinary tract infections and vitamin deficiencies, especially vitamin A, thiamine, riboflavin and niacin. Eventually, there is stupor, coma and death.
Marasmic Kwashiorkor

Children with severe muscle and fat wasting, but with presence of edema are called marasmic kwashiorkor. This syndrome is seen in children who have marasmus, but suddenly develop edema due to increased deficiency of protein than before. Thus, the clinical features are those of both marasmus and kwashiorkor (Fig. 4.3.14). Anemia may be moderate and one or more vitamin deficiencies may be evident. The usual differentiating features of marasmus and kwashiorkor are summarized in Table 4.3.3.

Overnutrition and Obesity

Once considered a problem of high income countries, over-weighted obesity are now on the rise in low and middle income countries, particularly in urban settings. In developing countries with emerging economics, the rate of increase of childhood overweight and obesity has been more than 30% higher than that of developed countries and worldwide obesity has more than doubled since 1980. Most of the world population lives in countries where overweight and obesity kills more people than underweight. In 2014, more than 1.9 billion adults (18 years and older) were overweight, of these over 600 million were obese. In 2013, 42 million children under age of 5 or 1 year per every ten children were overweight or obese, i.e. an approximate rate of 4%.

There are problems unique to India. The combination of people living in poverty and the recent rapid economic globalization has led to rapid changes in diets and lifestyle resulting in coemergence of two types of malnutrition, i.e. (1) undernutrition and (2) overnutrition. Although underweight and malnutrition are rampant in poor families, urbanization with increased consumption of food with higher fat and sugar content of ‘fast food’, along with sedentary lifestyles, i.e. TV, computers, lack of space for outdoor games and exercise with

<table>
<thead>
<tr>
<th>Table 4.3.3: Differences between marasmus and kwashiorkor</th>
</tr>
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<tbody>
<tr>
<td>Marasmus</td>
</tr>
<tr>
<td>A. Usual age</td>
</tr>
<tr>
<td>B. Essential features</td>
</tr>
<tr>
<td>1. Edema</td>
</tr>
<tr>
<td>2. Wasting</td>
</tr>
<tr>
<td>3. Muscle wasting</td>
</tr>
<tr>
<td>4. Growth retardation</td>
</tr>
<tr>
<td>5. Mental changes</td>
</tr>
<tr>
<td>C. Variable features</td>
</tr>
<tr>
<td>1. Appetite</td>
</tr>
<tr>
<td>2. Diarrhea</td>
</tr>
<tr>
<td>3. Skin changes</td>
</tr>
<tr>
<td>4. Hair changes</td>
</tr>
<tr>
<td>D. Biochemistry/pathology</td>
</tr>
<tr>
<td>1. Serum albumin</td>
</tr>
<tr>
<td>2. Urinary urea per g creatinine</td>
</tr>
<tr>
<td>3. Urinary hydroxyproline per g creatinine</td>
</tr>
<tr>
<td>4. Serum essential amino acid index</td>
</tr>
<tr>
<td>5. Anemia</td>
</tr>
<tr>
<td>6. Liver biopsy</td>
</tr>
</tbody>
</table>

*These are the most characteristic or useful distinguishing features.
improved technology and transport (cars, trains, etc.) has unknowingly increased overweight and obesity especially in well to do families. However, micronutrient deficiency is present in both of them, i.e. in undernutrition and in overnutrition. In general, the average Indian diet remains largely deficient in green leaf vegetable, milk and milk products and thus micronutrient such as vitamin A, iron and iodine remain deficient in all children with improper diet. In adolescents (14–18 years), the overall prevalence of overweight and obesity was 24.2% and 11% respectively in 2009, Punjab, Kerala and Delhi have highest rate of overweight and obesity.

Overweight and obesity are defined by World Bank as age and gender specific. BMI greater than or equal to 25 is overweight and BMI greater than or equal to 30 is obesity, or BMI greater than or equal to 85–95% is overweight and BMI greater than or equal to 95% is obesity. As already mentioned the main cause for obesity is imbalance between energy consumption and energy expenditure with higher practice of energy consumption.

Overeating, overweight and obesity lead to many diseases such as early onset diabetes, heart disease and stroke, musculoskeletal disorders especially osteoarthritis and some cancer like endometrial, breast and colon. Childhood obesity is associated with a higher chance of obesity, disability and premature death. In addition to future risks, obese children experienced breathing difficulties, increased risk of fractures, hypertension and early markers of cardiovascular disease, insulin resistance and psychological effects. The treatment of overweight and obesity in children and adolescents requires a multidisciplinary, multiphase approach. Overall obesity is a preventable disorder.

Multiple Nutritional Deficiencies

The nutritional deficiencies are generally multiple and anemia due to deficiencies of iron, vitamin B12, or folate may be associated. Anemia may be hypochromic microcytic, macrocytic, or normocytic normochromic. Deficiencies of vitamin B-complex factors, especially arboflavinosis, vitamin A (manifesting as keratomalacia and xerophthalmia) and vitamin C occur commonly. Since growth gets arrested in severe PEM, rickets may become manifest only when the child starts growing with nutritional rehabilitation.

Electrolyte Imbalances

Potassium

The total body potassium may be markedly decreased in PEM. The loss is due partly to the cellular breakdown but more so by the loss in diarrheal stools.

Other Electrolytes

There is also deficit of total body sodium, calcium, phosphorus, magnesium and chloride. The body sodium is 93% of the expected values. There is significant loss of magnesium from the cells, levels being significantly low in children with moderate and severe malnutrition, and in children with marked linear growth retardation.

Endocrine Changes

Growth hormone, plasma cortisol levels, thyroid stimulating hormone (TSH) and T4 levels may be raised in PEM, while insulin levels and T3 levels are reduced both in marasmus and kwashiorkor.

Infection and Immunity

Infectious disease worsens when malnutrition is present and conversely malnutrition usually weakens resistance to various infections which are more serious in a malnourished host than in a well-nourished child. Malnourished children with PEM have recurrent episodes of acute infections or chronic insidious infections which may go undetected unless carefully looked for. Thus, recurrent diarrheal diseases, lower respiratory tract infections and occult urinary tract infection are common, and have high mortality. Measles is usually a preceding illness. Tuberculosis and malaria must be always ruled out, and intestinal parasitosis like ascariasis, hookworm, and giardiasis, must be treated. Septicemia, especially in infants and toddlers, may be life threatening.

Regarding humoral immunity IgG, IgM and secretory IgA, blood concentrations are not significantly affected in mild and moderate forms of PEM and show a good response when challenged with bacterial and viral vaccines, but is depressed in severe forms of PEM with infections. The cell-mediated immunity (CMI) is impaired in all grades of malnutrition except in Grade I. It is severely impaired in grades III and IV, PEM and kwashiorkor. This explains a high incidence of Gram-negative bacterial infections and serious morbidity and high mortality to viral infection like herpes simplex and measles. Due to depressed CMI, the tuberculin skin test is often negative in marasmus and kwashiorkor in spite of active tuberculosis. Following dietary treatment of 4–6 weeks, the CMI might improve and the skin test may become positive. Serum C-reactive protein and C3 complement levels are depressed in severe malnutrition but rise in presence of infections and thus behave as acute phase reactants.

Complications

The complications of PEM are usually seen with severe malnutrition. They are dehydration, hypothermia, hypoglycemia, infections, anemia, xerophthalmia, congestive heart failure, hypomagnesemia, hypocalcemia, zinc, copper, chromium and manganese deficiency and deficiencies of vitamins.

Long-Term Consequences

Malnourished children are more susceptible to disease, have a reduced capacity to learn, have deficits in cognitive function, less likely to perform well in school and are likely
to drop out. The evidence suggests that undernutrition has pervasive effects on immediate health and survival as well as on subsequent performance. These include not only acute effects on morbidity and mortality but also long-term effects on cognitive and social development, physical work capacity, productivity and economic growth. The magnitude of both the acute and the long-term effects is considerable. Survivors of undernutrition have deficits in height and weight that persist beyond adolescence into adulthood. These may be accompanied by deficit in frame size as well as muscle circumference and strength. The implications of these deficits with respect to the work capacity of both men and women and to women’s reproductive performance are obvious. Once in the job market, their productivity is low. For the economy as a whole, this translates into losses of nearly 3% of gross domestic product (GDP). All this places India’s large population, the basis of its much awaited demographic dividend, at a growing disadvantage in today’s globalizing world. These deficits are related to the severity of PEM and can be decreased probably by a combination of dietary and behavioral interventions, coupled with improvements to the overall quality of home and/or school environment. Such interventions appear to be much more effective if instituted in early life.

**MANAGEMENT**

The management of PEM depends on nutritional status, degree of hypermetabolism, expected duration of illness and associated complications. The goals are to minimize weight loss, to maintain body mass and to encourage body mass repletion or growth. The principles of management are as follows:

- The patient is evaluated for the severity, presence of systemic infections, other nutritional, micronutrients deficits, anemia and fluid and electrolyte disturbances.
- The intake of food is promoted by all available means. Locally available, culturally acceptable, and affordable foods are advised.
- Complications of malnutrition and sequelae are prevented by careful surveillance and prompt remedial action.
- Possible epidemiological factors for malnutrition are considered and attempt is made to eliminate these as far as possible.

Mild-to-moderate PEM is best managed at home. Majority of cases of severe PEM are associated with some of the complications listed above and hence are best managed in hospital.

**Domiciliary or Community Management**

This is recommended for mild-to-moderate PEM, and those uncomplicated severe PEM who have fairly good appetite, normal body temperature, who are conscious and active, and without evidence of serious infection. These children are managed at home by parents under observation and supervision. They are monitored through weekly visits by paramedicals or visits to the hospital or at a nutritional rehabilitation center every week. The main goal of treatment is to provide adequate calories to replace losses, to build up nutrition, and to promote growth. Caution must be taken to gradually build up the calories and proteins. The expected calories and proteins are calculated on the present weight. Once this is achieved, then over next 7 days, calories and proteins are calculated of the average weight. After that, over 2 weeks the diet (calories and proteins) are increased for the expected weight for that age. It takes about 6 months to achieve this target.

The examples are cereal pulse combinations—double or triple mixes, like *dal rice, khichdi*, with seasonal green leafy and yellow orange vegetables, root vegetables with sugar, jaggery; thick butter milk based diets, milk based diets, either of them supplemented with proteins like ground nut, soya, and amylase-based food formulations. Emphasis must be laid on adding enough oil/ghee/butter to the diet to increase calories and palatability. The energy recommended is 80–100 kcal/kg/day and protein 0.70–1.0 g/kg/day, stepped up gradually to 120–150 kcal/kg/day and protein 2–3 g/kg/day of high biological value.

Basically, these should be from locally available, seasonal and affordable food sources, commonly consumed by the family. The diet should be liquid, semisolid or solid depending on the child’s acceptability and appetite. Frequent small feeds are encouraged, increased gradually rather than one or two major bulky meals. Non-vegetarian articles like egg, fish, chicken, meat, etc. are recommended for those whose cultural, religious practices permit them. Zinc is added when child improves weight. Parents are educated about proper cooking, clean drinking water, sanitation and personal hygiene. Some basic advice is also given for management of common problems like diarrhea by oral rehydration solution (ORS), of anemia with oral iron and folic acid, vitamin deficiencies, infestations and infection as well as immunization.

**Severe Malnutrition**

The management of uncomplicated SAM using simple, ready to use therapeutic foods—*ready-to-use therapeutic food* (RUTF), (fortified with all essential nutrients like zinc, potassium, magnesium, and phosphate with low levels of sodium, protein and iron), with community based care is encouraging and worthwhile. With these diets children recover their appetite. Home based management has the advantage of easier access by rural population, promoting early intervention in the disease, improving coverage rates and preventing nosocomial infections. The limited hospital staff can focus on inpatient complicated cases.

The risk of death rises progressively with worsening nutritional status. However, over 80% malnutrition deaths occur in mild to moderately malnourished children as these greatly outnumber children with severe malnutrition.
Hence, for better child survival, intervention is necessary for management of mild and moderately malnourished children in addition to that of severely malnourished children. Hence, there is an urgent need to identify these malnourished children timely and plan the treatment based on the need of an individual child.

Case fatality rates in children with severe malnutrition have remained unchanged at 20–30% over the past five decades. Infections, including diarrheal dehydration and electrolyte disturbances are common in severely malnourished children and found to be the poor prognostic factors. According to the WHO, a death rate of more than 20% is considered unacceptable in the management of severely malnourished children, 11–20% is poor, 5–10% is moderate, 1–4% is good and less than 1% is excellent. Appropriate feeding, micronutrient supplementation, broad-spectrum antibiotic therapy, less use of intravenous fluids for rehydration, and careful management of complications are factors that can reduce death, morbidity and cost of treating these children. On the basis of these factors, WHO have prepared guidelines for the inpatient case management of severe malnutrition. Not all severely malnourished children need hospitalization (Table 4.3.4).

### Table 4.3.4: Indications for hospitalization in severe malnutrition

- Hypothermia
- Infection
- Fluid and electrolyte imbalance
- Convulsions
- Unconsciousness
- Jaundice, purpura
- Raised liver enzymes
- Severe anemia and congestive cardiac failure
- Xerophthalmia
- Severe dermatosis
- Extreme weight deficit
- Bleeding
- Marked hepatomegaly
- Persistent vomiting
- Severe anorexia
- Distended tender abdomen
- Age less than 1 year

Majority of children usually have some complications, they need hospitalization for critical care and intense monitoring. The management of SAM can be achieved by three ways and in three phases (Fig. 4.3.15):

- **Three ways:**
  1. Traditional nutrition therapy (community based management of SAM).
  2. Hospital based therapy using F-75 and F-100 diets.
  3. Initial stabilization in hospital using F-75 diet and rehabilitation at home using RUTF.

- **Three phases:**
  1. **Phase 1:** Phase of resuscitation. The initial or acute phase (0–7 days) when the child is being treated for complications, dietary therapy is started simultaneously.
  2. **Phase 2:** Phase of restoration or recovery (1–2 weeks) when the child will increase dietary intake and gain weight.
  3. **Phase 3:** Phase of rehabilitation and follow-up (2–26 weeks) which may be after discharge.

### General principles for routine care

<table>
<thead>
<tr>
<th>Rehabilitation steps</th>
<th>Stabilization Day 1–2</th>
<th>Phase Day 2–7+</th>
<th>Week 2–6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Hypoglycemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Hypothermia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Dehydration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Electrolytes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Micronutrients</td>
<td></td>
<td>No iron—With iron</td>
<td></td>
</tr>
<tr>
<td>7. Cautious feeding</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Rebuild tissues</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Sensory stimulation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Prepare follow-up</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Figure 4.3.15 Stepwise management of severe malnutrition**

**Source:** The World Health Organization

**Phase 1: Resuscitation or Stabilization and Treatment of Complications**

**Dehydration:** Cautious management to avoid over hydration.

- Severe/shock: Intravenous Ringer lactate 20–30 mL/kg in 1 hour followed by 70 mL/kg over next 2 hours, followed by 0.45% glucose saline/Isolyte-P as maintenance fluid. Total fluid and sodium not to exceed 75% of allowance.
- Mild: Oral rehydration solution—hypo-osmolar/preferably ReSoMal 5 mL/kg every 30 minutes—orally/by nasogastric tube (ReSoMal: 45 mmol sodium, 40 mmol potassium and 3 mmol magnesium).

**Electrolyte imbalance:**

- Potassium: 2–5 mmol/kg/day.
- Magnesium: 0.3–0.6 mmol/kg/day or 50% MgSO₄ intramuscularly 0.3 mL/kg (2 mL maximum) OD.
- Sodium: Restrict salt, no diuretics.

**Hypoglycemia:**

- 10% glucose 1–2 mL/kg intravenously bolus followed by 10% dextrose in N/5 saline as maintenance for 24 hours. If 10% intravenous glucose not available, give 10% sucrose (one full teaspoonful sugar in 3.5 tablespoonful of water) orally/by nasogastric tube then every 30 minutes for 2 hours.
- Early and frequent feeding.
Hypothermia: Warm bed and room, keep the baby with mother, double clothing, and cover head and feet. Treat hypoglycemia and sepsis. Start feeds early.

Septicemia: Intravenous ampicillin 50 mg/kg, 6 hourly for 2 days, gentamicin 7.5 mg/kg intramuscularly/intravenously. If no improvement, add chloramphenicol/cephalosporin. If anorexia persists, continue antibiotics for 10 days. Give metronidazole 7.5 mg/kg 8 hourly for 7 days for potential anaerobic infections.

Congestive heart failure: This mostly occurs due to fluid overload (e.g., overuse of intravenous fluids, unmonitored blood transfusion) or due to severe anemia. When congestive heart failure is due to fluid overload, administer frusemide 1–2 mg/kg, and reduce/stop fluid infusion. Avoid digitalis. Diuretics should never be used to correct edema in case of edematous malnutrition.

Anemia: If hemoglobin is less than 5 g/dL, give packed cells transfusion 5–10 mL/kg. Iron should be started only after resolution of infection (2–3 weeks).

Micronutrients:
- Iron: Oral ferrous sulfate or fumarate syrup 4 mg (elemental)/kg
- Calcium gluconate-IV: 1–2 mL/kg or oral calcium lactate powder—3 g/day
- Zinc: 2 mg/kg/day
- Copper: 20 µg/kg/day
- Chromium: 0.2 µg/kg/day
- Manganese: 10 µg/kg/day
- Vitamin A: 100,000 IU for age less than 1 year and 50,000 IU for age less than 6 months
- Vitamin D: Rickets—oral vitamin D
- Other vitamins: B-complex, vitamin K (5 mg weekly).

Phase II: Restoration

After the initial phase of resuscitation, there is improvement in child’s condition with return of appetite, beginning of loss of edema and return of smile. During this phase the aim is to make the child gain weight and restore weight for height.

Dietary management of severe PEM: In the initial stabilization phase, because of child’s fragile physiological and metabolic state, great caution is required in dietary intervention of SAM children. The rule of “go slow, rather than hurry” is appropriate. Feeding is designed to provide 75–80 cal/kg/day and proteins 0.7 g/kg/day. Each feed should be small, gradually increased, of low osmolality and lactose offered at frequent intervals, according to the child’s tolerance (Table 4.3.5). Breastfeeding should be continued and Starter formulas like F-75 (milk based containing 75 cal/100 mL and 0.9 g/100 mL protein and fluid volume 130 mL/kg) are satisfactory for most children (Table 4.3.6). Very weak children or those with anorexia may be fed with spoon, dropper, or nasogastric tube.

As per the child’s progress and response to the treatment of complications by the end of 1 week, the calories and proteins may be stepped up to 100 cal/kg and proteins 1–1.5 g/kg/day. Minerals and vitamins are also added. In the phase of restoration, the principle is to increase weight and catch up growth as the child’s appetite has regained.

---

**Table 4.3.5: Feeding schedule**

<table>
<thead>
<tr>
<th>Days</th>
<th>Frequency</th>
<th>Volume/kg/feed</th>
<th>Volume/kg/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–2</td>
<td>2 hourly</td>
<td>11 mL</td>
<td>130 mL</td>
</tr>
<tr>
<td>3–5</td>
<td>3 hourly</td>
<td>16 mL</td>
<td>130 mL</td>
</tr>
<tr>
<td>6–7+</td>
<td>4 hourly</td>
<td>22 mL</td>
<td>130 mL</td>
</tr>
</tbody>
</table>

---

**Table 4.3.6: Composition of F-75 starter formula**

<table>
<thead>
<tr>
<th>Type of milk</th>
<th>Milk (g)</th>
<th>Eggs (g)</th>
<th>Sugar (g)</th>
<th>Oil (g)</th>
<th>Cereal powder (g)*</th>
<th>CMV** (red scoop = 6 g)</th>
<th>Water (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry skim milk</td>
<td>25</td>
<td>0</td>
<td>70</td>
<td>27</td>
<td>35</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Dry whole milk</td>
<td>35</td>
<td>0</td>
<td>70</td>
<td>20</td>
<td>35</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Fresh cow milk</td>
<td>280</td>
<td>0</td>
<td>65</td>
<td>20</td>
<td>35</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Fresh goat milk</td>
<td>280</td>
<td>0</td>
<td>65</td>
<td>20</td>
<td>40</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Whole eggs</td>
<td>0</td>
<td>80</td>
<td>70</td>
<td>20</td>
<td>40</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Egg yolks</td>
<td>0</td>
<td>50</td>
<td>70</td>
<td>15</td>
<td>40</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
</tbody>
</table>

*Cereal powder should be cooked for around 10 minutes and then the other ingredients be added.

**CMV special mineral and vitamin mix adapted to severe acute malnutrition treatment.

Abbreviation: CMV, combined mineral vitamin mix.
Table 4.3.7: Composition of F-100 catch-up formula

<table>
<thead>
<tr>
<th>Type of milk</th>
<th>Milk (g)</th>
<th>Eggs (g)</th>
<th>Sugar (g)</th>
<th>Oil (g)</th>
<th>CMV**</th>
<th>Water (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry skim milk</td>
<td>80</td>
<td>0</td>
<td>50</td>
<td>60</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Dry whole milk</td>
<td>110</td>
<td>0</td>
<td>50</td>
<td>30</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Fresh cow milk</td>
<td>900</td>
<td>0</td>
<td>50</td>
<td>25</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Fresh goat milk</td>
<td>900</td>
<td>0</td>
<td>50</td>
<td>25</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Whole eggs</td>
<td>0</td>
<td>220</td>
<td>90</td>
<td>35</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
<tr>
<td>Egg yolks</td>
<td>0</td>
<td>170</td>
<td>90</td>
<td>10</td>
<td>2</td>
<td>Up to 1,000</td>
</tr>
</tbody>
</table>

**CMV special mineral mix adapted to severe acute malnutrition.
Abbreviation: CMV, combined mineral vitamin mix.

Table 4.3.8: Composition of peanut butter based RUTF

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>Contents (% by weight)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full fat milk powder</td>
<td>30</td>
</tr>
<tr>
<td>Sugar</td>
<td>28</td>
</tr>
<tr>
<td>Vegetable oil</td>
<td>15</td>
</tr>
<tr>
<td>Peanut butter</td>
<td>25</td>
</tr>
<tr>
<td>Fortified with micronutrients</td>
<td>1.6</td>
</tr>
<tr>
<td>(sodium, potassium, calcium, phosphorous,</td>
<td></td>
</tr>
<tr>
<td>magnesium, iron, zinc, copper, selenium,</td>
<td></td>
</tr>
<tr>
<td>iodine, vitamin A, vitamin D, vitamin E,</td>
<td></td>
</tr>
<tr>
<td>vitamin K, vitamin B₁, vitamin B₂, vitamin B₃, vitamin B₁₂, vitamin C, folic acid, niacin, pantothenic acid, biotin)</td>
<td></td>
</tr>
<tr>
<td>Energy</td>
<td>520–550 kcal/100 g</td>
</tr>
<tr>
<td>Proteins</td>
<td>10–12% of total energy</td>
</tr>
<tr>
<td>Fat</td>
<td>45–60% of total energy</td>
</tr>
</tbody>
</table>

The calories and proteins (preferably 50% should have high biological value, e.g. milk, chicken, meat or egg) are stepped up gradually, approximately from 25 cal/kg/day on every other day, to 100–120 cal/kg/day and proteins 1–2 g/kg/day based on patient’s changing weight.

F-100 formulas (skimmed milk/fresh egg based) are used in inpatients only (Table 4.3.7). Diets/RUTF based on cereal pulse, (rice, moong dal) combinations, fortified with oil, jaggery and seasonal vegetables, or buttermilk based diets added with whey soy/casein, fine roasted powdered groundnut protein and sugar, and oil are recommended and can be prepared and fed as RUTF. A typical recipe RUTF (peanut based) (Table 4.3.8) can also be given. Occasionally, children with secondary lactose intolerance do not tolerate milk based feeds. Depending on the severity, either the amount of milk may be diminished in the diet by replacing with other articles like rice, egg, curds, etc. In severe lactose intolerance, milk will have to be temporarily completely omitted and replaced by cereals, pulses—rice, dal, soya, rice gruel, egg, soy milk, chicken gruel/rice, curds and rice, etc. (Table 4.3.9).

Phase III: High Energy Feeding

By now the child has progressed well with return of appetite, tolerance to high energy and protein feeds. In this phase, emphasis is on intensive feeding to restore lost weight, catch up growth and recover emotionally and physically. The calories are gradually increased from 150 cal/kg/day to 180 cal/kg/day and proteins 1.5–2.5/3 g/kg/day. Milk is gradually withdrawn, semisolids and solids are introduced. Ideally, this phase extends from 6 weeks to 26 weeks to give the child his/her immune system, the best chance to recover before being challenged to home environment if the child is in hospital (if not already discharged) or a nutritional rehabilitation unit.

Phase IV: Transfer to Family Diet or Phase of Rehabilitation

By now as the child is accustomed to semisolid or solid diet. The child and parents are encouraged and taught to make the child share family diet. Additional supplements can be offered as shown in Table 4.3.10. The discharge criteria of
Table 4.3.9: Diet for lactose intolerance (of varying severity)

<table>
<thead>
<tr>
<th>Name</th>
<th>Constituents</th>
<th>Calories</th>
<th>Proteins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low lactose diet</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Dried skimmed milk</td>
<td>Skimmed milk (60 g) Sucrose (12 g)</td>
<td>400</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>Vegetable oil (15 g)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Milk and rice</td>
<td>Milk (75 mL) Rice (5 g) Sugar (25 g)</td>
<td>79</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Water (100 mL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactose free diet</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Rice and egg</td>
<td>Rice (50 g) Glucose (45 g) Egg (one)</td>
<td>710</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Oil (30 g)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Cereal pulse</td>
<td>Rice (50 g) Green gram pulse (25 g)</td>
<td>715</td>
<td>9.2</td>
</tr>
<tr>
<td></td>
<td>Jaggery (50 g)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Oil (25 g)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Soya rice gruel</td>
<td>Rice (25 g) Soybean (25 g) Glucose (50 g)</td>
<td>715</td>
<td>12.5</td>
</tr>
<tr>
<td></td>
<td>Oil (35 g)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Chicken gruel</td>
<td>Chicken (100 g) Glucose (40 g) Oil (50 g)</td>
<td>720</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>Water (1 L)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 4.3.10: Energy-rich foods

<table>
<thead>
<tr>
<th>Name</th>
<th>Ingredients</th>
<th>Calories/100 g</th>
<th>Proteins/100 g</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Besan mix/ladoo Panjiri</td>
<td>Bengal gram flour Wheat flour Jaggery, ghee (1 part of each)</td>
<td>500</td>
<td>9</td>
</tr>
<tr>
<td>2. Sooji (Rawa) Kheer</td>
<td>Toned milk (750 mL) Sugar (100 g) Sooji (25 g) Oil (5 g)</td>
<td>1432</td>
<td>28.4</td>
</tr>
<tr>
<td>3. Hyderabad mix</td>
<td>Whole wheat (40 g) Bengal gram (16 g) Groundnuts (10 g) Jaggery (20 g)</td>
<td>330/86</td>
<td>11.3/86</td>
</tr>
<tr>
<td>4. Shakti Ahar</td>
<td>Roasted peanut (10 g) Roasted wheat (40 g) Roasted gram (20 g) Jaggery (30 g)</td>
<td>390</td>
<td>11.4</td>
</tr>
</tbody>
</table>

A severely malnourished child may vary and are summarized in Table 4.3.11.

During recovery and follow-up, tender loving care should be provided. Provide a cheerful stimulating environment in form of structured play therapy 15–30 minutes/day. Physical activity should be encouraged as soon as possible. Mother should be involved in caring for the baby as far as possible (e.g. comforting, feeding, bathing and play).

Prevention requires a coordinated approach of many disciplines: nutrition, agriculture, food technology, education, health administration, social services, nongovernmental organizations, community and religion. A strong political commitment is must for tackling malnutrition in the country.
Nutrition should be a priority at national and sub-national levels as it is central for human, social and economic development. As the World Bank advocates nutrition needs to be repositioned in national development if the millennium development goals (MDGs) are to be achieved. Achieving the MDG target halving the proportion of underweight children between 1990 and 2015 will involve effort at micro, meso, macro, and global levels (Fig. 4.3.16) as well as partnerships among all sectors of society (Fig. 4.3.17).

**Table 4.3.11: Discharge from hospital**

<table>
<thead>
<tr>
<th>No definite guidelines</th>
<th>Criteria vary from hospital to hospital: Of weight/age • Difficult to attain and associated with high morbidity and mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>General guidelines</td>
<td>Acute problems are over • Appetite has returned • Oral dietary intake adequate • Weight gain has started</td>
</tr>
<tr>
<td>Prolonged hospitalization</td>
<td>Risks of acquiring nosocomial infection • Cost of hospital therapy increases • Inconvenience to family and loss of daily wages</td>
</tr>
<tr>
<td>Premature discharge</td>
<td>Incomplete recovery, morbidity, recurrence of illness, rarely death 10–30%</td>
</tr>
<tr>
<td>Follow-up after discharge</td>
<td>Continued supervision is vital for sustained and complete recovery • Prevent recurrence</td>
</tr>
</tbody>
</table>

*Note: Along with medical and nutritional management providing care and stimulation is vital.*

![Figure 4.3.16 Prevention of protein energy malnutrition (PEM)](image)

![Figure 4.3.17 Action from micro to global level](image)

Abbreviation: MDGs, millennium development goals.

INTRODUCTION

Water-soluble vitamins include all vitamins in B-complex group [thiamine (vitamin B_1), riboflavin (vitamin B_2), niacin (vitamin B_3), pyridoxine (vitamin B_6), cobalamin (vitamin B_12), folate, biotin, pantothenic acid, choline, inositol] and ascorbic acid (vitamin C). These vitamins act as coenzymes in many interrelated metabolic pathways. As these vitamins are water soluble and heat-labile, large amounts are lost from the ingredients, if they are repeatedly washed, and when the cooking water is discarded. These vitamins are not stored for prolonged period in the body; significant toxicities at therapeutic doses are uncommon.

VITAMIN B COMPLEX

Thiamine

Active form of this vitamin acts as a cofactor for enzymes involved in carbohydrate catabolism, nucleic acid synthesis and nerve conduction.

Dietary Sources

Rice, wheat, legumes, fortified flours, fish and meat are good sources of thiamine. Polished rice is depleted of thiamine. It can be retained in rice by steaming the rice in the husk before milling (parboiling).

Deficiency State

Malnutrition, gastrointestinal disorders such as malabsorption, blind loops and short bowel, and chronic debilitation conditions such as malignancies are common causes of deficiency. Fatigue, irritability, poor mental concentration, anorexia and nausea are early manifestations of deficiency. A full-fledged deficiency state (beri beri) manifests as peripheral neuropathy resulting in tingling, paresthesias, leg cramps, hyporeflexia, ataxia and lack of coordination. Cardiac manifestations include cardiomegaly and congestive heart failure. Central nervous system (CNS) involvement occurs late in disease, and is characterized by psychic disturbances, optic atrophy, hoarseness, raised intracranial pressure and coma.
Treatment

Oral administration of thiamine is sufficient in mild-to-moderate deficiency states. Children with cardiac and CNS manifestations should be given 10 mg of thiamine intramuscularly or intravenously daily for the first week, followed by 3-5 mg/day orally for 6-12 weeks. Most children show dramatic improvement to oral or parenteral thiamine.

Riboflavin

Majority of this vitamin in tissues is found in the form of coenzyme flavin adenine dinucleotide (FAD), which participates in oxidation-reduction reactions in numerous metabolic pathways and in energy production via the respiratory chain.

Dietary Sources

This included milk and its products, eggs, fortified cereals and grains and liver. Vegan diets are poor sources.

Deficiency State

Ariboflavinosis is characterized by glossitis, angular cheilosis, keratitis, conjunctivitis, photophobia, corneal vascularization and seborrheic dermatitis. Malnutrition, malabsorption and gastrointestinal infections commonly precipitate the deficiency.

Treatment

Oral administration of riboflavin (3-10 mg/day) as a part of vitamin B complex mix rapidly corrects the deficiency and controls the associated symptoms.

Niacin

Niacin is a component of the coenzymes nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP), which are important for many redox reactions involved in carbohydrate metabolism, fatty acid synthesis and steroid synthesis.

Dietary Sources

Niacin is rapidly absorbed from the stomach or the intestine. Good sources are fish, meat, cereals, legumes, milk and green leafy vegetables. Predominantly maize eating populations suffer from niacin deficiency.

Deficiency State

The classical triad of niacin deficiency (pellagra) is diarrhea, dermatitis and dementia. Certain inborn errors of tryptophan metabolism (carcinoid syndrome, Hartnup’s disease) are also associated with niacin deficiency. Skin lesions of pellagra start as symmetric erythematous areas, resembling sunburn. These lesions are distributed on hands and feet in a “glove and stocking” pattern, and are sometimes present over the neck (Casal necklace). Infants and young children often do not develop classical deficiency signs, and present with irritability, fatigue, anorexia and scaly skin.

Treatment

Oral administration of niacin (50-200 mg/day) as a part of vitamin B complex mix is indicated for pellagra. Severe cases may require intravenous administration. Sun exposure should be avoided during active skin lesions, and soothing agents are applied.

Pyridoxine

Pyridoxine (vitamin B₆) is a component of pyridoxine hydrochloride, pyridoxal and pyridoxamine cofactors in metabolism of carbohydrates, amino acids, steroids and nucleic acids.

Dietary Sources

Poultry, meat, fish, fortified cereals and bananas are good sources of pyridoxine. Drugs inhibiting pyridoxine activity (e.g. isoniazid, oral contraceptives, penicillamine and phenytoin) may precipitate the deficiency.

Deficiency State

Early symptoms or signs are electroencephalography (EEG) abnormalities, irritability and vomiting. Seizures, failure to thrive, skin lesions and microcytic anemia may occur in severe deficiency states.

Treatment

Oral administration of 10-100 mg/day is sufficient for correcting the deficiency and dependency. Intramuscular or intravenous administration of 100 mg pyridoxine is administered in case of seizures. Treatment should be followed by ensuring adequate dietary consumption.

Folate and Vitamin B₁₂

Folate is involved in a variety of reactions involved in amino acid and nucleotide metabolism. Vitamin B₁₂ (cobalamin) is required as a cofactor for methyl group transfer from a folic acid cofactor to form methionine. The unmethylated folate cofactor then participates in single carbon reactions for nucleic acid synthesis. Thus, some B₁₂ and folic deficiency symptoms are similar.
Dietary Sources

Legumes, fortified cereals, citrus fruits and leafy vegetables are good sources of folate. On the other hand, vitamin B₁₂ is found only in foods from animal sources. Organ meats, sea foods, egg yolk, fish and poultry are rich sources of vitamin B₁₂. Vegetarians get their requirements mainly from fortified cereals and milk.

Deficiency State

Megaloblastic anemia results from either (or combined) folate or vitamin B₁₂ deficiency. Maternal folate deficiency increases the risk of neural tube defects in the fetus. Vitamin B₁₂ also may result in neurological manifestations such as irritability, poor attention span, hypotonia, abnormal movements and peripheral neuropathy progressing to subacute combined degeneration. Hyperpigmentation of knuckles is another commonly observed sign of vitamin B₁₂ deficiency.

Treatment

Megaloblastic anemia due to folate deficiency requires oral administration of 0.5–1 mg/day of folic acid until a definite hematologic response has occurred. Intramuscular or intravenous administration of single dose of 1,000 µg of vitamin B₁₂ is adequate for achieving hematological response in vitamin B₁₂ deficiencies. Repeated doses are required for those with severe deficiency, neurological manifestations or malabsorption. A combined treatment is required, if the blood levels for both are not available.

Others

Biotin

Biotin (found in a variety of vegetarian and nonvegetarian foodstuffs) deficiency causes scaly periorificial dermatitis, alopecia, hypotonia and apathy.

Pantothenic Acid

Pantothenic acid (found in seafood, organ meats, egg yolk, legumes and milk) deficiency causes muscle cramps and burning feet syndrome; clinical deficiency is extremely rare.

Choline and Inositol

Though important for normal body functions, these are not known to be associated with any specific deficiency syndromes.

VITAMIN C (ASCORBIC ACID)

Vitamin C has important roles in collagen synthesis and synthesis of steroid hormones, neurotransmitters and bile acids. Vitamin C increases the gastrointestinal absorption of iron and also has important antioxidant activity.

Dietary Sources

Citrus fruits, tomatoes, capsicum and green leafy vegetables are good sources of vitamin C. Breast milk is a good source of vitamin C, and children-consuming animal milk are at risk of deficiency.

Deficiency State

Scurvy, resulting from severe vitamin C deficiency, manifests as gum bleeding, petechial hemorrhages, painful and swollen bones, and poor wound healing. Children having scurvy often have other nutrient deficiencies including severe malnutrition, anemia and vitamin B complex deficiency. The diagnosis of scurvy is usually made by radiographs; the changes being most prominent at the knee (Fig. 4.4.1). The shafts of the long bones have a typical ground-glass appearance with thin and dense cortex (pencil thinning). Metaphyseal changes are characterized by thickened bands (white line of Frankel) with a zone of destruction underneath (Trummerfeld zone). Ends of the white lines often end abruptly into a spur (Pelkan’s spur). Epiphyses are also outlined by dense line giving the appearance of a ring (Wimberger’s ring). Subperiosteal hemorrhages may complicate the disease causing periosteal elevation and underlying calcifications.

Treatment

Oral administration of vitamin C (100–200 mg/day) results in complete recovery. Resolution of clinical symptoms is rapid whereas bony changes and subperiosteal hemorrhages take more time to recover. Treatment must be followed by ensuring adequate dietary vitamin C to prevent recurrence.

Figure 4.4.1 Bony changes of scurvy at knee
Abbreviations: WL, White line; TZ, Trummerfeld’s zone; PS, Pelkan’s spur.
4.5  Fat-soluble Vitamins

(Late) Panna Choudhury

VITAMIN A

Vitamin A is a generic descriptor of retinoids that exhibit qualitatively the activity of all transretinol compounds. Retinol signifies vitamin A alcohol and is found in foods of animal origin only. Some carotenoids, which are found in plants, bacteria, algae and fungi, can be converted into retinol and are called provitamin A. The carotenoid with the highest vitamin A activity is beta-carotene. Beta-carotene yields two molecules of retinol. Retinol is esterified in the mucosal cell with palmitic acid. Retinyl palmitate is stored in the liver. Being fat soluble, retinol mobilized from the liver must be bound in serum to retinol binding protein (RBP), which is synthesized in the liver. RBP also protects retinol from oxidation and releases it to specific receptor sites on the surface of the target cell. Zinc deficiency impairs synthesis of RBP which affects retinol transport from the liver to the blood and other tissues.

Physiology

Retinol is the predominant circulating form of vitamin A in the blood. In response to tissue demand, it is released from the liver in a 1:1 ratio with RBP. In the blood, this complex combines with transthyretin. Specific receptors on target cell surfaces or nuclei bind this complex or its active metabolites, thereby regulating many critical functions in the body, including vision, growth and hematopoiesis. It is often termed as anti-infective vitamin for its role in maintaining epithelial tissue integrity and immune competence. However, the key to most of these functions is the role of vitamin A in regulating the expression of several hundred genes and cell differentiation practically for every cell in the body. The importance of vitamin A in functioning of the retina for vision has been well established. Visual pigment in rods is called rhodopsin, which is composed of a protein called opsin and a pigment, 11-cis retinine (vitamin A). Rhodopsin is light sensitive and when light falls on eyes, rhodopsin splits and 11-cis retinine is converted to 11-trans retinine. This initiates an electrochemical signal to be carried to the brain where visual images are constructed. In vitamin A deficiency, the threshold for stimulating rods is raised, thus, affecting the vision under dim light.

Quantification of vitamin A is expressed in various ways. One international unit (IU) equals 0.3 mcg retinol and 0.6 mcg of beta-carotene (1 mcg retinol will equal 3.31 IU).

Dietary Sources

Rich sources of vitamin A include fish liver oils, whole milk and milk products like butter, cheese and egg yolk. Carotenoids are plentiful in fruits and vegetables that are green or deep yellow or orange in color, such as green leafy vegetables, carrots, tomatoes, sweet potatoes, papaya and mango. Food fortified with vitamin A is an important source. Breastfeeding protects children during infancy.

Vitamin A Deficiency

Daily intake of vitamin A as retinol equivalent has been recommended as 350 mcg for infants, 400 mcg for preschool children and 600 mcg for school children and adolescents by Indian Council of Medical Research. Deficiency of vitamin A can occur from deficient diet, decreased absorption due to chronic intestinal disorders or reduced storage in liver diseases. Vitamin A deficiency leads to impaired vision

BIBLIOGRAPHY

besides many ocular and extraocular lesions. Vitamin A deficiency was also believed to be associated with higher mortality and morbidity in children. However, recent evidence has not substantiated the mortality reduction claim of vitamin A supplementation. A large trial covering one million children in Uttar Pradesh has not found significant difference in mortality rates between children who received the massive-dose of vitamin A and those who did not.

Clinical Features of Deficiency State

Ocular Lesions

These affect the posterior segment of eye initially with impairment of dark adaptation and night blindness. Often, the mother of the infant notices that, the child takes considerable time to adjust to dim light or darkness (twilight blindness). Xerosis of conjunctiva is usually the first sign that can be seen on examination. The conjunctiva becomes dry, lusterless, wrinkled and dirty brown in color. These changes are most obvious in the interpalpebral bulbar conjunctiva. Conjunctival xerosis may lead to formation of the so-called “Bitot’s spot”, which consists of almost a triangular area, usually about the temporal aspect of the limbus covered by a fine, white foamy or greasy substance. It is composed of heaped up sloughed-off keratinized cells and saprophytic bacilli, which collect on conjunctival surface (Fig. 4.5.1A). Corneal xerosis reflects more advanced deficiency (Fig. 4.5.1B). Keratomalacia is seen in the late stage and consists of softening, necrosis and ulceration of the cornea. Once cornea gets involved, photophobia accompanies the clinical profile. World Health Organization (WHO) has proposed a classification for xerophthalmia (Table 4.5.1).

Extraocular Lesions

These include dry, scaly skin, especially over the outer aspect of the limbs, called follicular hyperkeratosis, toad skin or phrynoderma. Increased susceptibility to infections due to squamous metaplasia of respiratory, urinary and vaginal tract epithelium; renal and vesical calculus may occur more often in such subjects.

Diagnosis of Vitamin A Deficiency

In the presence of clinical manifestations, diagnosis is not difficult. Dark adaptation tests can be used to assess early stage vitamin A deficiency. Serum retinol level below 20 mcg/dL, is indicative of vitamin A deficiency state. Conjunctival impression cytology is a noninvasive technique that assesses vitamin A status by detecting early losses of vitamin A-dependent, mucus secreting goblet cells and early metaplasia of the epithelium.

Prevention of Vitamin A Deficiency

It is heartening that prevalence of clinical signs of vitamin A deficiency has declined drastically. There is virtual disappearance of keratomalacia. Survey of the National Nutrition Monitoring Bureau showed that prevalence of Bitot’s spots had declined in 2006 compared with 1985 from

<p>| Table 4.5.1: WHO classification for xerophthalmia |</p>
<table>
<thead>
<tr>
<th>Classification</th>
<th>Primary signs</th>
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<tbody>
<tr>
<td>X1A</td>
<td>Conjunctival xerosis</td>
</tr>
<tr>
<td>X1B</td>
<td>Bitot’s spots</td>
</tr>
<tr>
<td>X2</td>
<td>Corneal xerosis</td>
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<tr>
<td>X3A</td>
<td>Corneal ulceration</td>
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<td>X3B</td>
<td>Keratomalacia</td>
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<td>Night blindness</td>
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<td>XF</td>
<td>Fundal changes</td>
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<tr>
<td>XS</td>
<td>Corneal scarring</td>
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Figures 4.5.1A and B (A) Bitot’s spots; (B) Corneal xerosis
Prevalence of Bitot's spots of 0.5% and more (conventional cut-off to define public health problem) is mostly limited to population groups in backward areas. However, subclinical vitamin A deficiency may still be a significant problem.

Prevention of vitamin A deficiency can be achieved by making available the recommended daily allowances of vitamin A to all children. According to the National Program for Prevention of Blindness, children in the age group of 6–11 months should receive 100,000 IU (30 mg) of vitamin A orally (preferably during measles immunization), and other children between 1 year and 5 years should receive 200,000 IU (60 mg) vitamin A every 6 months in the target areas. Infants who are not breastfed should also receive a dose of 50,000 IU (15 mg) at 2 months of age. Considering that country is endowed with various varieties of fruits, and green leafy vegetables, which are rich sources of beta-carotene, vitamin A deficiency is easily preventable. Daily intake of 100 g of leafy vegetables is an efficient way to meet the requirement of vitamin A. Fortification of commonly eaten foods with vitamin A can be an effective prophylactic measure.

Treatment of Vitamin A Deficiency

For treatment of xerophthalmia, according to WHO guidelines, 200,000 IU vitamin should be given orally on presentation, the following day and whenever possible, 1–4 weeks later. Infants aged between 6 months and 12 months should receive a half dose, and infants less than 6 months should receive one-quarter the dose, following the same schedule. Children with severe malnutrition, recurrent diarrhea, pneumonia and severe infections should also receive full treatment course of vitamin A. Cochrane review showed that vitamin A megadoses (200,000 IU on each day for 2 days) lowered the number of deaths from measles in hospitalized children under the age of 2 years.

Hypervitaminosis A

Signs of toxicity may appear with massive doses (e.g. 300,000 IU) or with large doses over a long period (e.g. over 20,000 IU per day). Child may have nausea, vomiting, drowsiness, papilledema and symptoms suggestive of raised intracranial tension. This syndrome is known as pseudotumor cerebri. In chronic cases, marked anorexia, failure to thrive, alopecia, seborrheic dermatitis, hepatomegaly and tender bone swelling may develop. Radiographic examination may show hyperostosis of the shafts of long bones. Symptoms subside on withdrawal of the vitamin. However, beta-carotene ingestion is seemingly without toxicity. With chronic high consumption, the skin but not the sclera is stained yellow-orange, which is benign and reversible.

VITAMIN D

Antirachitic properties of vitamin D are the result of small structural changes, under the influence of ultraviolet irradiation in a number of steroids related to cholesterol. However, only ergosterol and 7-dehydrocholesterol have practical importance. Ergosterol is of plant origin, and on irradiation, it transforms to vitamin D₂ (calciferol). The 7-dehydrocholesterol is normally present under the skin, and on exposure to ultraviolet rays of the sunlight, it converts to vitamin D₃ or cholecalciferol. The latter is converted to 25-hydroxy calciferol in the liver and is further converted to 1,25-dihydroxy cholecalciferol, which is specifically helpful in promoting synthesis of "calcium transport protein" in the intestinal wall. Parathormone controls the production of 1,25-dihydroxy cholecalciferol, the metabolically active form of vitamin D. Quantification of vitamin D is expressed as 1 mcg will equal 40 IU.

Sources

Vitamin D, unlike other vitamins is not abundantly available in foodstuffs. Rich source of vitamin D is fish liver oil and to some extent, it is available in butter and egg. Milk provides minimal amounts of vitamin D (0.1 mcg/dL). Cutaneous synthesis is an important source of vitamin D, but efficiency of the process is affected, if ultraviolet rays are cut-off by haze, windowpane, oblique rays, etc. Pigmented skin also requires more sunlight exposure to be effective.

Daily Requirement

The normal daily requirements of vitamin D for infants and children is 400 IU (10 ug). In Indians, due to our skin pigment and modest pattern of clothing, 45 to 60 min of direct sun exposure to face, neck, forearms and hands in summer, and 150–180 min to face and hands in winter, should provide adequate dermal vitamin D production. Considering the rampant vitamin D deficiency and the vulnerability to its serious consequences during infancy, the current recommendations suggest daily supplementation of 400 IU/day for all infants beginning in the first few days of life.

Vitamin D Deficiency

Infants are more prone to vitamin D deficiency, as natural diet of infants like milk, cereals, vegetables and fruits are deficient in vitamin D. This gets aggravated, if there is also lack of access to sunlight. In a study from Delhi, clinical vitamin D deficiency was noted in 11.5% apparently healthy school girls, whereas biochemical hypovitaminosis D (serum 25-hydroxyvitamin D <50 nmol/L) was seen in 90.8% of girls. Vitamin D deficiency also occurs in presence of malabsorption, liver and kidney diseases. Rickets, a metabolic disorder of growing bone leading to bony deformities, when results from vitamin D deficiency are known as nutritional rickets.

Biochemical Changes

Vitamin D deficiency causes decreased absorption of calcium from gut. The resulting hypocalcemia leads to increase in
parathormone secretion. This helps in release of calcium from bone. Parathormone also reduces the excretion of calcium by kidneys and renal tubular reabsorption of phosphate. As a result, the serum calcium level tends to become normal, while the serum phosphate level falls. After sometime, this compensatory mechanism fails and both calcium and phosphorus levels fall. Since calcium phosphate is necessary for deposition of calcium in growing bones, decrease in blood levels of calcium, phosphorus or both interfere with the calcification of the osteoid tissue. Serum alkaline phosphatase level also gets increased due to increase in osteoblastic activity.

**Pathology of Rickets**

The epiphyseal plate is a narrow well-defined strip from where cartilage cells grow in parallel column towards the metaphysis. After initial proliferation, the old cartilage cells degenerate and disappear, leaving spaces into which the blood vessels and osteoblasts of the shaft can penetrate. Calcium is deposited in the zone of degenerating cartilage, which is then called “zone of preparatory calcification”. In rickets, the cartilage cells go on multiplying giving rise to a broad, irregular cartilaginous zone. The process of degeneration and calcification becomes incomplete, leading to softness of the bone. Rapidly growing cartilage cells, particularly, affect the costochondral junctions and the end of long bones. There is also defective mineralization in the subperiosteal bone. In long-standing cases, the bones under stress may become deformed or even have pathological fractures. Supplementation of vitamin D restores the normal development of bone with calcification starting at the zone of preparatory calcification, which in radiography would be seen as a thin dense line near the epiphysis.

**Clinical Features**

Rickets is a disease of growing bones and its incidence is particularly high between 4 months and 18 months. Skeletal deformities are the most striking feature of rickets. One of the early signs of rickets is craniotabes. In this condition, on pressing occipital or posterior part of parietal bone, a sensation like pressing a ping-pong ball can be felt. It results from the thinning out of inner table of the skull due to absorption of noncalcified osteoid tissue. Fontanel may remain wider than normal and close late. Other early evidences of osseous changes are palpable enlargement of costochondral junctions, i.e. rachitic rosary and widening of the wrists (Fig. 4.5.2) and ankles.

Signs of advanced rickets can be easily recognized. Bossing of skull generally starts after the age of 6 months. It occurs due to heaping up of osteoid tissue in the frontal and parietal regions so that the skull appears square-like or box-like shape. In thorax, the sternum is pushed forward producing a “pigeon chest”. A horizontal depression known as Harrison’s groove, corresponding to costal insertion of the diaphragm develops. The chest deformities decrease the lung resilience and predispose the child to intercurrent infections. Bending of the spine backwards (kyphosis) and laterally (scoliosis) may occur. Pelvis may become softened, and the promontory of the sacrum is pushed anteriorly and the acetabulae inwards, resulting in a narrowed pelvic inlet. This is helped by lax ligaments. Deformity of the pelvis in a female results in difficulty during labor at a later stage. Long bones of the legs get deformed when the child starts bearing weight and is thus, usually seen after the age of 1 year. Bending of the femur, tibia and fibula result in deformities as, “bow-legs” or “knock-knees”. Coxa vara and green-stick fractures may also occur. All deformities of bones result in rachitic dwarfism. Dentition may be delayed and disordered eruption of temporary teeth occurs. In children between 8 months and 18 months, permanent teeth, which are undergoing calcification, may be affected.

Besides skeletal deformities, there is a generalized hypotonia with delay in motor development. The abdomen is protuberant, and generalized flabbiness of muscles may result into visceroptosis with downward displacement of spleen and liver.

**Diagnosis**

The diagnosis of rickets is based on the clinical features, biochemical findings and characteristic radiological picture. The serum calcium level may be normal or low, the serum phosphorus level is below 4 mg/dL, and the serum alkaline phosphatase is usually elevated. Radiological changes are best seen in the lower end of radius and ulna. Skiagram of the wrist shows widening, cupping and fraying of the epiphyses in contrast to the normally sharply demarcated and slightly
convex epiphyseal line (Figs 4.5.3A and B). The density of shafts decreases with prominent trabeculae. There is an increase in distance between concave epiphyseal line and the ends of metacarpals. Green-stick fractures, expansion of bone ends and bending of bones may be evident on radiographs. Periosteum may be raised due to excess of osteoid lying under the periosteum.

**Differential Diagnosis of Rickets**

Nutritional rickets should be differentiated from other types of rickets and chondrodystrophy. Other conditions producing bony deformities may, sometimes, need consideration. Craniotabes and a large head apart from rickets occur in hydrocephalus, congenital syphilis and osteogenesis imperfecta. Enlargement of costochondral junctions may also be seen in scurvy and chondrodystrophy.

**Management**

Vitamin D is given in a dose of 2000 (for infants) to 5000 (for older children) IU orally daily. If there is no sign of healing line in skiagram taken 3–4 weeks after therapy, the same dose can be repeated. If there is no response within 3–4 weeks of the second dose, investigations for refractory rickets should be initiated. Rickets can also be treated with single large dose of 60,000 (older infants) to 120,000 (older children) IU orally, with a repeat dose if necessary, after 2 months. Single large dose therapy may cost less and can be given under direct observation. However, it is not recommended below 3 months of age, due to fear of toxicity. After the healing of rickets, normal daily requirement of calcium and vitamin D should be ensured. Deformities of bones are corrected by orthopedic measures.

**Hypervitaminosis D**

High dose of vitamin D given over a long period may cause anorexia, vomiting, hypotonia, irritability, polydypsia and polyuria. There is hypercalcemia and hypercalciuria. Radiological examination reveals evidence of metastatic calcification and osteoporosis of long bones.

**VITAMIN E**

Vitamin E is a group of closely-related, naturally occurring fat-soluble compound of which tocopherol is functionally the most potent. It is active as an antioxidant and, probably involved in the metabolism of nucleic acids. It is widely present in most foods. One milligram of alpha-tocopherol provides 1.5 IU activity of vitamin E. The deficiency of vitamin E is rare. The most common causes are diarrhea and poor intake of food. Deficiency may result in areflexia, ataxia, muscle weakness and dysarthria. In premature infant, low levels of vitamin E are associated with hemolytic anemia, hyperbilirubinemia and intraventricular hemorrhage. This responds quickly to 5–25 mg of vitamin E therapy. Generally, infants should receive 3 mg of alpha-tocopherol daily.

**VITAMIN K**

Vitamin K is a naphthoquinone derivative. Absence or failure of its absorption from the intestine leads to hypoprothrombinemia and decreased synthesis of some coagulation factors (VII, IX and X). The normal requirement of vitamin K is met by bacterial synthesis in the intestine. In addition, it is also found in high concentration in a wide variety of foods.
foods and vegetables like spinach, cabbage, peas, tomatoes, soybean and liver.

The deficiency of vitamin K can occur in malabsorption states, biliary obstruction, after oral antibiotic therapy or in newborn before colonization of the guts. In general, vitamin K deficiency or hypoprothrombinemia should be considered in all patients with hemorrhagic disturbances. Hemorrhagic disease of the newborn is one of the most common manifestations. The bleeding is variable and can occur anywhere, though, most common is gastrointestinal bleeding. A daily dose of 1–2 mg of vitamin K orally is sufficient for treatment. In severe deficiency state, 5 mg of aqueous vitamin K can be given parenterally.

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**INTRODUCTION**

Of the 109 well-characterized elements, several of them are found essential for human growth, development and function. These include trace elements (TEs). Synonyms are mineral nutrients, microminerals, bionutrients or bioelements. World Health Organization (WHO) expert consultation has categorized nutritionally significant trace elements into three groups: (i) Essential trace elements: Iron, iodine, zinc, selenium, copper, molybdenum and chromium; (ii) Elements which are probably essential: Manganese, silicon, nickel, boron and vanadium; and (iii) Potentially toxic elements: Fluorine, lead, cadmium, mercury, arsenic, aluminum, lithium and tin.

**DEFINITION**

Trace elements are those minerals present only in minute amounts in a particular sample or environment (usually <10 parts per million) or those required only in minute amounts by living organisms for normal growth. By definition, trace elements are those present in concentration less than 0.01% body dry weight, i.e. less than 0.1 mg/g or less than 100 µg/g, previously denoted as detected in trace quantities. Ultratrace elements occur in smaller quantities less than 0.0001% by weight less than 1,000 ng or 1 µg/g.

**EPIDEMIOLOGY**

A balanced diet that includes all the food groups can supply all the trace elements. Infant and young child feeding (IYCF) practices, especially in the minus nine to plus 24 months that corresponds to the first 1,000 days of life, is crucial in preventing deficiency and ensuring enough stores of trace elements. Trace elements deficiencies occur usually in those with protein energy malnutrition (PEM), picky eaters, low birth weight (LBW) babies and in those on exclusion diets and total parenteral nutrition (TPN). Trace element deficiencies noted with TPN are given in Table 4.6.1. Excess intake of dietary fiber, phytates and oxalates reduce trace element absorption. Both, deficiency or excess and current trace element status can be detected from hair with accuracy. The various aspects related to trace elements are summarized in Table 4.6.2.

**DIAGNOSIS**

Except in a few items like iron, iodine, zinc and selenium, specific features of deficiency may not occur. A high index of suspicion and therapeutic response to supplementation help in clinching the diagnosis. Levels can be estimated by colorimetry, atomic absorption spectrophotometry (AAS) and neutron activation analysis (NAA).
Boron is recognized essential for healthy bones and utilization of vitamin D and calcium in the body. Cobalt is an essential component of vitamin B12; increases iron absorption and iodine utilization. Deficiency produces anemia and goiter and excess can lead to goiter and cardiomyopathy. Germanium is said to be the secret behind the health benefits of garlic, ginseng and mushrooms. Fluorine is not considered an essential mineral by some as humans do not require it for growth or to sustain life. However, it prevents dental caries and primary action occurs topically. Strontium is involved in utilization of calcium. It promotes calcium uptake into bone at moderate dietary levels, but has rachitogenic action at higher dietary levels. Arsenic, bromine, cadmium, silicon, tungsten, and vanadium have biochemical roles as structural or functional cofactors in other organisms. Arsenic is thought to promote nail and hair growth. Excess residue found in cow’s milk may be toxic to skin, CNS and respiratory tract. Mercury is toxic to enzymes and ribonucleic acid (RNA). Excess leads to Minamata disease in fetus and acrodynia in others.

**Others**

Anemia due to iron deficiency anemia (IDA) is a common health issue in infants and children. Iron is essential for hematopoiesis, and its deficiency leads to microcytic hypochromic red blood cells (MCV). Cobalt is an essential component of vitamin B12 and is necessary for the utilization of vitamin D and calcium in the body. Deficiency produces anemia and goiter, and excess can lead to goiter and cardiomyopathy. Germanium is said to be the secret behind the health benefits of garlic, ginseng, and mushrooms. Fluorine is not considered an essential mineral by some as humans do not require it for growth or to sustain life. However, it prevents dental caries and primary action occurs topically. Strontium is involved in the utilization of calcium. It promotes calcium uptake into bone at moderate dietary levels, but has rachitogenic action at higher dietary levels. Arsenic, bromine, cadmium, silicon, tungsten, and vanadium have biochemical roles as structural or functional cofactors in other organisms. Arsenic is thought to promote nail and hair growth. Excess residue found in cow’s milk may be toxic to skin, CNS, and respiratory tract. Mercury is toxic to enzymes and ribonucleic acid (RNA). Excess leads to Minamata disease in fetus and acrodynia in others.

**Practice Guidelines and National Prophylaxis Programs with Respect to Trace Elements**

- **Iron deficiency anemia (IDA):** Iron insufficiency leads iron depletion, iron deficiency, and IDA with microcytic hypochromic red blood cells (RBCs) with increased red cell distribution width (RDW), reduced physical stamina, lack of concentration and learning ability, pica, and koilonychia. Pica includes eating disorders like geophagia (mud), amylophagia (eating raw rice), and pagophagia (ice cubes). Even mild-to-moderate anemia in infancy and childhood may lead to permanent changes in the brain. IDA is an added risk factor for breath holding spell, febrile fit, and hypercyanotic blue spell. As part of “iron plus” prophylaxis program, iron folic acid (IFA) pediatric tablets or syrup with 20 mg elemental iron and 100 mcg folic acid are given to 6 months to 6-year-old children for 100 days/year preferably as twice a week. IFA adult has 5 times more concentration and 1 mL IFA syrup is equivalent to one IFA pediatric tablet and 5 mL is equivalent to one IFA adult tablet. Weekly iron folic acid supplementation (WIFS) is found beneficial in children and adolescent boys and girls and is integrated with school health program and adolescent clinics in some states of India. Normal Hb (g/dL) levels vary in different age groups (WHO 1968): During pregnancy and children, 6 months to 6 years, 10–11 g/dL is mild anemia, 7–10 is moderate and less than 7 g/dL is severe anemia. Newborn: More than 13; 2–6 months: More than 9; 6 months to 6 years: 11; 6–12 years: 12; 13 years–13 and pregnancy: >11 g/dL. In very severe deficiency, hemoglobin less than 4–5, packed red cell transfusion is advised initially followed by iron.

- **Iodine deficiency disorder (IDD):** About two-thirds of this is generally derived from the drinking water and one-third from diet. Universal iodization of salt ensuring potassium iodate 15 ppm (15 mcg/g) is recommended in India. Salt testing kits are made available to ensure optimum iodization. In commercial iodized salt; up to 30–50 ppm is added to cover losses. Double fortified salt contains potassium iodate and ferrous sulphate to tackle IDA and IDD. Urinary iodine excretion is reduced in deficiency. Urinary iodine can be tested using dipstick or laboratory method. Excretion less than 100 mcg/L indicates iodine deficiency, 50–100 mild, 20–50 moderate and less than 20 indicates severe deficiency. Goiter is an indication of previous deficiency state and rates more than 5% in prepubertal children indicates endemic goiter; 5–20% mild, 20–30% moderate and >30% severe. Grade 0: goiter not visible or palpable, Grade 1: goiter palpable and Grade 2: both visible and palpable.

- **Zinc deficiency:** Subclinical zinc deficiency is not uncommon. It is useful in treatment of Wilson’s disease. Zinc supplementation results in better catch up growth in LBW and PEM. In field settings, ORS and Zn are promoted in diarrhea for diarrhea control program and also for prevention of diarrhea as Zn results in repair of mucosa, replenishment of brush border enzymes and fluid regulation. About 20 mg锌 sulfate tablets are made available for infants and young children.
<table>
<thead>
<tr>
<th>Trace Element</th>
<th>Sources</th>
<th>Functions</th>
<th>Deficiency</th>
<th>Clinical features</th>
<th>Requirement</th>
<th>Remarks and toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron</td>
<td>Heme— 7–35% and nonheme—2–20% Fish, meat, liver, 3 Gs: grams, grains, greens Jaggery/molasses, asafoetida, turmeric, dates, watermelon Cooking in iron vessels</td>
<td>Constituent of myoglobin and enzymes, role in oxygen transport For physical stamina, learning ability and myelination Also known as the ‘energy mineral’</td>
<td>LBW, excess cow’s milk, blood loss, hookworm, malabsorption poor intake, increased demand</td>
<td>Pallor, dyspnea, CCF, irritability, lack of concentration, pica, kollonychia Investigation: S. iron (50–150 µg/dL), iron-binding capacity (100–400 µg/dL) S. ferritin (50–250 mg/mL), transferrin saturation &lt;15%, blood smear hypochromic microcytic anemia, MCV, MCH, MCHC reduced and RDW increased</td>
<td>Prophylaxis: 2–3 mg/kg/day. children 10–20 mg/day, pregnancy and lactation 30–40 mg/day Treatment oral: 4–6 mg/kg/day for 3–4 months. Inj weight in kg × deficit in g/dL × 2.5+25%, Iron sucrose IV/IM Packed red cell transfusion: 5–10 mL/Kg. Always treat the cause</td>
<td>Oxalate, phytates, Zn, coffee/tea inhibit absorption. Vitamin C, cobalt, lime juice and acid medium increase absorption. Toxicity: Chronic-hemolysiderosis, hemochromatosis Acute: GI upset, Stages of quiescent phase, metabolic derangement, hepatic failure and strictures. Gastric lavage with soda bicarbonate and desferrioxamine. IV Desferal 50 mg/kg followed by 10 mg/kg/hour × 24 hour S. iron &gt;1800 µg/dL is fatal</td>
</tr>
<tr>
<td>Iodine</td>
<td>Seafoods, drinking water (two-thirds requirement), iodized salts</td>
<td>Constituent of thyroxine, for metabolic control, modulation of estrogen and fetal health</td>
<td>Low content in water, especially mountainous areas, excessive intake of brassica species: cabbage, cauliflower</td>
<td>Endemic goiter, hypothyroidism, stillbirth, CNS defect Investigation: urinary iodine, PBI, T3, T4, TSH, urinary iodine, PBI, T3, T4, TSH, iodine uptake study</td>
<td>50–150 µg/day</td>
<td>Iodized salt to contain 15 µg/g (15ppm); up to 30 ppm added to tackle loss. Excess can cause iodism, reversible dermatitis and cobalt deficiency/excess, iron deficiency, manganese excess and fluorine excess may aggravate goiter</td>
</tr>
<tr>
<td>Copper</td>
<td>Liver, fish, meat, oyster, legumes, competes with Zn and Mo for absorption</td>
<td>Constituents of enzymes, ceruloplasmin, and hormone function, role in hemopoiesis, essential for Zn, iron and vitamin C function, bone. Also called twin mineral of iron</td>
<td>LBW, preterm TPN, PEM, nephrotic syndrome, Menke’s kinky hair syndrome is an X-linked metabolic disturbance of copper metabolism characterized by mental retardation, abnormal hair texture, hypocupremia (&lt;65 mcg/dL) and low circulating ceruloplasmin (&lt;20 mg/dL).</td>
<td>Hypochromic anemia, neutropenia, hypopigmented hair, bony defects Investigation: S. Cu 75–150 µg/dL S. ceruloplasmin 10–50 µg/dL</td>
<td>1–2 mg/day</td>
<td>Toxicity: Indian childhood cirrhosis, hepatitis, cirrhosis, Coomb’s negative hemolytic anemia, Zn deficiency. Cu deposition occurs in Wilson’s disease</td>
</tr>
<tr>
<td>Zinc</td>
<td>Liver, beef, oyster, cereals, nuts, grapes</td>
<td>Constituents of enzymes, role in protein and nucleic acid synthesis, epithelial repair, fluid electrolyte transport, taste function, immunity</td>
<td>PEM, TPN, hepatitis, nephrotic syndrome, acrodermatitis enteropathica, especially as a genetic defect</td>
<td>Growth retardation, anorexia, gonadal atrophy, alopecia, dermatitis, diarrhea, reduced taste sensation. Investigation: S. Zn 60–150 µg/dL, Zn in hair</td>
<td>5–15 mg/day Treatment: Diarrhea: ORS and Zn: 2–6 m: 10 mg/day and &gt;6 m: 20 mg/day × 2 weeks Dose 1–2 mg/kg/day up to 150 mg elemental zinc</td>
<td>Phytates reduce absorption, excess reduces iron and copper levels. Used as adjuvant in Wilson’s disease. Toxicity: GI upset, Cu deficiency</td>
</tr>
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<tr>
<th>Element</th>
<th>Foods</th>
<th>Functions</th>
<th>Symptoms</th>
<th>Investigation</th>
<th>Treatment</th>
<th>Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chromium</td>
<td>Yeast, liver cereals, nuts, cocoa, pepper</td>
<td>Facilitates insulin action and weight loss, help to prevent diabetes</td>
<td>Hyperglycemia, encephalopathy. <em>Investigation</em>: S. Cr 0.02 µg/dL</td>
<td>10 µg/d <em>Treatment</em>: Single dose 180 µg in hyperglycemia</td>
<td>Toxicity: Renal failure, dermatitis</td>
<td></td>
</tr>
<tr>
<td>Fluorine</td>
<td>Drinking water, seafoods, tea, cheese</td>
<td>Constituent of bone and teeth</td>
<td>Dental caries</td>
<td>1–5 mg/day <em>Drinking water up to 1 ppm</em></td>
<td>Excess: Dental and skeletal fluorosis, genu valgum with excess in drinking water and sorghum intake, &gt; 2–3 ppm in drinking water needs defluoridation by alum and/or by reverse osmosis (OS)</td>
<td></td>
</tr>
<tr>
<td>Selenium</td>
<td>Meat groups, green, garlic</td>
<td>Antioxidant, cofactor of enzyme function, maintains liver integrity</td>
<td>Keshan cardiomyopathy, arthritis, myalgia, growth retardation, liver necrosis, risk of liver cancer. <em>Investigation</em>: S. Se 13 µg/dL</td>
<td>100 µg/day</td>
<td>Dental caries, alopecia, garlic odor in breath</td>
<td></td>
</tr>
<tr>
<td>Manganese</td>
<td>Cereals, legumes, greens, tea</td>
<td>Component of superoxide dismutase, role in oxidative phosphorylation</td>
<td>Growth retardation, reddening of hair, increased prothrombin time <em>Investigation</em>: S. Mn 0.06 µg/dL</td>
<td>1–5 mg/day</td>
<td>Iron decreases Mn absorption. Toxicity: Encephalitis, goiter, cardiomyopathy, cholestasis</td>
<td></td>
</tr>
<tr>
<td>Nickel</td>
<td>Chocolate</td>
<td>Component of urease, and nickel plasmin, stabilizes membranes</td>
<td>Not known</td>
<td>Not known</td>
<td>Excess: Dermatitis, liver necrosis, nasal and lung cancers</td>
<td></td>
</tr>
<tr>
<td>Silicon</td>
<td>Cross-linkage of collagen</td>
<td>Growth retardation, defective bone growth</td>
<td>Not known</td>
<td>Not known</td>
<td>Excess: Granuloma and fibrosis of lung</td>
<td></td>
</tr>
<tr>
<td>Vanadium</td>
<td>Protein rich food</td>
<td>TPN</td>
<td>PEM</td>
<td>Deficiency associated with nutritional edema</td>
<td>Not known</td>
<td></td>
</tr>
<tr>
<td>Molybdenum</td>
<td>Legumes and green leafy vegetables, liver</td>
<td>Molybdenum containing enzymes in humans are xanthine oxidase/dehydrogenase, aldehyde oxidase and sulfite oxidase</td>
<td>Molybdenum deficiency coexisting with selenium deficiency may be obligatory for the development of Keshan disease. Tachycardia, central scotoma, irritability, coma and probably increased incidence of mouth and esophageal cancers</td>
<td>2–3 mcg/kg/day</td>
<td>Excess may unmask hyperuricemia, gout and genu valgum. Abnormally, high level leads to copper deficiency</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations:** TE, trace element; LBW, low-birth-weight; TPN, total parenteral number; PEM, protein energy malnutrition; PBI, protein-bound iodide; TSH, thyroid-stimulating hormone; ORS, oral rehydration salts; IM, intramuscular; IV, intravenous; CNS, central nervous system; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; RDW, red cell distribution of width.
available in reproductive and child health (RCH) kit, 1/2 tablet in 2–6 months old and 1 tablet more than 6 months old for 14 days. In those less than 2-month-old, it is not given as a routine medication, but can be prescribed, whenever indicated. Skin changes induced by zinc deficiency in acrodermatitis enteropathica is symmetrical and periorificial (Fig. 4.6.1) compared to more extensive lesions in nutritional dermatosis of severe malnutrition, which is also associated with growth failure.

**KEY MESSAGES**

- Most of the trace elements are essential or probably essential, but a few are potentially toxic  
- Trace elements are required only in minute quantities and are present in selected food items  
- Deficiency of these micronutrients causes various pathologic effects in human body. Excess can also cause serious adverse reactions in body. So optimum trace element balance in body is essential  
- Optimum trace element balance should be considered in TPN and food fortification  
- Trace element deficiencies should be diagnosed in the early phase and corrected  
- Along with optimum dietary intake recommendations, factors influencing the absorption and bioavailability should be considered.

**BIBLIOGRAPHY**


**4.7 Human Milk Banking**

**INTRODUCTION**

History of breastfeeding dates back since evolution in mammals (around 4200 BC) on this mother earth. Each mammalian milk is species specific perfectly suited to their offspring.  

Millennium Development Goals 4 aims to reduce under-five child mortality by two-thirds by 2015. In order to accelerate the progress on child survival, there is heightened global interest in increasing the rates of optimal infant and young child feeding practices, especially the human milk for the first 6 months. The under-5 mortality rate (U5MR) for India was estimated at 59 for the year of 2010 (Sample registrations survey 2010). Infant mortality rate (IMR) in India has registered a decline from 58 in 2005 to 44 in 2011. World Health Organization (WHO) estimates that 53% of acute pneumonia and 55% of diarrhea deaths are attributable to poor feeding practices during the first 6 months of life? WHO and United Nations Children’s Fund (UNICEF) made a joint statement in 1980: “Where it is not possible for the biological mother to breastfeed, the first alternative, if available, should be the use of human milk from other sources. Human milk banks should be made available in appropriate situations”.

Breastfeeding is the best method of infant feeding because human milk is uniquely suited to the human infant. All mothers should be encouraged to breastfeed their infants.
When a mother, for some reason, is unable to feed her infant directly, her breast milk should be expressed and fed to her infant. If mother’s own milk is unavailable or insufficient, the next best option is to use pasteurized donor human milk (PDHM). In India, the burden of low birth weight babies is about 20–30% with significant mortality and morbidities. Feeding these babies with breast milk can significantly reduce the risk of infections.

Though “wet nursing” had been in practice since mythological ages, modern human milk banking is in its infancy in India. The first human milk bank in Asia was established by Armida Fernandez in 1989 in Mumbai. Currently, the number of human milk banks has grown to nearly 18 but the growth has been very slow. One of the major reasons for loss of interest in human milk banking was the promotion of milk formula by the industry.

**LOCATION OF HUMAN MILK BANKING**

Human milk banks are primarily focused to provide donor milk to high-risk newborns. Thus, a location in close proximity or even inside the boundaries of neonatal unit is desirable. Postnatal wards are most suited for the purpose as the number of donors is likely to be in large number where counselors can encourage them to donate milk. Certain nongovernmental organization (NGOs) who take care of abandoned babies should have a human milk bank in their facility. Presence of human milk banks in the neonatal intensive care units (NICUs) is associated with elevated rates of exclusive breastfeeding rates in very low birth weight (VLBW) babies.

**THE RECIPIENTS**

Pasteurized donor human milk can be prescribed on priority for:

- Preterm babies and sick babies
- Babies of mothers with postpartum illnesses
- Babies whose mothers have lactation failure till their milk output improves.

Therapeutic benefits are noted in short gut syndrome, sepsis, and postsurgical gut healing in omphalocele, gastrochisis, bowel obstruction and intestinal fistulas. In extremely preterm infants given exclusive diets of preterm formula versus human milk, there was a significantly greater duration of parenteral nutrition and higher rate of surgical necrotizing enterocolitis (NEC) in infants receiving preterm formula. It is possible to do gut priming exclusively with human milk.

If PDHM supplies are sufficient donor milk may be supplied for:

- **Absent or insufficient lactation**: Mothers with multiple births, which cannot secrete adequate breast milk for their neonates initially
- For babies of nonlactating mothers, who adopt neonates, and if induced lactation is not possible
- Abandoned neonate and sick neonate
- Temporary interruption of breastfeeding
- Infant at health risk from breast milk of the biological mother
- Babies whose mother died in the immediate postpartum period.

**INFRASTRUCTURE**

There are no standard recommendations. The average requirement is a room of 250 square feet which can accommodate the equipment required for milk banking, a work area for the technician as well as some storage space for records and administration and area for counseling donors, etc. Privacy is of paramount importance for area of breast milk expression. Provision of music or television and crèche for her baby helps in reducing stress of donors. Teaching videos of Kangaroo mother care (KMC), expression of breast milk and advantages of breast milk feeding can be shown.

**Equipment**

**Pasteurizer or Shaker-Water Bath**

It is essential to have a device to carry out heat treatment of donor milk at the recommended temperature of 62.5°C for 30 minutes (Holder pasteurization). A conventional pasteurizer is extremely expensive and of dairy-industry size and is not suitable for a human milk banks. A well-accepted alternative is the use of a shaker water bath with a microprocessor controlled temperature regulator, an electronic timer device and a shaker speed controller. The milk in the container is boiled through the steam and hot water in the water-shaker bath. To avoid coagulation of the milk and to distribute heat evenly, the tray on which the stainless steel containers are placed is shaken wobbled. This shaker water bath should be double walled and made of steel. The size of the shaker-water bath varies according to the need of the milk bank with the tray capacity varying from 9 to 24 flasks or stainless steel containers of 200–400 mL capacity. This has been shown to inactivate human immunodeficiency virus (HIV) in breast milk samples and has been found to be nutritionally safe as well.

**Deep Freezer**

A deep freezer to store the milk at -20°C is essential. It is desirable to order a deep freezer with a digital display of the temperature with an alarm setting, preferably deep freezers. First for storage awaiting culture; used for storage of the milk till the postpasteurization milk culture reports are available. This freezer should be locked at all times, so that no milk is accidentally used till the culture reports are available.
The second deep freezer is used for storage of the pasteurized milk once the culture reports are negative and the milk is considered safe for disbursement. Refrigerators are required to store the milk till the whole day’s collection is over and the milk is ready to be mixed and pooled for further processing. It is also required for thawing the milk before being dispatched.

**Hot Air Oven or Autoclave**

A hot air oven or autoclave in the milk bank is essential for sterilizing the containers used for collection from donors, containers for pasteurizing and for storing the milk, as well as the test tubes needed for sending milk culture samples to the microbiology laboratory.

**Breast Milk Pumps**

For milk banking, hospital grade electric pumps are preferred as they result in better volumes of expressed milks and are relatively painless and comfortable to use. There is no major difference in the types of electrical breast pumps. If there are cost constraints, manually operated breast milk pumps can be used. They can be reused with chemical sterilization. Simultaneous breast expression in breastfeeding women is more efficacious than sequential breast expression. Breast pumps can be a source of infection. They should be cleaned properly. Detachable parts should be sterilized as per manufacturer’s instructions.

**Containers**

For collection and storing the collected milk, single use hard plastic containers of polycarbonates, Pyrex or propylene are used; however cylindrical, wide-mouthed stainless steel containers of 200 mL capacities with tight-fitting or screwed caps are equally effective. They are easily available, durable, easy to clean and autoclave. There is no significant decrease in nutrient composition on storage; however, cellular components are reduced. Polythene milk bags are not suitable as they are fragile, associated with loss of lipids and vitamins and there is a risk of contamination.

**Generator or Uninterrupted Power Supply**

Every milk bank should have a centralized source of uninterrupted power supply in the form of a generator, UPS or inverter to run the deep freezers and refrigerators in case of electricity failure.

**Milk Analyzer**

It is desirable to have macronutrient analysis of breast milk to estimate the calorie, protein and fat of a milk sample, using infrared spectroscopy technology, in teaching hospitals as a step toward lacto-engineering.

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**ADMINISTRATIVE STAFF**

Milk banks should have a panel from pediatrics, neonatology, microbiology, nutrition, lactation management, public health and food technology, milk bank officer (usually a doctor), lactation management nurses (for counseling mothers and assisting expression of breast milk), technician (for pasteurization of breast milk and microbiological surveillance), attendant (for collecting, sterilization of the containers and maintaining hygiene), receptionist (for record keeping and public relations) and a microbiologist (for microbiology testing and infection-control policies).

**General Guidelines for Staff**

- Hygienic practices like proper handwash, donning gowns, mask, gloves, trimming nails, locking long hairs should be maintained
- Gloves should be worn and changed between handling raw and heat-treated milk
- Standard operating procedures (SOP) of the bank (which should be displayed at proper places) should be adhered to
- Staff should undergo regular health checks and be immunized against hepatitis B virus (HBV).

Bank should have an operational objective of ensuring full traceability from individual donation to recipient and maintaining a record of all storage and processing conditions.

**DONOR POPULATION**

The donor population is formed by healthy lactating mothers with healthy babies, who are voluntarily willing to give their extra breast milk for other babies without compromising the nutritional needs of their own baby. Donors are not paid for their donations in India. The donors can include mothers attending well baby clinics, mothers whose babies are in NICU, mothers who have lost their babies but are willing to donate the milk, or lactating working staff in the hospital and motivated mothers from the community.

Maximum donor population should be reached by spreading awareness and mass communication. NGOs, social clubs and senior female students can play a good role in it.

Who can donate? Those lactating women can donate who;
- Are in good health and not regularly on medications or herbal supplements (with the exception of prenatal vitamins, human insulin, thyroid replacement hormones, nasal sprays, asthma inhalers, topical treatments, eyedrops, progestin-only or low dose estrogen birth control products).
- Willing to undergo blood testing for screening of infections
- Has enough milk after feeding her baby satisfactorily.

Who cannot donate? A donor is disqualified who:
- Uses illegal drugs or uses tobacco products or nicotine replacement therapy

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• Regularly takes more than two ounces of alcohol or equivalent or three caffeinated drinks per day
• Has a positive blood test result for HIV, human T-lymphotropic virus (HTLV), hepatitis B or C or syphilis
• Has she or has a sexual partner having high-risk behavior or suffering from HBV, HIV, hepatitis C virus (HCV) and venereal diseases in last 12 months
• Has received organ or tissue transplant, any blood transfusion or blood product within the prior 12 months
• Is taking radioactive or other drugs or has chemical environmental exposure or over-the-counter prescriptions or megadoses of vitamins, which are known to be toxic to the neonate and excreted in breast milk
• Has mastitis or fungal infections of the nipple or areola; reactivation of herpes simplex or varicella zoster infections in the mammary or thoracic region.

COlLecTion of bReast milk

After checking suitability for donation; getting written informed consent; history taking; physical examination and sampling for lab tests, the donor mother is sent to designate breast milk collection area. The breast milk is collected by trained staff after method of breast milk expression is chosen by the donor. Although home collection of the breast milk is being practiced in some countries abroad but the experts felt that due of the risk of contamination and difficulty in tracking, it is better avoided it at present in our country, however, milk collection camps can be practiced under proper supervision.

Washing the breast with simple water before expression is as good as washing with disinfectant. There is no rationale in discarding the first few milliliters of foremilk to reduce the infection rate. The breast milk may be expressed with the help of breast pumps either electrical or manual. Donation should be collected in properly labeled sterile container.

Processing

All batches of collected raw breast milk should be refrigerated immediately in preprocess refrigerator till the serological report comes negative. Fresh raw milk should not be added to the frozen milk since this can result in defreezing with hydrolysis of triglycerides. While mixing fresh raw breast milk to frozen raw breast milk previously collected from same donor, it should be chilled before adding to frozen milk. But for sick or preterm babies, it is advisable to use a new container for each pumping.

Microbiological screening of donated milk is done before (if there is no cost constraint) and as soon as possible after pasteurization. Prepasteurization microbiology can result in wastage of milk to the tune of 30% in some cases. Even after pasteurization, the endotoxins of organisms are still present in the milk in some cases but they have not been found to have any clinical effect on the baby. A bacterial count of 10^5 or more in raw breast milk can be considered as an indicator of the milk’s poor quality. Based on this and on the theoretical concern that heavily contaminated milk with specific bacteria (e.g. Staphylococcus aureus, Escherichia coli) may contain enterotoxins and thermostable enzymes even after pasteurization, expert panel selected 10^5 CFU/mL for total bacterial count, 10^4 CFU/mL for Enterobacteriaceae and S. aureus as threshold values which are in consonance with milk banks operating in other parts of the world.

No growth is acceptable in postpasteurization microbiology cultures. Whole pasteurized batch of culture positive container of processed milk should be discarded. Before pasteurization, pooling and mixing may be carried out, from multiple donors to ease the process of processing and storage.

Storage

Pasteurized milk awaiting culture report should be kept in separate freezer area taking precaution not to disburse it till the culture is negative. Culture negative processed milk should be kept at –20°C in tightly sealed container with clear mention of expiry date and other relevant data on the label. It can be preserved for 4–6 months. Random cultures of preserved milk before disbursal can aid quality assurance.

Disbursal

Pasteurized donor human milk should be disbursed on requisition from NICU after informed consent from recipient’s parents. It should be done on “First-in-first-out” basis from the storage.

Frozen PDHM should be thawed by either defrosting the milk rapidly in a water bath at a temperature not exceeding 37°C or under running lukewarm water. It should never be thawed in the microwave as this result in reduction in the immunoglobulin A (IgA) content of the milk. Milk should not be refrozen after being thawed as this increases the hydrolysis of the triglycerides in the milk. While bringing to room temperature, it should be gently agitated to make a homogeneous mixture before use and be used preferably within 3–4 hours to prevent contamination. Preterm baby should preferably get PDHM from preterm donors.

Labeling and Record Keeping

Proper labeling at all levels is mandatory, from sterile container for collection of donation, pooling vessel, pasteurization container to storage containers. Labels should be water resistant and read clearly names and identification number of donors and dates of pasteurization batch numbers. Record keeping at all levels should be meticulous for donor consent form, donor’s data and screening reports, pasteurization batch files; and PDHM disbursal record file containing relevant data including recipient consent form.

As incubation period for most infection varies from a few weeks to six months and appearance of symptoms is faster in infants and children, there seems to be no rationale for
keeping records beyond five years, unless one is working in an area where milk kinship issue is of paramount importance. In India, the bloodbank records are preserved for a minimum period of 5 years.

**Economic Implications**

There has been a definite cost effectiveness of using banked human milk in NICUs observed in western countries largely by reduction in the rate of NEC. In a country like ours, the cost of running a milk bank with potential cost saving due to reduction in NEC and sepsis rate and ultimately, duration of hospital stay have not been adequately evaluated, but given the high incidence of sepsis and a large burden of premature births, this intervention is likely to result in substantial saving for the nation.

**CONCLUSION**

It is clear that artificial formula will never provide the broad range of benefits of human milk. Given the high rate of preterm births in the country and level of malnutrition that ensues in the postnatal growth in such babies after birth, there is an urgent need for establishing milk banks across the country, especially in the large NICUs of all hospitals. The author is of the opinion that human milk banks be opened in all 676 districts of India to reduce IMR and under-5 mortality. It only requires a pediatrician and pathologist and very little budget.

**REFERENCES**