Source : *(80)*

The food guide pyramid

(f) protein should account for approximately 10–15 per cent of the daily intake;

(g) junk foods such as colas, ketchups and other foods that supply empty calories should be reduced.

There may be conditions under which the above recommendations for daily food intake do not apply. For example, diet should be adapted to the special needs of growth, pregnancy, lactation, physical activity, and medical disorders (e.g., diabetes).

NUTRITIONAL PROBLEMS IN PUBLIC HEALTH

There are many nutritional problems which affect vast segments of our population. The major ones which deserve special mention are highlighted :

1. Low birth weight

Low birth weight (i.e., birth weight less than 2500 g) is a major public health problem in many developing countries. About 28 per cent of babies born in India are LBW *(81)* as compared to 4 per cent in some developed countries. In countries where the proportion of LBW is high, the majority are suffering from foetal growth retardation. In countries where the proportion of LBW infants is low, most of them are preterm *(82)*. Although we do not know all the causes of LBW, maternal malnutrition and anaemia appear to be significant risk factors in its occurrence. Among the other causes of LBW are hard physical labour during pregnancy, and illnesses especially infections. Short maternal stature, very young age, high parity, smoking, close birth intervals are all associated factors. All these factors are interrelated.

Since the problem is multifactorial, there is no universal solution. Interventions have to be cause-specific. This matter has already been discussed in Chapter 9.

The proportion of infants born with LBW was selected as one of the nutritional indicators for monitoring progress towards Health for All by the year 2000.

2. Protein energy malnutrition

Protein energy malnutrition (PEM) is identified as a major health and nutrition problem in India. It occurs particularly in weaklings and children in the first years of life. It is not only an important cause of childhood morbidity and mortality, but leads also to permanent impairment of physical and possibly, of mental growth of those who survive *(9)*. The current concept of PEM is that its clinical forms – kwashiorkor and marasmus – are two different clinical pictures at opposite poles of a single continuum.

The incidence of PEM in India in pre-school age children is 1–2 per cent *(83)*. The great majority of cases of PEM, nearly 80 per cent, are the "intermediate" ones, that is the mild and moderate cases which frequently go unrecognized. The problem exists in all the States and that nutritional marasmus is more frequent than kwashiorkor.

In the 1970s, it was widely held that PEM was due to protein deficiency. Over the years, the concept of "protein gap" has given place to the concept of "food gap". That is, PEM is primarily due to (a) an inadequate intake of food (food gap) both in quantity and quality, and (b) infections, notably diarrhoea, respiratory infections, measles and intestinal worms which increase requirements for calories, protein and other nutrients, while decreasing their absorption and utilization. It is a vicious circle – infection contributing to malnutrition and malnutrition contributing to infection, both acting synergistically (Fig. 2).

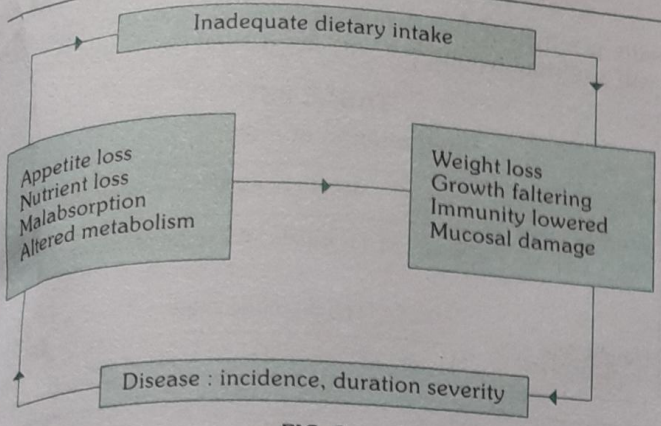

FIG. 2
Malnutrition/Infection cycle

Source : *(68)*

There are numerous other contributory factors in the web of causation, viz. poor environmental conditions, large family size, poor maternal health, failure of lactation, premature termination of breast-feeding, and adverse cultural practices relating to child rearing and weaning such as the use of over–diluted cow's milk and discarding cooking water from cereals and delayed supplementary feeding *(84)*.

Malnutrition is self-perpetuating. A child's nutritional status at any point of time depends on his or her past nutritional history, which may particularly account for the present status. To some extent, this nutritional history is linked to the mother's health and nutritional status. This in turn has been influenced by her living conditions and nutritional history during her own childhood (Fig. 3).

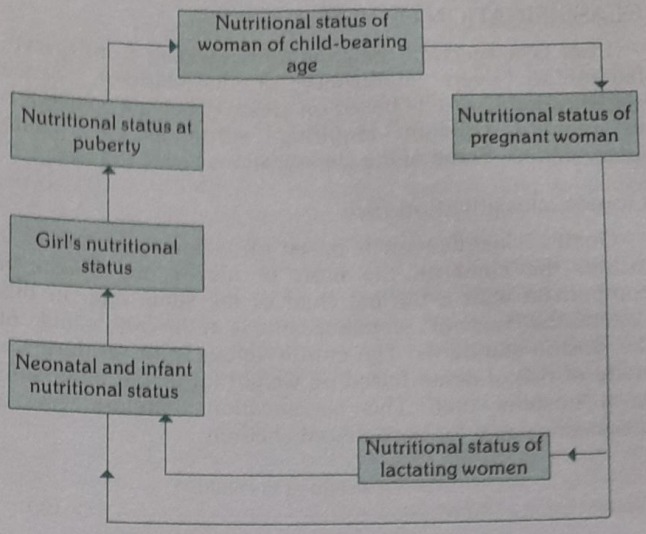

FIG. 3
Influence of each generation's nutritional status on the following generation.

Source : *(68)*

Early detection of PEM

The first indicator of PEM is under-weight for age. The most practical method to detect this, which can be employed even by field health workers, is to maintain growth charts. These charts indicate at a glance whether the child is gaining or losing weight.

The principal features of kwashiorkor and marasmus are shown in Table 31.

TABLE 31

Principal features of severe PEM

FEATURES	MARASMUS	KWASHIORKOR
CLINICAL	*ALWAYS PRESENT*	
Muscle wasting	Obvious	Sometimes hidden by oedema and fat
Fat wasting	Severe loss of subcutaneous fat	Fat often retained but not firm
Oedema	None	Present in lower legs, and usually in face and fore arms
Weight for height	Very low	Low but may be masked by oedema
Mental changes	Sometimes quiet and apathetic	Irritable, moaning, apathetic
CLINICAL	*SOMETIMES PRESENT*	
Appetite	Usually good	Poor
Diarrhoea	Often (current and past)	Often (current and past)
Skin changes	Usually none	Diffuse pigmentation, sometimes 'flaky paint dermatosis'
Hair changes	Seldom	Sparse, silky, easily pulled out
Hepatic enlargement	None	Sometimes, due to accumulation of fat
BIOCHEMICAL		
Serum albumin	Normal or slightly decreased	Low (<3 g/100 ml blood)
Urinary urea per g creatinine	Normal or decreased	Low
Hydroxyproline/creatinine ratio	Low	Low
Plasma/amino acid ratio	Normal	Elevated

Source : *(85)*

CLASSIFICATION OF PEM

PEM is a spectrum of conditions ranging from growth failure to overt marasmus or kwashiorkor, hence classification has to be based on arbitrary cut-off-points. It is to identify children requiring nutritional or health interventions. Some of the classifications are as follows :

Gomez' classification *(68)*

Gomez' classification is based on weight retardation. It locates the child on the basis of his or her weight in comparison with a normal child of the same age. In this system, the "normal" reference child is in the 50th centile of the Boston standards. The cut-off values were set during a study of risk of death based on weight for age at admission to a hospital unit. This classification therefore, has a prognostic value for hospitalized children.

$$\text{Weight for age (\%)} = \frac{\text{weight of the child}}{\text{weight of a normal child of same age}} \times 100$$

Between 90 and 110%	:	normal nutritional status
Between 75 and 89%	:	1st degree, mild malnutrition
Between 60 and 74%	:	2nd degree, moderate malnutrition
Under 60%	:	3rd degree, severe malnutrition

Weight is widely recorded and the classification is easy to compute. The disadvantages are (a) A cut-off-point of 90 per cent of reference is high *(80* per cent being approximately equivalent to –2SD or the 3rd percentile), thus some normal children may be classified as 1st degree malnourished. (b) By measuring only weight for age it is difficult to know if the low weight is due to a sudden acute episode of malnutrition or to long-standing chronic undernutrition *(85)*.

Waterlow's classification *(68)*

When a child's age is known, measurement of weight enables almost instant monitoring of growth : measurements of height assess the effect of nutritional status on long-term growth. Table 32 shows the Waterlow's classification.

TABLE 32

Waterlow's classification

H/A \ W/H	> m – 2 SD	< m – 2 SD
> m – 2 SD	Normal	Wasted
< m – 2 SD	Stunted	Wasted and stunted

m = mean, SD = standard deviation

Source : *(68)*

Waterlow's classification defines two groups for protein energy malnutrition :

- malnutrition with retarded growth, in which a drop in the height/age ratio points to a chronic condition– shortness, or stunting :
- malnutrition with a low weight for a normal height, in which the weight for height ratio is indicative of an acute condition of rapid weight loss, or wasting.

This combination of indicators makes it possible to label and classify individuals with reference to two poles : children with insufficient but well-proportioned growth and those with a normal height, but who are wasted (Table 33)

TABLE 33

Interpretation of indicators

$$\text{Weight/Height (\%)} = \frac{\text{Weight of the child}}{\text{Weight of a normal child at same height}} \times 100$$

$$\text{Height/Age (\%)} = \frac{\text{Height of the child}}{\text{Height of a normal child at same age}} \times 100$$

Nutritional status	Stunting (% of height/age)	Wasting (% of weight/height)
Normal	> 95	> 90
Mildly impaired	87.5 – 95	80 – 90
Moderately impaired	80 – 87.5	70 – 80
Severely impaired	< 80	< 70

Source : *(68)*

Arm circumference : *(68)*

Arm circumference yields a relatively reliable estimation of the body's muscle mass, the reduction of which is one of the most striking mechanisms by which the body adjusts to inadequate energy intakes. Arm circumference cannot be used before the age of one year; between ages one and five years, it hardly varies.

An arm circumference exceeding 13.5 cm is a sign of a satisfactory nutritional status, between 12.5 and 13.5 cm it indicates mild–moderate malnutrition and below 12.5 cm, severe malnutrition.

For the purpose of comparison, growth charts are provided with reference curves. These curves show the limit of normal growth. The WHO reference curves are based on extensive cross-sectional data of well-nourished healthy children, assembled by the National Centre for Health Statistics (NCHS). Malnutrition is defined by WHO as a weight-for-age below the median minus two standard deviations of the NCHS reference population *(86)*.

Preventive measures

There is no simple solution to the problem of PEM. Many types of actions are necessary. The following is adapted from the 8th FAO/WHO Expert Committee on Nutrition *(1)* for the prevention of PEM in the community :

(a) *Health promotion*

1. Measures directed to pregnant and lactating women (education, distribution of supplements).
2. Promotion of breast-feeding.
3. Development of low cost weaning foods : the child should be made to eat more food at frequent intervals.
4. Measures to improve family diet.
5. Nutrition education – Promotion of correct feeding practices.
6. Home economics.
7. Family planning and spacing of births.
8. Family environment.

(b) *Specific protection*

1. The child's diet must contain protein and energyrich foods. Milk, eggs, fresh fruits should be given if possible.
2. Immunization.
3. Food fortification.

(c) *Early diagnosis and treatment*

1. Periodic surveillance.
2. Early diagnosis of any lag in growth.
3. Early diagnosis and treatment of infections and diarrhoea.
4. Development of programmes for early rehydration of children with diarrhoea.
5. Development of supplementary feeding programmes during epidemics.
6. Deworming of heavily infested children.

(d) *Rehabilitation*

1. Nutritional rehabilitation services.
2. Hospital treatment.
3. Follow-up care.

3. Xerophthalmia

Xerophthalmia (dry eye) refers to all the ocular manifestations of vitamin A deficiency in man. It is the most widespread and serious nutritional disorder leading to blindness *(87)* particularly in South-East Asia.

Xerophthalmia is most common in children aged 1–3 years, and is often related to weaning. The younger the child, the more severe the disease. It is often associated with PEM. Mortality is often high in this age group *(21)*. The victims belong to the poorest families. Associated risk factors include ignorance, faulty feeding practices and infections particularly diarrhoea and measles which often precipitate xerophthalmia. In some countries, "epidemics" of xerophthalmia have occurred in association with food donation programmes involving skimmed milk, which is totally devoid of vitamin A *(88)*.

The States badly affected are the southern and eastern States of India notably Andhra, Tamil Nadu, Karnataka, Bihar and West Bengal. These are predominantly rice-eating States and rice is devoid of carotene. The North Indian States have relatively few cases of xerophthalmia *(89)*.

Prevention and control

Prevention and control of xerophthalmia must be an integral part of primary health care. An overall strategy can be defined, according to WHO, in terms of short-term, medium-term and long-term action *(24)*.

(a) *Short-term action :* A short-term preventive approach that has already demonstrated its efficacy is the administration of large doses of vitamin A orally, in recommended doses to vulnerable groups, on a periodic basis. This can be organized quickly and with a minimum of infrastructure.

(b) *Medium-term action :* An approach widely used to promote regular and adequate intake of vitamin A is **fortification** of certain foods with vitamin A. Addition of vitamin A to *dalda* in India is a typical example. Many other foods have also been considered for vitamin A fortification, viz. sugar, salt, tea, margarine and dried skimmed milk. Fortifying an appropriate food with vitamin A is a complex process. The greatest challenge to successful fortification programmes is choosing a food that is likely to be consumed in sufficient quantities by groups at risk *(90)*.

(c) *Long-term action :* These are measures aimed at reduction or elimination of factors contributing to ocular disease, e.g., persuading people in general, and mothers in particular, to consume generously dark green leafy vegetables or other vitamin A rich foods; promotion of breast-feeding for as long as possible; improvements in environmental health such as ensuring safe and adequate water supply and construction and maintenance of sanitary latrines to safeguard against diarrhoea; immunization against infectious diseases such as measles, prompt treatment of diarrhoea and other associated infections; better feeding of infants and young children; improved health services for mothers and children; social and health education. All these are components of primary health care.

Vitamin A deficiency in India (VAD) *(91)*

VAD has been recognized as a major controllable public health and nutritional problem in India. An estimated 5.7 per cent children in India suffer from eye signs of VAD. Recent evidence suggests that even mild VAD probably increases morbidity and mortality in children, emphasizing the public health importance of this disorder. VAD is one of the major deficiencies among lower income strata population in India.

Though the prevalence of severe forms of VAD such as corneal ulcers/softening of cornea i.e. keratomalacia has in general become rare, Bitot spots were present in varying magnitudes in different parts of the country as reported b National Nutritional Monitoring Bureau in 2003. Th prevalence was higher than WHO cut-off level of 0.5 p cent, indicating the public health significance of the proble of VAD. There is huge inter-state variation in the prevalen of VAD among children. It is also a matter of concern th only 21 per cent of children of age 12 to 35 months rece a vitamin A dose. Less than 10 per cent coverage reported in Nagaland, Uttar Pradesh reported 7.3 per coverage. Only states such as Tamil Nadu (37.2 per ce Goa (37.3 per cent), Kerala (38.2 per cent) and West Be (41.2 per cent) have better coverage, though it is still lo

In India, in 1970 a national programme for preventi nutritional blindness was initiated to fight this defici The beneficiaries of this programme were pre-s children (1–5 years). The programme was modified in to cover children in age group of nine months to three only. Since Tenth Five Year Plan vitamin A supplemen exists as an integral component of RCH prog which is now a part of NRHM. The guidelines is November 2006 cover children upto 5 years of age.

The programme focusses on *(92)*: (a) Pr consumption of vitamin A rich foods by pregnant and women and by children under-five years of appropriate breast-feeding; (b) Administration o dose of vitamin A up to five years. First dose of 1 with measles vaccination at nine months and s doses of 200,000 IU each, every six months up 5 years; (c) For sick children – all children with xer to be treated at health facilities; all children suf measles to be given one dose of vitamin A if th received it in the previous one month; all case malnutrition to be given one additional dose of vit

4. Nutritional anaemia

Nutritional anaemia is a disease syndrome caused by malnutrition in its widest sense *(54)*. It has been defined by WHO as "a condition in which the haemoglobin content of blood is lower than normal as a result of a deficiency of one or more essential nutrients, regardless of the cause of such deficiency" *(52)*. Anaemia is established if the haemoglobin is below the cut-off points recommended by WHO (Table 13). By far the most frequent cause of nutritional anaemia is iron deficiency, and less frequently folate or vitamin B_{12} *(49)*.

The problem

WORLD

Nutritional anaemia is a worldwide problem with the highest prevalence in developing countries. It is found especially among women of child-bearing age, young children and during pregnancy and lactation. It is estimated to affect nearly two-thirds of pregnant and one-half of non-pregnant women in developing countries *(93)*. The populations of developed countries are not by any means completely free of anaemia, and a significant percentage of women of child-bearing age (estimated between 4 and 12 per cent) suffer from anaemia *(94)*.

INDIA

Iron deficiency anaemia is the most widespread micronutrient deficiency affecting all age groups irrespective of gender, cast, creed and religion. In India, this silent emergency is rampant among women belonging to reproductive age group (15–49 years), children (6–35 months) and low socio-economic strata of the population. Overall, 72.7 per cent of children up to the age 3 years in urban areas and 81.2 per cent in rural areas are anaemic.

While analyzing the data for states with anaemia level of 70% among children, it was found that, except for Punjab, all other states had more than 50% prevalence of anaemia among pregnant women. This again reiterates the strong relationship between anaemia levels of mothers and children. Also, the overall prevalence has increased from 74.2% (1998–99) to 79.2% (2005–06). Nagaland had the lowest prevalence (44.3%), Goa was next (49.3%) followed by Mizoram (51.7%). Bihar had the highest prevalence (87.6%) followed closely by Rajasthan (85.1%), and Karnataka (82.7%). Moderate and severe anaemia is seen even among the educated families both in urban and rural areas. There are inter-state differences in prevalence of anaemia that are perhaps attributable partly to differences in dietary intake and partly to access to health care *(91)*.

As per District Level Health Survey (DLHS) (2002–04), prevalence of anaemia in adolescent girls is very high (72.6%) in India, with prevalence of severe anaemia among them much higher (21.1%) than that in preschool children (2.1%). In adolscent girls, educational or economic status does not seem to make much of a difference in terms of prevalence of anaemia. Prevention, detection, or management of anaemia in adolescent girls has till now not received much attention. In view of the high prevalence of moderate and severe anaemia in this group and the fact that many of them get married early, conceive, and face the problems associated with anaemia in pregnancy, it is imperative to screen them for anaemia and treat them.

Iron deficiency can arise either due to inadequate intake or poor bioavailability of dietary iron or due to excessive losses of iron from the body. Although most habitual diets contain seemingly adequate amounts of iron, only a [illegible] amount (less than 5 per cent) is absorbed (96). This [illegible] bio-availability is considered to be a major reason for [illegible] widespread iron deficiency *(95)*. Women lose a considerable amount of iron especially during menstruation. Some of [illegible] other factors leading to anaemia are malaria and hookworm infestations. In addition mothers who have born children at close intervals become anaemic due to the additional demands of the rapid pregnancies and the loss of blood at each delivery.

Megaloblastic anaemia is not encounterd frequently in general population, but it occurs occasionally in pregnant women from poor income groups. It is possible that [illegible] widespread iron deficiency (microcytic anaemia) could mask megaloblastic anaemia. In a recent study, sub-clinical folate deficiency was found to be about 30 per cent in pregnant women from rural North India. A high level of sub-clinical folate deficiency was also reported in semi-urban school children. There are some sporadic reports indicating its prevalence in adults *(9)*.

Detrimental effects

The detrimental effects of anaemia can be seen in three important areas *(49)* : (a) *Pregnancy* : Anaemia increases the risk of maternal and foetal mortality and morbidity. In India, 19 per cent of maternal deaths were found to be due to anaemia *(54)*. Conditions such as abortions, premature births, postpartum haemorrhage and low birth weight are especially associated with low haemoglobin levels in pregnancy. (b) *Infection* : Anaemia can be caused or aggravated by parasitic diseases, e.g., malaria, intestinal parasites. Further, iron deficiency may impair cellular responses and immune functions and increase susceptibility to infection (c) *Work capacity* : Anaemia (even when mild) causes a significant impairment of maximal work capacity. The more severe the anaemia, the greater the reduction in work performance, and thereby productivity. This has great significance on the economy of the country.

Interventions

An estimation of haemoglobin should be done to assess the degree of anaemia. If the anaemia is "Severe", less than 10 g/dl high doses of iron or blood transfusion may be necessary. If haemoglobin is between 10–12 g/dl, the [illegible] interventions are :

(1) Iron and folic acid supplementation

In order to prevent nutritional anaemia among mothers and children, the Government of India sponsored a National Nutritional Anaemia Prophylaxis Programme during the Fourth Five Year Plan. The Programme is based on [illegible] supplementation with iron and folic acid tablets to prevent mild and moderate cases of anaemia. The beneficiaries are "at risk" groups viz pregnant women, lactating mothers and children under 12 years.

Eligibility criteria (97) : These are determined by the haemoglobin levels of the patients. If the haemoglobin is between 10 and 12, daily supplement with iron and folic acid tablets is advised; if it is less than 10 g, the patient is referred to the nearest primary health centre.

Dosage : (a) MOTHERS : One tablet of iron and folic acid containing 100 mg of elemental iron (300 mg of ferrous sulphate) and 0.5 mg of folic acid should be given daily. The daily administration should be continued until 2 to 3 months after haemoglobin level has returned to normal so that iron

stores are replenished. It is necessary that estimation of haemoglobin is repeated at 3–4 month intervals. The exact period of supplementation will depend upon the progress of the beneficiary. (b) CHILDREN : If anaemia is suspected, a screening test for anaemia may be done on infants at 6 months, and 1 and 2 years of age. One tablet of iron and folic acid containing 20 mg of elemental iron (60 mg of ferrous sulphate) and 0.1 mg of folic acid should be given daily for 100 days. For children 6–60 months, ferrous sulphate and folic acid is to be provided in a liquid formulation. For safety sake, the liquid formulation should be dispensesd in bottles so designed that only 1 ml can be dispensed each time. School children, 6 to 10 year old and adolescents are also to be included in the national programme. Children 6-10 years of age are to be provided 30 mg. elemental iron and 250 mcg. folic acid per day for 100 days. Adolescents are given the same dosage and duration as adults *(92)*.

(2) Iron fortification

The WHO experts *(49)* did not recommend iron fortification strategy for control of anaemia in regions where its prevalence is high. However, studies in India at the National Institute of Nutrition, Hyderabad showed that simple addition of ferric ortho-phosphate or ferrous sulphate with sodium bisulphate was enough to fortify salt with iron *(98)*. When consumed over a period of 12–18 months, iron fortified salt was found to reduce prevalence of anaemia significantly. Fortification of salt with iron has been accepted by the Government of India as a public health approach to reduce prevalence of anaemia. Commercial production of iron fortified salt was started in 1985 *(98)*.

Iron fortification has many advantages over iron supplementation. As salt is a universally consumed dietary item, all segments of the population stand to benefit. No special delivery systems are required *(53)*.

(3) Other strategies

There are other strategies such as changing dietary habits, control of parasites and nutrition education. These are longterm measures applicable to situations where the prevalence and severity of anaemia are lower. Cost and time involved to meet the desired goals through these strategies are disproportionately high *(53)*.

5. Iodine deficiency disorders (IDD)

Iodine deficiency is yet another major nutrition problem in India. Previously, iodine deficiency was equated with goitre. In recent years, it has become increasingly clear that iodine deficiency leads to a much wider spectrum of disorders commencing with the intrauterine life and extending through childhood to adult life with serious health and social implications. Table 16 presents the iodine deficiency disorders in approximate order of increasing severity. The social impact of iodine deficiency arises not so much from goitre as from the effect on the central nervous system *(56)*.

The problem

Whereas goitre has ceased to be a major problem in many developed countries (although not eradicated) it continues to be a serious health problem in many Third World Countries. For example iodine deficiency is a health problem of considerable magnitude in India and the neighbouring countries of Bangladesh, Bhutan, Myanmar, Indonesia, Nepal, Sri Lanka and Thailand. More people are affected and levels of severity are higher in South-East Asia than anywhere else in the world *(99)*.

It has always been thought in India that goitre and cretinism were only found to a significant extent in the "Himalaya goitre belt" which is the world's biggest goitre belt. It stretches from Kashmir to the Naga Hills in the east, extending about 2,400 km and affecting the northern States of Jammu and Kashmir, Himachal Pradesh, Punjab, Haryana, Delhi, Uttar Pradesh, Bihar, West Bengal, Sikkim, Assam, Arunachal Pradesh, Nagaland, Mizoram, Meghalaya, Tripura and Manipur. In recent years renewed surveys outside the conventional goitre belt have identified endemic foci of iodine deficiency and the associated IDD in parts of Madhya Pradesh, Gujarat, Maharashtra, Andhra Pradesh, Kerala, Karnataka and Tamil Nadu. More and more new areas are being identified. Even areas near the sea coast like Bharuch district in Gujarat and Ernakulam district in Kerala are found goitre-affected. In short, no State in India can be said to be entirely free from goitre (Fig. 4).

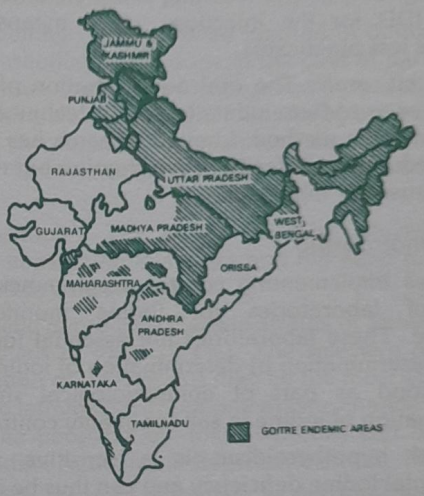

FIG. 4
Goitre endemic areas in India

The magnitude of the problem in India is far greater than what had been estimated in 1960s, when it was estimated that about 9 million persons were affected by goitre. Results of sample surveys conducted in 325 districts covering all the states/UTs have revealed that 263 districts are endemic where the prevalence of IDD is more than 10 per cent. It is estimated that more than 71 million persons are suffering from goitre and other iodine deficiency disorders in the country *(100)*.

Goitre control

There are four essential components of national goitre control programme. These are iodized salt or oil, monitoring and surveillance, manpower training and mass communication.

1. Iodized salt

The iodization of salt is now the most widely used prophylactic public health measure against endemic goitre. In India the level of iodization is fixed under the Prevention of Food Adulteration (PFA) Act and is not less than 30 ppm at the production point, and not less than 15 ppm of iodine at the consumer level *(98)*. Iodized salt is most economical, convenient and effective means of mass prophylaxis in

endemic areas. Under the national IDD control activities, the Government of India proposed to completely replace common salt with iodized salt in a phased manner *(98)*.

The National Institute of Nutrition at Hyderabad has come out with a new product, common salt fortified with iron and iodine. Community trials have been launched to examine the efficacy of the "two-in-one" salt *(98)*.

Iodized oil : Another method which has demonstrated its efficacy for controlling goitre is intramuscular injection of iodized oil (mostly poppy-seed oil). Scientists at the National Institute of Nutrition, Hyderabad have now successfully developed a process to produce iodized oil in safflower or safola oil *(101)*.

The advantage of the injection procedure is that an average dose of 1 ml will provide protection for about 4 years. Although more expensive than iodated salt, this method has the advantage that it can be applied rapidly and in places where iodization of salt is not feasible or iodated salt is in short supply. However, the difficulty with this procedure is one of logistics, i.e., in reaching every victim or potential victim of IDD for the injection, which means that this approach is less practicable.

Iodized oil, oral : The oral administration of iodine as iodized oil or as sodium iodate tablets, is technically simpler than the injection method. Limited research has found that these procedures are effective against goitre but more costly than intramuscular injections.

2. Iodine monitoring

Countries implementing control programmes require a network of laboratories for iodine monitoring and surveillance. These laboratories are essential for a) iodine excretion determination b) determination of iodine in water, soil and food as part of epidemiological studies, and c) determination of iodine in salt for quality control.

Neonatal hypothyroidism is a sensitive pointer to environmental iodine deficiency and can thus be an effective indicator for monitoring the impact of a programme *(99)*.

3. Manpower training

It is vital for the success of control that health workers and others engaged in the programme be fully trained in all aspects of goitre control including legal enforcement and public education.

4. Mass communication

Mass communication is a powerful tool for nutrition education. It should be fully used in goitre control work. Creation of public awareness is central issue of a successful public health programme.

5. Hazards of iodization

A mild increase in incidence of thyrotoxicosis has now been described following iodized salt programmes. An increase in lymphocytic thyroiditis (Hashimoto's disease) has also been claimed. The risk of iodism or iodide goitre however seems to be very small *(58)*.

6. Endemic fluorosis

In many parts of the world where drinking water contains excessive amounts of fluorine (3–5 mg/L), endemic fluorosis has been observed. Endemic fluorosis has been reported to be an important health problem in certain parts of the country, e.g., Andhra Pradesh (Nellore, Nalgonda and Prakasam districts), Punjab, Haryana, Karnataka, Kerala and Tamil Nadu *(60)*. The toxic manifestation of fluorosis comprise the following :

(a) *Dental fluorosis* : Fluorosis of dental enamel occurs when excess fluoride is ingested during the years of tooth calcification – essentially during the first 7 years of life *(101)*. It is characterized by "mottling" of dental enamel which has been reported at levels above 1.5 mg/L intake *(102)*. The teeth lose their shiny appearance and chalky white patches develop on them. This is the early sign of dental fluorosis. Later the white patches become yellow and sometimes brown or black. In severe cases, loss of enamel gives the teeth a corroded appearance. Mottling is best seen on the incisors of the upper jaw. It is almost entirely confined to the permanent teeth and develops only during the period of formation *(61)*.

(b) *Skeletal fluorosis* : This is associated with lifetime daily intake of 3.0 to 6.0 mg/L or more *(102)*. There is heavy fluoride deposition in the skeleton. When a concentration of 10 mg/L is exceeded, crippling fluorosis can ensue *(103)*. It leads to permanent disability.

(c) *Genu valgum* : A new form of fluorosis characterized by genu valgum and osteoporosis of the lower limbs has been reported in some districts of Andhra Pradesh and Tamil Nadu *(104)*. The syndrome was observed among people whose staple was sorghum *(jowar)*. Further studies showed that diets based on sorghum promoted a higher retention of ingested fluoride than do diets based on rice *(14)*.

Intervention

(a) *Changing the water source* : One solution to the problem is to find a new source of drinking water with a lower fluoride content (0.5 to 0.8 mg/L) if that is possible. Running surface water contains lower quantities of fluorides than ground water sources such as wells. (b) *Chemical treatment* : If the above is not possible, the water can be chemically defluoridated in a water treatment plant, even though such treatment is moderately expensive *(101)*. The National Environmental Engineering Research Institute, Nagpur developed a technique for removing fluoride by chemical treatment. It is called **Nalgonda technique** for defluoridation of water *(105)*. It involves the addition of two chemicals (viz. lime and alum) in sequence followed by flocculation, sedimentation and filtration. (c) *Other measures* : Fluoride supplements should not be prescribed for children who drink fluoridated water. The use of fluoride toothpaste in areas of endemic fluorosis is not recommended for children upto 6 years of age *(101)*.

7. Lathyrism

Lathyrism is a paralyzing disease of humans and animals. In the humans it is referred to as **neurolathyrism** because it affects the nervous system, and in animals as **osteolathyrism** (odoratism) because the pathological changes occur in the bones resulting in skeletal deformities *(8)*. Neurolathyrism is a crippling disease of the nervous system characterized by gradually developing spastic paralysis of lower limbs, occurring mostly in adults consuming the pulse, Lathyrus sativus in large quantities.

The problem

Neurolathyrism is prevalent in parts of Madhya Pradesh, Uttar Pradesh, Bihar and Orissa. It has also been reported in Maharashtra, West Bengal, Rajasthan, Assam and Gujarat where the pulse is grown. The magnitude of the problem can

be assessed from the fact that at one time in Rewa and Satna districts of Madhya Pradesh alone, there were 25,000 and 32,000 cases respectively. According to reports, there are no fresh outbreaks of the disease in endemic areas. This is attributed to the shifting trends in agronomical practices in the region *(106)*. Lathyrism has also been reported to occur in Spain and Algeria where *Lathyrus* is eaten *(8)*.

The pulse

Lathyrus sativus is commonly known as "Khesari dhal". It is known by local names such as Teora dhal, Lak dhal, Batra, Gharas, Matra etc. *(106)*. The seeds of lathyrus have a characteristic triangular shape and grey colour. When dehusked the pulse looks similar to red gram dhal or bengal gram dhal. Like other pulses, lathyrus is a good source of protein, but for its toxin which affects the nerves. It is eaten mostly by the poor agricultural labourer because it is relatively cheap. Studies have shown that diets containing over 30% of this dhal if taken over a period of 2–6 months will result in neurolathyrism.

The toxin

The toxin present in lathyrus seeds has been identified as Beta oxalyl amino alanine (BOAA). It has been isolated in crystalline form and is water soluble; this property has been made use of in removing the toxin from the pulse by soaking it in hot water and rejecting the soak water. Studies indicate that there is a blood-brain barrier to this toxin. In order to overcome this barrier, the pulse must be eaten in large amounts over a period of time for 2 months or more. Besides BOAA several other toxins have also been reported *(107)*.

The disease

The disease affects mainly young men between the age of 15 to 45 years and manifests itself in stages : (a) *Latent stage* : The individual is apparently healthy, but when subjected to physical stress exhibits ungainly gait. Neurological examination shows characteristic physical signs. This stage is considered important from the preventive aspect, since at this stage, if the pulse is withdrawn from the diet, it will result in complete remission of the disease. (b) *No-stick stage* : the patient walks with short jerky steps without the aid of a stick. A large number of patients are found in this stage. (c) *One-stick stage* : The patient walks with a crossed gait with a tendency to walk on toes. Muscular stiffness makes it necessary to use a stick to maintain balance. (d) *Two-stick stage* : the symptoms are more severe. Due to excessive bending of knees and crossed legs, the patient needs two crutches for support. The gait is slow and clumsy and the patient gets tired easily after walking a short distance. (e) *Crawler stage* : Finally the erect posture becomes impossible as the knee joints cannot support the weight of the body. There is atrophy of the thigh and leg muscles. The patient is reduced to crawling by throwing his weight on his hands *(106)*.

Interventions

The possible interventions for the prevention and/or control of lathyrism are :

(a) *Vitamin C prophylaxis* : Although this condition is believed to be irreversible, in certain instances the damages could be repaired by the daily administration of 500–1000 mg of ascorbic acid for a week or so. The damage could also be prevented by generous provision of ascorbic acid in the lathyrogenic diet, as demonstrated in guinea pigs and monkeys.

(b) *Banning the crop* : This is an extreme step not feasible for immediate implementation. The Prevention of Food Adulteration Act in India has banned *lathyrus* in all forms – whole, split or flour. But the ban is not operative where it is needed, viz. Madhya Pradesh, Bihar, Orissa and Gujarat where the pulse is widely grown.

If however, it is not possible to avoid consuming khesari dhal, it is desirable that the proportion of the *dhal* should never form more than a quarter of the total amount of cereals and pulses eaten per day.

(c) Removal of toxin

(1) *Steeping method* : Since the toxins are water soluble, they can be removed by soaking the pulse in hot water. This method can be practised at home. A large quantity of water is boiled and the pulse is soaked in hot water for 2 hours; after which the soaked water is drained off completely. The pulse is washed again with clean water, then drained off and dried in the sun. The pulse is then used for consumption. The drawback with this method is that it entails loss of vitamins and minerals.

(2) *Parboiling* : An improved method of detoxicating the pulse is "parboiling" as is done in the case of parboiled rice. This technique is suitable for large scale operation. Simple soaking in lime water overnight followed by boiling is credited to destroy the toxin. This treatment also destroys trypsin inhibitiors. Lime is easily available as it is used with betel leaves.

(c) *Education* : The public must be educated on the dangers of consuming this pulse and the need for removing its toxin before consumption.

(d) *Genetic approach* : Certain strains of *lathyrus* contain very low levels of toxin (0.1%). The selective propagation and cultivation of such strains may be the most effective way to eradicate lathyrism without any drastic change in the food habits of the people. Low toxin varieties can be obtained from the Indian Agricultural Research Institute, New Delhi.

(e) *Socio-economic changes* : In the final analysis, it is only socio-economic changes or overall development that can root out lathyrism.

NUTRITIONAL FACTORS IN SELECTED DISEASES

1. Cardiovascular disease

It is now generally agreed that diet governs many situations favouring the onset of "heart disease", particularly coronary heart disease. Of all the factors associated with CHD (e.g., plasma cholesterol, high blood pressure, cigarette smoking, lack of physical activity) plasma cholesterol has a very high statistical significance with the incidence of CHD. The risk of CHD appears to increase as the plasma cholesterol concentration rises *(108)*. Various studies have supported the role of elevated blood levels of cholesterol and low density lipoproteins (LDL) in the development of atherosclerosis. Geographical studies have shown that there is no population in whom CHD is common that does not have a relatively high mean level of plasma total cholesterol (TC) in adults *(15)*. These observations have been reinforced by metabolic studies. In addition trials of the effect of dietary changes on CHD have suggested that altering the fatty acid composition of the diet in favour of greater intake of polyunsaturated fatty acids (PUFA) and less intake of saturated fats, while restricting the intake of fat