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Graduate Course

Elements of Public Health Nutrition

Contents

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Lesson 1

HEALTH CARE SERVICES I

PUBLIC HEALTH

The term "public health" came into general use around 1840. It arose from the need to protect "the public" from the spread of communicable diseases. Later, it appeared in 1848 in the name of a law, the Public Health Act in England to crystallize the efforts organized by society to protect, promote and restore the people's health.

In 1920, C.E.A. Winslow, a former professor of public health at Yale University, gave the oft-quoted definition of public health. The WHO Expert Committee on Public Health Administration, adapting Winslow's earlier definition has defined it as :

> *"the science and art of preventing disease, prolonging life, and promoting health and efficiency through organized community efforts for the sanitation of the environment, the control of communicable infections, the education of the individual in personal hygiene, the organization of medical and nursing services for early diagnosis and preventive treatment of disease, and the development of social machinery to ensure for every individual a standard of living adequate for the maintenance of health, so organizing these benefits as to enable every citizen to realize his birthright of health and longevity".*

Whereas in developing countries such as India, public health has not made much headway in terms of sanitary reforms and control of communicable diseases, it has made tremendous strides in the industrialized western countries resulting in longer expectation of life and significant decline in death rates. As a result of improvements in public health during the past 50 or 60 years, public health in the developed countries has moved from sanitation and control of communicable diseases (which have been largely controlled) to preventive, therapeutic and rehabilitative aspects of chronic diseases and behavioural disorders.

A EURO symposium in 1966 suggested that the definition of public health should be expanded to include the organization of medical care services. This was endorsed by another Expert Committee of WHO in 1973. Thus modern public health also includes organization of medical care, as a means of protecting and improving the health of people. Since the organization of public health tends to be determined by cultural, political and administrative patterns of the countries, there is a wide mosaic of organizational arrangements.

Public health, in its present form, is a combination of scientific discipliness (e.g., epidemiology biostatistics, laboratory sciences, social sciences, demography) and skills and strategies (e.g. epidemiological investigations, planning and management, interventions, surveillance, evaluation) that are directed to the maintenance and improvement of the health of the people.

Health

Health is a state of complete physical, mental and social well-being which is essential for leading a productive life, and is not merely the absence of disease or infirmity. Provision for health should be considered a fundamental human right and attainment of highest level of health is a most important social goal.

Concept of Health Care

Since health is influenced by a number of factors such as adequate food, housing, basic sanitation, healthy life styles, protection against environmental hazards and communicable diseases; the frontiers of health extend beyond the narrow limits of medical care. It is thus clear that "health care" is not synonymous with "medical care". Health care covers a broad spectrum of personal health services ranging from health education and information through prevention of disease, early diagnosis and treatment and rehabilitation.

This health care services may be defined as *"all those personal and community health services, including medical care and related education and research directed towards the protection and promotion of the health of the community."*

The term "health services" implies organization, delivery, staffing.

The major themes have emerged in the recent years in the delivery of health services : (a) First, that health services should be organised to meet the needs of entire population and not merely selected groups. Health services should cover the full range of preventive, curative and rehabilitation services. Health services are now seen as part of the basic social services of the community; (b) secondly, it is now fully realised that the best way to provide health care to the vast majority of underserved rural people and urban poor is to develop effective "primary health care" services supported by an appropriate referral system. The current social policy throughout the world is to build up health systems based on primary health care, towards the policy objective of Health for All by 2000AD.

Basis for Health Care in India in Modern Time

India gained independence in August, 1947, after a long colonial rule. Although, the National Health Policy in India was not framed and announced until 1983. The basis for organisation of health services in India through the primary health care approach in modern time was laid by recommendations and guidance provided by the 'Health Survey and Development Committee' (Bhore Committee) in 1946. The community development was launched in October 1952, as the first integrated all round rural development programme. It was proposed to establish one primary health centre (PHC) for each community development block. At that time, the operational responsibilties of the PHC were to cover medical care, control of communicable diseases, maternal and child health (MCH), nutrition, health education, school health, environmental sanitation and the collection of vital statistics. Each PHC had three sub-centres, being looked after by a trained midwife for providing MCH services.

Subsequently, over the past decades the health services organization and infrastructure have undergone extensive changes and expansion in stages following a review by a number of expert committees, namely the Mudaliar Committee (1961), the Mukherjee Committee (1966), the Kartar Singh Committee (1974), and the Srivastava Committee (1975). Progressive changes have been introducted into the programme over the six five-year plan periods. The first five-year plan (1951-56) initiated a process of all-round balanced development to ensure a steady improvement in the living standard over a period of time. New programmes for control of communicable diseases such as Malaria, filaria, tuberculosis, leprosy etc. was instituted. Health and medical care infrastructure and facilities, and water supply and sanitation were expanded with a view to improve the accessibility and availability of services.

The subsequent five-year plans aimed at expanding health and family planning services to bring these services within the reach of all people for improving their health status. During the

fifth plan (1974-80), removal of poverty and achievement of self-reliance on the part of the community were given emphasis. For preventing and correcting nutritional deficiencies, supplementary feeding programmes for children and expectant mothers were initiated on a country-wide basis. The family planning programme was integrated with the MCH and Nutrition programmes.

India is a signatory to the *Alma Ata Declaration* of 1978, as is committed to attaining the goals of "Heath for All" (HFA) by the year 2000 A.D., through the primary health care approach. Consequently, while formulating the sixth five-year plan (1980-85), a critical review was made of the approaches in the first five-year plans. Based on these, a long term perspective plan was outlined by the Government for achieving the HFA goals. Also efforts were initiated for the formulation of the National Health Policy keeping in view the HFA principles and strategies.

HEALTH FOR ALL

Since 1975, the WHO has been developing the concept of achieving health for all by 2000 AD. In the 30th World Assembly in 1977, it was decided that the main social goal of the governments and the WHO should be *"the attainment by all the people of the world by the year 2000, of a level of health that will permit them to work productively and to participate actively in the social life of their community."* This was popularly known as "Health for All (HFA) by 2000 AD."

In the International Conference on Primary Health Care, jointly organised by WHO and UNICEF in Alma Ata, USSR in 1978, this concept of HFA by 2000 AD was endorsed and it was further stated that primary health care would be the key to attaining this target. These also spelt out the minimum essential components of primary health care and the supportive activities needed for their successful implementation.

To achieve the goal of Health for All, a number of immediate goals or milestones had been planned in between as below:

1985— Providing right kind of food for all

1986— Providing essential drugs for all

1990— (a) Providing basic sanitation for all

(b) Providing adequate supply of drinking water for all

(c) Immunization of children against six common diseases, viz. measles, whooping cough, tetanus, diptheria, polio and tuberculosis.

Besides the above, a number of indicators (e.g. health status indicators, health care indicators, social and economic indicators and health policy indicators) have also been developed to enable countries to measure and monitor as they work towards the goal. However, it was left to each country to decide its own norms, while suggesting a minimum life expectancy of 60 years and maximum infant mortality rate of 50 per 1000 live births.

Most recent estimates indicate that some 83% habitations are 'fully covered', 15% are 'partially covered' and only 2% of habitations are not covered.

There has been marked impovement in the health status of the people in the country. Life expectancy has increased to 64.6 years during 2000. Crude birth rate has reduced to 26.1 during 1999. Crude death rate has declined to 8.7 and infact mortality rate to 70 during the same period. Planned initiatives taken in the public health field have also resulted in improvement in epidemiological scenario in the country. Small pox and Guinea worm diseases have been eradicated, Polio is on the verge of eradication, Leprosy, Kala-Azar and Filariasis can be expected to the

eliminated in the foreseeables future. Incidence of malaria which showed resergence during early seventies has reduced to 2.2 million cases during 2000.

Primary Heath Care

The term "primary health care" was first used to mean the care given to the patient by the health worker who saw him first. It was also called FIRST CONTACT CARE. If the patient was referred to the hospital, it was called "secondly care." More recently, the Alma Ata Conference in 1978 gave primary health care a wider meaning. The Alma Ata Conference defined primary health care as follows:

> *"Primary health care is essential health care made universally accessible to individual and acceptable to them, through their full participation and at a cost the community and country can afford."*

For achieving success in HFA development at least eight components of primary health care need to be properly implemented. For this, the cooperation and support of other social and economic development sectors, such as education, social and women's welfare, food and agriculture, animal husbandry, water resources, housing, rural development, energy, environmental protection, industry, communication, etc. would be vital.

Level of Care

Health services are usually organised at various levels (a) *Primary level:* The first level is the point of contact between individuals and the health system, where primary health care is delivered. The primary health centres and their sub-centres constitute this level of care in India, (b) *Intermediate level:* At this level, more complex problems are dealt with. The sub-divisional/ district hospitals usually constitute the second level. They provide support to the primary health care institutions, (c) *Central level:* The central level institutions (viz. Regional hospitals, Medical College Hospitals, specialised Hospitals) not only provide highly specialised care but, also to sustain primary health care as part of comprehensive national health system.

Principle underlying Primary Health Care

Primary health care is based on four principles:

1. **Equitable distribution:** Health service should be accessible to all the sections of the society with special attention to the needy and vulnerable groups. It is well known, that rural population, in most developed countries, are widely scattered and do not have adequate transportation facilities. City hospitals are beyond the reach of most rural people and are usually patronised by those in their immediate vicinity. Primary health care aims to correct this imbalance and bring health services as near people's home as possible and is supported by a higher level of health care, to which the patient can be referred.
2. **Community participation:** The involvement of individual families and communities in promotion of their health and welfare, including self-care, is an essential ingredient of primary health care. Community involvement also implies that the community should participate in the planning, implementation and maintenance of health services.
3. **Multisectorial approach:** Joint effort of the health sector and other health related sectors viz. education, food and agriculture, social welfare, animal husbandry, housing and public works, rural reconstruction etc. are needed for attainment of health.
4. **Appropriate technology:** Appropriate technology is not cheap primitive technology for poor, primitive people. It calls instead for scientifically sound materials and methods that are socially acceptable, directed against relevant health problems.

Health System Infrastructure

The country is divided into 22 major States and 9 smaller union territories, which in turn are divided into administrative districts. At present, there are 431 districts. Each district is divided into sub-districts or talukas, under which are situated the community development blocks. There are about 6,000 community development blocks in the country.

As mentioned earlier, over the past three decades the health services infrastructure and health care facilities have been expanded considerably. It is aimed to further improve the facilities as noted below:

Facilities at Village Level : In a village, for about 1,000 population, there will be one health guide and one trained dai or traditional birth attendant (TBA), both will be selected from the community. They will be trained at the level of the primary health centre (PHC) and the sub-centre. These two village level functionaries are to receive technical support and continuing education from the multi-purpose health workers (male and female) posted at the sub-centre. Other administrative control and supervision should ideally be carried out by the village health committee or the village panchayat.

Facilities at Sub-centre Level : The most peripheral health institutional facility will be at the sub-centre, manned by one male and one female multi-purpose health workers. At present, in most places there is one sub-centre for about 10,000 population. It is, however, aimed to have one sub-centre per 5,000 population (3,000 population in hilly and desert areas, and difficult terrain) by the end of the Seventh Five Year Plan i.e. 1990. To date, about 83,000 sub-centre (both on the old and new patterns mentioned above) have been established.

Facilities at PHC Level : At present there is one PHC in each community development block, which covers about 1,00,000 or more population. It was aimed to establish one PHC for every 30,000 population by the year 1990. Many rural dispensaries are being upgraded to create the subsidiary health centres or these new PHCs. Each new PHC will have one medical officer, two health assistants—one male and one female, and the health workers and other supporting staff. For strengthening preventive and promotive aspects of health care, a new non-medical post called community health officer (CHO) will be provided at each new PHC. To date, there are about 11,000 PHCs (both old and new combined).

Facilities at Community Health Centre : For a successful primary health care programme, effective referral support to be provided. For this purpose one community health centre (CHC) will be established for every 1,00,000 population, and this centre will provide the main specialist services. The CHCs will be established either by upgrading the sub-district/taluka hospitals or some of the block level PHCs, or by creating a new centre wherever absolutely needed.

Facilities at District Level : District health organisation is to be appropriately strengthened to cater to the needs of the expanding rural health and family welfare programmes. Not only the planning and implementation and monitoring of health and family welfare programmes are to be carried out at the district level (preferably on a decentralised basis), all the referral services from the periphery i.e. PHCs, community health centres and taluka hospitals, are to be attended to satisfactorily.

The primary contact care will be provided by the health functionaries at the village level and by the multipurpose workers at the sub-centres level. The cases needing further help will be dealt

with at the PHC, and those needing referral support by the specialist would be referred to the community health centre. The second and third level referral supports will be provided at the district hospital and the medical college/specialised hospitals respectively.

Organisation at State Level

Under the Ministry of Health and Family Welfare in each State, there is one technical directorate functioning under the Director(s) of Health Services, and one executive wing headed by the Health Secretary, who has also the overall administrative control.

Organisation at National Level

Under the Union Ministry of Health and Family Welfare, there are two technical departments— the Health Department is headed by the Director General of Health Services and Family Welfare Department is headed by the Commissioner, Family Welfare. The executive wing as well as the technical departments are under the overall administrative control of the Secretary to the Government of India.

Lesson 2

HEALTH CARE SERVICES-II

ESSENTIAL COMPONENTS OF PRIMARY HEALTH CARE

In the Alma Ata Declaration, it was stated that at least the following components should be included in primary health care:

1. Education of people about prevailing health problems and methods of preventing and controlling them.
2. Promotion of food supply and proper nutrition.
3. Adequate supply of safe water and basic sanitation.
4. Maternal and child health care and family planning.
5. Immunization against major infectious diseases.
6. Prevention and control of major endemic diseases.
7. Appropriate treatment of common diseases and injuries.
8. Provision of essential drugs.

Current Status of Various Components of Primary Health Care in India and Remedial Measures

1. **Education of people about Health Matters**

Problems—People in general, particularly in rural areas and urban slums are not knowledgeable about health matters, such as what are the prevailing health problems in the community and how to prevent and control these; what are the needs for the maintenance and promotion of health; what are the resources available and how and when to utilise these etc. Socio-economic backwardness, ignorance, traditions and superstitions had been acting as blocks to progressive thinking including development of the concept of positive health. Health education efforts have been very inadequate. Illiteracy, particularly of the women, has acted as barriers to communication in health and related matters.

Remedial measures—Appropriate educational programmes are to be organised for different groups of people. Health education to the community should be a prime function of health workers and village level functionaries. In this endeavour, functionaries of other sectors such as social and women's welfare, education, agriculture and youth clubs can contribute very significantly. Health education in schools and adult education sessions should incorporate various health problems, and the methods for their prevention and control.

2. **Promotion of Food Supply and Proper Nutrition**

Problems—Nutritional deficiency states of varying degrees in regard to protein-calorie malnutritional, vitamin A and iodine deficiency and nutritional anaemia are prevalent in a wide section of population. Nutritional deficiency states are particularly noticeable among pregnant and nursing mothers, and in infants and children. Available statistics indicate that of the deaths occuring among the age group of 0 to 5 years, in 7 per cent of deaths, malnutrition is the causative factor and in another 46 per cent, it is an associated factor.

Remedial measures—This dismal condition can be substantially improved by organizing and conducting nutrition education in the community and in the schools; encouraging people to

make kitchen gardens and community gardens; and educating the people on food hygiene. Steps also need to be taken to encourage growing locally more foods such as cereals, pulses, vegetables, fruits, milk, fish and poultry products through cooperative and other efforts so as to make these easily accessible and affordable to the people. Simultaneously, the purchasing capacity of the families might be improved through a variety of income generating schemes. In addition, for the moderately and severely malnourished groups, special nutrition programmes are to be organised.

In these endeavours, functionaries from other sectors such as agriculture, animal husbandary, irrigation, banks and cooperatives, social and women's welfare, panchayat and voluntary organisations can play a very significant role.

3. **Supply of Safe Water and Basic Sanitation Measures**

Problems—Many health problems have their roots in various aspects of community life and cannot be influenced by medical or health interventions alone. Safe and potable water is not available to a majority section of the population. Many of the water borne diseases prevalent in the country are preventable, but the importance of the use of pure and safe water as well as the personal hygiene are not properly appreciated. Environmental sanitation is very poor, particularly in rural areas and in urban slums. In most of the places, there are no proper arrangements for disposal of human and animal wastes, sewage and sullage etc.

Remedial measures—Systematic approach should be made to survey and identify resources of safe water and to carry out proper analysis of the water. Arrangements should be made for regular purification of water through chlorination etc. before using for drinking and other household purposes. People at all levels including the village leaders, women, and children at schools should be educated on continuous basis about the importance of proper maintenance of water resources, simple means of purification of water and the use of safe water. Observation of personal hygienic practices should be emphasised.

It would be important to organise the people and the resources for constructing household and community latrines, and making arrangements for collecting and disposal of human and animal wastes. Proper and imaginative disposal of waste water is also very important. Construction of composting facilities, soakage pits and use of some of the waste water in kitchen gardens should be encouraged and helped. Educating the women about the proper maintenance of water sources and the importance of kitchen garden would be helpful. Proper educational programmes on all these aspects for the children, youths, adults and the mothers should be organised in a systematic manner.

4. **Maternal and Child Health Care**

Maternal Care

Problems—In India, the Maternal Mortality Rate (MMR) which is calculated as the number of maternal deaths per 1,00,000 live births is among the highest in the world and has worsened in the recent years. It has gone from 424 in 1992-93 to 407 in 1998. (source SRS 1998). No valid information of morbidity data on mothers is available. Maternal care—antenatal, natal and post-natal, in rural areas and urban slums, is totally inadequate. In rural areas, majority (about 80 per cent) of births-are occurring outside the institutions, and are being attended by untrained birth attendants.

In India most deliveries take place at home without professional assistance under unhygienic surroundings. In 1998-99, 42% deliveries were assisted by a health professional and only 34% of births took place in a medical institution. The performance of Kerela and Goa surpass all other

states in terms of deliveries taking place in medical institutions with a similarly high percentage of births being assisted by a health professional. By contrast in Nagaland, Bihar and Uttar Pradesh only 12-16 percent of births are in medical institutions and in Meghalaya, Assam, Uttar Pradesh and Bihar only 21-23% deliveries are assisted by a health professional.

Some of the important causes of maternal mortality are sepsis, haemmorhage, toxaemia, illegal abortion and malnutrition. Liberalisation of abortion laws and enactment of medical termination of pregnancy (MTP) act in 1971 were the direct outcome of the realization of the fact that induced abortion performed by unqualified persons under unhygienic conditions significantly increased maternal mortality and morbidity.

Remedial measures—Systematic efforts are to be made to progressively increase antenatal registration and care of pregnant women from the present level of 35 to 50 per cent to 100 per cent. It is also to be ensured that progressively almost all deliveries are conducted under aseptic conditions by trained health personnel i.e. the dais or female multi-purpose workers. Pregnant and nursing mothers should get prophylactially two to three doses of tetanus toxoid, and iron and folic acid supplement for nutritional anaemia. During post-natal checkups, mothers are to be educated on breast feeding, growth monitoring, proper weaning practice and immunization of the child; and on personal hygiene, exercises, proper diet and family planning.

Facilities for MTP services by properly trained and skilled doctors are to be provided, wherever needed, in rural areas and urban slums.

Infant Care

Problems—India has established a reliable system for reporting infant mortality rate (IMR) which is the number of infant deaths per 1,000 live births. Latest data for 1999 reveal an IMR of 70 deaths per 1,000 live births — down from 129 in 1971 and 80 in 1990. An IMR of 70 inplies that of the 27 million children born every years in India, close to 1.7 million die before celebrating their first birthday. About 50 to 60 per cent of this is caused by mortality during the neonatal period (0-28 days) and particularly in the first week of life. Several factors contribute to this mortality and these include poor maternal health during pregnancy, frequent child births, inadequate care of mothers at risk, poor infrastructure facilities, lack of care of new born at birth and practically no facilities for new bom care from primary to tertiary levels.

Low-birth-weight infants, either due to prematurity or due to infra-uterine growth retardation, result from various factors such as how maternal weight and height, frequent pregnancies, maternal malnutrition and anaemia, chronic maternal diseases and pregnany complications. Low birth weight if particularly associated with pre-maturity is a major underlying factor for neonatal or infant mortality. Non-immunization of pregnant women with tetanus toxoid may result in death due to tetanus neonatorum.

Remedial measures—For dealing with these problems, the dais and female health workers and health assistants have to be properly trained in perinatal and neonatal are adopting a high-risk approach. Proper facilities for referrals to the secondary and tertiary levels are also to be developed and organised. Communities are to be properly educated about the importance of antenatal and neonatal care, and be encouraged to actively participate in these programmes.

Care of Young Children

Problems—Among the children aged 0-5 years i.e. the pre-school children, the major problems are the morbidity and mortality due to malnutrition, diarrhoeal diseases—respiratory infections

(three accounts for 30% under 5 deaths) and other preventable infections. Malnutrition predisposes the children to infection, the morbidity rates being three times higher in malnourished children.

Remedial measures—Two types of intervention programmes would be needed (a) prevention and treatment of malnutrition, and (b) reduction of mortality due to diarrhoea accounts for 20% under 5 deaths respiratory infections and other infections preventable by immunization.

The strategies for reduction of prevalence rate of malnutrition in pre-school children would be: (a) to provide nutrition education to the mothers; (b) to detect the cases of malnutrition and to grade them; (c) to rehabilitate moderately/severally malnourished cases by supplementary feeding from home resources; (d) supplementary feeding at sub-centres; and (e) referral grade III cases associated with diarrhoea or infection to secondary level of care i.e. the community health centres or district or taluka level hospitals.

The strategies for reduction in infant mortality due to diarrhoeal diseases and respiratory infections would be (a) to educate the mothers how to prevent and treat diarrhoeal and respiratory diseases; (b) to train the health functionaries about how to recognise and treat these disorders, and to judge which patients would need referral to higher levels of health service(b) and cotrimoxagole is distributed through all health outlets (c) to create facilities for secondary level care to referred cases; and (d) to provide drugs, oral rehydration salts (ORS) and other supportive measures. The government has launched the Oral Dehydration Therapy Programme to prevent death due to dehydration. In 1998-99, 62% of the mothers knew about ORS packets—an increase from 42% in 1992-93.

All children, preferably at the age of under one year must be immunised against tuberculosis, poliomyelitis, diphtheria, tetanus, whooping cough, and measles (where feasible).

Family Planning

Problems—Even though India was the first country in the world to take up family planning as an official programme in 1952, achievements over the past 33 years have not been as good as would be desirable. Currently 1996 the crude birth rate is around 27.5 per 1000 population. For reducing the birth rate to 25 per 1000 population and to achieve a net reproduction rate (NRR) of unity by 2000 A.D., 60 per cent of the eligible couples in the reproductive are to be effectively protected through contraceptive practice. It is estimated that about 32 per cent of the couples in the reproductive age group have been protected by contraceptive measures. Out of this nearly 27 per cent have been protected by sterilization alone, and only 5 per cent have been using spacing methods. More than 80 per cent of the acceptors of sterilization had three or more living children. Obviously, we may not expect the desired demographic gain from such contraceptive measures.

Remedial Measures—Now more concentrated attention has to be given to younger couples with low parity—i.e. the newly married couples, and one-child and two-child families for contraceptive protection with spacing methods.

The acceptance and continued use of contraceptives are influenced by several factors such as the character of the method including its advantages and disadvantages; individual and social acceptability; provider's knowledge, skill and attitude; effective communication, motivation, and counselling; the nature and quality of delivery services including supply logistics and follow up care; and the cost.

Small family norm has to become a way of life; for this purpose, organisation of population education in the schools and colleges, for the out of school youths and in adult education programmes would be most vital.

5. **Immunization Against Major Infectious Diseases**

Immunization of the children and pregnant mothers has already been referred to earlier. India has a long history of vaccination programmes. In the sixties vaccination against smallpox and tuberculosis was started, and in mid-seventies vaccination against diphtheria, pertusis and tetanus was taken up. Polio vaccination was initiated in 1980 and, in a limited scale, the measles vaccination in 1985. Presently, the package of immunization services for infants includes vaccination against tuberculosis, diphtheria, pertusis, tetanus, poliomyelitis and measles. Expectant mothers are to be given two doses or one booster dose of tetanus toxoid. In some endemic areas immunization against cholera and typhoid has also to be considered.

The immunization schedule presently being followed in the country is given below:

Beneficiaries	Age	Vaccine	Doses
Pregnant women	16-26 weeks	TT	2*
Infants	3-12 months	DPT	3
		Polio	3
		BCG	1
	9-15 months	Measles	1
	18-24 months	DPT	1
		Polio	1
Children	5-6 years	DT	1**
		Typhoid	2
	10 years	TT	1**
	10 years	TT	1**
		Typhoid	1**
	16 years	TT	1**
		Typhoid	1**

* One booster only if vaccinated previously.

** Two doses if not vaccinated previously.

The vaccination of children against 6 serious but preventable diseases (diptheria, pertusis, polio, measles, tetanus and tuberculosis) has been a cornerstone of the child care system in India. The Universal Immunization Programme (UIP) was launched in 1985-86 specifically for this purpose. By 1998-99, 42% of children aged 12-23 months were fully vaccinated (as against 36% in 1992-93).

With the help of Pulse Polio Immunization Programme (PPI) launched in 1996, India has achieved considerable success in its campaign to eradicate polio.

6. **Prevention and Control of Locally Endemic Diseases**

Although the prevalence of endemic diseases will vary from one region to another, some of the important ones are: tuberculosis, malaria, leprosy, filaria, scabies, guinea-worm infestation, rabies, iodine deficiency goitre etc. People are to be educated for their early diagnosis and

treatment health functionaries are to be trained for early detection and the services and follow up care to be organized.

7. **Appropriate Treatment of Common Diseases and Injuries**

Treatment of minor ailments and first-aids may be given at village level. Treatment of common diseases and injuries are to be provided at the sub-centres and PHCs, and appropriate referral services are to be organized. People need to be educated about the availability of local remedies and other facilities to meet these needs. Other sectors such as Education, Social and Women's Welfare, Panchayat, Voluntary Organizations can play an important role in educating the people and school teachers etc. and in organization of resources.

8. **Provision of Essential Drugs**

For local health care and treatment of common diseases and disorders, at least 20 drugs should be available within one hour's walk and travel. Utilising locally available remedies and using indigenous system of medicines should be considered. Considering the financial constraints from Government sources, community's participation through cooperative functioning etc. may be explored.

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PROTEIN—ENERGY MALNUTRITION

Protein energy malnutrition (PEM) is the most widespread form of malnutrition, especially among infants and children between 1-3 years of age. PEM is largely responsible for the fact that in many areas particularly in the developing countries like India, South East Asia and Africa up to half the children born, do not survive to the age of 5 years, those children who do survive, live with permanent impairement of physical and possibly mental growth.

PEM describes a spectrum of clinical disorders, ranging from mild to moderate deficiency as well as its most serious forms, which are *Kwashiorkar* and *Marasmus.* These two serious forms are the extremes and in between are a great number of intermediate case which often go unrecognised. Until recently, PEM was referred to as protein caloric malnutrition, but this nomenclature has been changed, as it has been argued that malnutrition occurs more due to a "food gap" rather than a specific calorie deficit. Moreover, the term protein calorie malnutrition, is too narrow a concept, as malnutrition may not be just a deficiency of calorie and protein, but other nutrient deficiencies may be involved.

Kwashiorkar was first described in 1995 by Dr Cicely Williams. She referred to it as a "disease the child gets when the next baby is born." the disease develops due to ignorance regarding the best weaning foods to be given to a child or due to an inability to provide these foods to the child for some reason or the other. The older child gets displaced from the breast and has to subsit on largely carbohydrate diets, which are low in protein i.e. an imbalance between protein and carbohydrate.

Kwashiorkar is due to a quantitative and qualitative deficiency of protein, but in which energy intake may be adequate. But a diet deficient in calories, but in which protein may be adequate leads to marasmus. Marasmus is derived from the greek word, meaning "to waste". Nutritional marasmus is the childhood equivalent of starvation in adults. The marasmus form of protein energy malnutrition occurs mostly in infants under one year of age, occuring due to early weaning, replaced by inadequate, dilute milk. This condition arises due to a continued restriction of both dietary energy and protein, as well as other nutrients.

The acute form of protein energy malnutrition is often precipitated due to inability on the part of the mother to supply proper nutrients to the foetus and later inability on her part to lactate. Marasmus is most common in infants and children below the age of 15 month, and is most frequently found in twins; whereas kwashiorkor occurs most often among children beyond this age. The degree of severity of protein energy malnutrition is related to the extent of growth failure. It is expressed by the weight loss per age as compared to the weight of normal children. Loss of weight by 11-25% is classified as grade 1, weight loss by 26.40% as grade II and weight loss by more than 40% as grade III in protein energy malnutrition.

Causes of Protein-Energy Malnutrition (PEM)

PEM occurs characteristically in children under 5 years, whenever the diet is poor in protein and energy. No age is immune to this, but in the older persons the disease is much less frequent and the clinical manifestations are less obvious and severe, as both protein and energy requirements are relatively reduced as the age advances. The main factors are discussed below:

1. **Age group of sufferer:** PEM is found exclusively in children under 5 years and over 1 year—the pre-school group. It is uncommon under 1 year in countries where prolonged breast

feeding is the common practice. Similarly those children who have escaped PEM and reached the age group of 5 or 6 years can be regarded as "out of danger". Recent studies in many countries have revealed that the most vulnerable age group can be narrowed down to the period between 1 to 3 years of age.

Nutritionally this is the most dangerous age-period when the majority of young children do not get mothcr's milk in adequate amounts even when prolonged breast feeding is practised, at the same time, according to usual dietary practice in most countries, solid foods are also not given in appreciable amounts to meet the ever increasing demand of calories, proteins and other nutrients. Besides, the child also starts getting all types of infections cither by feeding by bottle or by picking things from outside and this age group therefore gets all stypes of infections and infestation along with dietary defecieney.

2. **Birth order of the child:** Commonly, the sufferer belongs to a family with large number of children and has a relatively high birth-order. There are several studies on correlation between malnutrition of family size, which confirms that a mother with a large number of children is herself malnourished and sick, does not have time and energy for child care. On top of this, she gradually loses her ability of lactation. With each addition of a child, the total condition worsens. A child with fifth or sixth birth order in a poor or even middle class family groups up in situations favourable for the developments of PEM.

3. **Unavailablity of Protein rich foods:** Weaning period in topical countries is often characterised by a shortage of suitable, easily digestible foods, containing protein of good quality. All over the world, the animal origin foods are in short supply and expensive; thus unavailable as weaning food.

Due to industrialization, there is a tendency to abandon breast feeding and return to work; leading to deficiency of protein and in some cases, of protein as well as energy in the young infants.

Finally the advertising of tinned milk preparation, which can never be afforded in adequate 'quantities, resulting in over dilution, thus the diet of the child deficit in protein.

4. **Ignorance and Poverty:** Unawareness regarding the needs of the child and available resources is one of the foremost important cause of PEM. The mother may be ignorant about the cheap available food sources of good protein. The child is given milk with no supplementation, leading to gross deficiency of protein. Many a times, due to high cost of food, proper supplementation is not started.

5. **Beliefs Taboos and Customs:** Beliefs and customs regarding the requirements of the child may lead to malnutrition. Some of the examples are a prejudice against giving eggs to infants, prolonged restriction or use of purges in diarrhoeal disease, the habit of reserving animal protein for the males, breast feeding the infant and the sibling at the same time and overvaluation of the largely carbohydrate foods as "superfood" for the infants. Citrus fruits are believed to cause for common cold & cough.

6. **Infection:** In developing countries, major factor in PEM is the effect of frequent infections experienced by children in the first few years of life. Infections adversly effect the nutritional status; decreased appetide leads to reduced food intake, deminished absorption when diarrhoea is present. Even the slightest infection increases the loss of nitrogen through urine. Infection increases the need for energy, vitamin A and C and some other nutrients. There is enough

evidence showing that PEM increases the susceptibility to infections, specially diarrhoeal and respiratiory diseases.

7. **Premature Weaning:** PEM is recent times has shown a tendency to occur at an earlier age due to several reasons. The period of breast feeding is becoming shorter in towns and cities of the developing countries. In circumstances where mothers work away from home, resorting to artifical feeds is a necessity. The duration of breast feeding is shortened sometimes in response to advertisement, in the mistaken belief that it is good for infants, or a desire to imitate the better educated affluent section of society. Artificial feeds is unsatisfactory for a number of reasons. The fomulations may be wrong, and unhygienic methods used, predisposing the infants to attacks of gastroenteritis and hence to early PEM. It has thus been recommended that every effort, should be made to encourage mothers, particularly in the developing countries to breast feed at least throughout the first year of life.

Marasmus: The predisposing factors leading to the marasmic condition are a rapid succession of pregnancies and early, abrupt weaning. This is followed by duly and unsound artificial feeding of the infants with very dilute milk or milk products given in inadequate amounts to avoid expense. Thus, diet is low in both proteins as well as energy. In such circumstances, in poor house the preparation of clean food is almost impossible, resulting in repeated infections, especially of the gastro intestinal tract. The mother often treats this condition by "starvation therapy". Typically the marasmic from of the syndrome occurs in infants under 1 year and is more frequently found in towns. In marasmus, weaning is often early, the mother being induced to stop breast feeding for various reasons. The reasons could be an influence of advertisements advocating the use of articial food products, or presence of infection in herself or in the infant. Quite often poor, uneducated mothers stop breast feeding when another pregnancy begins. There is a widespread belief that the milk of a pregnant mother is bad for her child. In towns, the necessity to return to paid work is another factor leading to the stoppage of breast feeding in early infancy.

Kwashiorkar: This condition arises when after a prolonged period on the breast, the child is weaned to the traditional family diet, which is often low in protein because of poverty. Most often there is no supplement of milk available to the child. Kwashiorkar is frequently precipitated by the outbreak of illnesses such as malaria, measles or gastroenteritis. The presence of the infection raises the requirement of protein and energy in the body, which are already limiting in the child's body.

Both marasmus and kwashiorkar arise as a result of poverty and ignorance. Even when food is available, customs and food taboos restrict the use of certain foods, Unsuitable commercial food preparations may be purchased or good preparation misused from a failure to understand how to use them, when the elementary rules of food hygiene are not followed. The incidence of marasmus in the underdeveloped countries is on the increase as a consequence of a decline in breast feeding, urbanisation of uneducated families in need of jobs, who live in poor unsanitary houses with insufficient money.

Chart below shows the factors leading to the development of Marasmus and Kwashiosrkar:

BIRTH—

Repeated Pregnancies	Continued breast feeding till one year or later
↓	↓
Breast feeding only upto 5 months or so	Late gradual weaning
↓	↓
Early, abrupt weaning	Starchy family diet fed to the child.
↓	↓
Dilute Dietary formulae	Acute infections
↓	↓
Repeated infections esp. Gastroentertis	Kwashiorkar
↓	
Starvation therapy	
↓	
Nutritional Marasmic	
Marasmus————Kwashiorkar	

Identification of PEM

The manifestations of PEM vary widely, according to the nature of the causative factors, the age of the patient, the time for which they operate etc. two severe clinical forms are recognized: Kwashiorkar and nutritional marasmus. The protein deficiency in adult may show itself in the form of loss of weight, reduced subcuanous fat, anaemia, great susceptibility to infection, frequent loose stoolsol, general lethargy, inability to do substantial hard work, delay in healing of wounds and oedema. Thc clinical features of marasmus and Kwashiorkar will be dealt separately.

Marasmus

The symptoms of marasmus are as follows (fig. 1) :

(1) The child is reduced to mere skin and bone due to diminution of muscle mass.

(2) He has a shrunken, shrivelled skin and a monkey like face.

(3) There is usually watery diarrhoea or acidic bulky stools.

(4) Dehydration may occur due to watery diarrhoea.

(5) The weight of the child is much below the standard.

(6) The temperature is usually subnormal and the pulse rate low.

(7) The hair is dull and dry.

(8) The abdomen way be shrunken or distended with gas.

(9) The eyes become large in size due to shrunken face and gives the appearance, of staring.

(10) There may be a continued feeling of hunger.

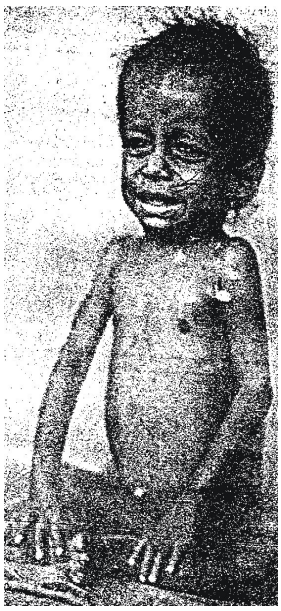

Fig. 1 : Clinical photograph of a child suffering from marasmus

Kwashiorkar

The physical features characteristics of Kwashiorkar can be classified as follows:

(a) Constant (b) Usual (c) Occassional

(a) Constant Features (fig. 2)

(1) *Psychmotor changes:* The child becomes extremely apethetic, with no interest in his surroundings. He is cheerless, miserable with no appetite. He remains seated where he is put down.

(2) *Growth retardation:* The supply of protein is limited and whatever is there, is used for energy, resulting in failure of growth. Body weight is reduced, but it may be covered by the presence of subcutaneous fat and oedema.

(3) *Oedema:* It occurs when there is excessive loss of nitrogen and the body tries to retain a state of normalcy by transfusion of extracellular fluid to the intracellular space of the cells. Oedema first occures on the feet and lower legs and then involves the back of hands, the thighs, the sacrum, the back arms and the face.

(4) *Muscle wasting with retention of some subcutaneous fat:* Muscle wasting is a constant feature of Kwashiorkar with a reduction in the circumference of the upper arm. This is due to muscle wasting. The relative thick layer of subcutaneous fat can also be observed both on the body and the limb.

(b) Usual Features

(1) *Hair Changes :* The hair is often sparse, soft and thin & can be pulled out easily causing pain. The characteristic curl in negro children is lost, loss of hair results in patchy alopecia. Their may be changes in pigmentation of hair, with streaks of blond and grey hair called ‘flag sign’.

(2) *Skin Changes:* The skin changes observed are areas of pigmenation and depigmentation. It is dry, and rough. The peeling of the pigmented areas gives the appearance of flaked paint. Patches of skin become inflammed and is called ‘crazy pavement dermatosis’.

(3) *Moon Face:* Oedema leads to a full, well rounded face with sagging cheeks and swollens eye lids.

Fig. 2 : Photograph of a child suffering from kwashiorkor

(4) *Anorexia and Diarrhoea:* Watery stools or large semisold acid stools are present. Poor sanitation is likely to be the cause of diarrhoea, which in turn rapidly advances the child to severe Kwashiorkar.

(5) *Abdomen:* distended but ascites is rare.

(c) Occassional Features

(1) *Fatty Liver:* Due to fat deposition inside the liver cells, the liver gets considerably enlarged down to the level of umbilicus.

(2) *Associated vitamins deficiency:* Vitamin A defeciency at times have been associated with PEM with manifestation of keratomalacia. Also angular stomatitis and glossitis may be observed due to riboflavin deficiency. Associated Fe (iron) and folate deficiency leads to anaemia.

(3) *Susceptibility to infection:* Children become prone to infectious desease.

The Principal Features of PEM

	Features	**Marasmus**	**Kwashiorkar**
	Essential features		
(1)	Oedema	None	Lower Legs, face or may be generalised.
(2)	Wasting	Marked. The childs appears to be skin and bones	Less Obvious
(3)	Muscle Wasting	Severe	Sometimes
(4)	Growth retardation in terms for body weight	Severe	Less than in marasmus
(5)	Mental Changes	Usually	Usually mental changes

Variable features

(1)	Appetite	Usually good	Usualy poor
(2)	Diarrhoea	Often	Often
(3)	Skin Change	Usually none	Often pigmentation observed.
(4)	Hair changes	Texture may be modified, rough	Sparse, straight, silky hair dispigmented often.
(5)	Moon face	None	Often
(6)	Liver enlargement	None	Often

The terms Kwashiorkar and nutritional marasmus are of little relevance in field studies, as the number of frank cases of either condition is always small compared to the total number of children who are malnourished. The original classification of PEM by Gomez into 3 degrees of malnutrition based on weight deficit takes into account the age of the child and the severity, but not the type or duration of the condition. It has also been suggested by some workers that cases with oedema be classified as 3rd degree malnutrition, regardless of the weight loss or extent of the growth failure. This implies that a child who is 50% of standard weight without oedema and a child who is 80% of standard weight with oedema are in the same category as regards the severity of their conditon. It has been realized that the measurement of deficit in height or body length gives valuable information about the chronicity of malnutrition, complementary to that given by the measurement of weight deficit. A reduction of stunting of height is evidence of past malnutrition; a low body weight in relation to height is evidence of present malnutrition, complementary to that given by the measurement of weight deficit. It is thus important to take into account both height and weight.

Treatment of PFM

The principles of treatment are :

(1) Adequate diet: The aim is to supply the lacking nutrients in the diet which are calories and proteins.

(2) Treatment of infections.

(3) Health education of parents, taking care of the children on how to prevent a relapse of the case.

A suitable diet is the main stay of treatment. The basic principle of treatment is to raise the child's nutritional status as quickly as possible, by providing sufficient calories and high quality protein. The child may have to be tube fed in the begining, which calls for a liquid diet. It is generally agreed that a daily intake of 170—200 Kcal per Kg. body weight and 3-4 gms of good quality protein per kg. body weight are sufficient to cover the child's needs. The protien used in treatment will depend on the local resources that are available. Whole milk is generally considered to be one of the best sources of protein being easy to administer as well as assimilate. A seriously ill child cannot tolerate whole milk and should be provided with skimmed milk or preprations of casein and mashed bananas. Gradually when the condition improves, whole milk can be resorted to alongwith vegetable protein multi mixes. The lack of Vitamin A is overcome by adding it to milk or giving a supplement. It is better to use milk and milk powders alongwith cereals, as excessive use of milk may cause diarrhoea. Once the milk toleration is better, the child can be put on other vegetable protein also. Vegetable proteins if carefully selected can be satisfactory if supplementing the cereals with dals, beans, peas in the ratio of 2:1 which can be a substitute for milk protein.

However, it should be borne in mind that the utilisation of protein depends on an adquate dietary supply of energy. Recovery and weight gain are not accelerated by intakes of over 3-5g Kg/day. Infants who have impaired digestive functions and are seriously ill, improve with an intake of 1g/kg/day, although larger amount are necessary at a slightly later stage for full recovery. It has been found that diets based on natural foods are better than protein concentrates and hydrolysates used.

Once the child is one the pathway of recovery, the metabolism is speeded up and the demand for vitamins and minerals is greater, thus the vitamin deficiencies may become more marked. Five or six small meals can alternate with snacks or beverages. Fish, egg, milk can be included in generous amounts. The same kind of diet should be continued at home, even after the patient is discharged from the hospital, which means education of the mother in terms of nutritional care is very important.

The importance of observing strict hygienic conditions while preperation of food is essential for successful treatment. Care should be taken to reconstitute milk powers with boiled water and use contamination free feeding bottles. It is safer to use the milk power by sprinkling it on the cereal or any other part of the child's meal.

In severe cases, when the child is hospitalised and is dehydrated, it is necessary to correct the water-electrolyte imbalance before starting the dietetic treatment. A. solution made of glucose, sodium, potassium, chloride and lactate can be given by mouth in small doses, upto a total of 100-200ml/Kg/ 24 hours. If vomiting continues, larger amounts may continue for 2-3 days if the diarrhoea and vomiting are severe. Once diet therapy begins, potassium, magnesium and iron are given as mineral supplements to make up the depletion in the body. For the first ten days in the hospital a multi vitamin preparation is given. Large doses are not required unless there is clinical evidence of a specific vitamin deficiency such as retinal if xeropthalmia is present or folic acid if megaloblastic anaemia is observed. Daily supplements of iron (60mg/day) and folic acid (100 mg/day) should be given to correct anaemia. When the child's apetite returns to normal it is best to meet the vitamin requirement through natural foods.

As PEM is frequently associated with infections, antibiotic therapy is required.

Amongst the general measures in the treatment of PEM-are keeping the child in a heated room as the body temperature is sub-normal. Education of the mother keeping in mind her economic position, education, experience and locally available foods is essential. Children can effectively be rehabilitated at home by convincing the mother that the cure can be effected by simple modifications of the diet within the economic reach of the family. The National Institute of Nutrition, has formulated an energy protein rich mixture to treat PEM at home level. It consists of

Whole wheat roasted	40 gms
Bengal gram roasted	15 gms
Groundnut	10 gms
Jaggery	20 gms

The energy value of this mixture is. 330 Kcalories and the protein content is 113 grams. Many children with PEM were cured within 3 months when treated with this food mixture. Similar mixtures based on cereals and legumes have been formulated. Incaparaina, Pronutro, AK-100 and Supramine arc available in some countries. India, Balamlul, Bal-Ahar, Pronurto, Protinex, protinules and Threptin biscuits are available.

Prevention of PEM

There is no simple solution to the problem of PEM. Many kinds of Inter-disciplinary action may be required. The following approach can be used for the prevention of PEM in the community.

(a) Health Promotion

It includes

1. Education of pregnant and lactating women. Distribution of supplements.
2. Promotion of breast feeding.
3. Development of low cost weaning foods.
4. Methods to improve the family diet.
5. Nutrition education regarding promotion of correct feeding practices.
6. Family planning.
7. Improving the family environment and sanitation.

(b) Specific Protection for Children

1. The child's diet must contain adequate calories and proteins. Foods rich in both should be used based on locally available foods like cereals and pulses.
2. Immunization programme should be intensified.
3. Food fortification.
4. Addition of fat to increase calorie density of food.
5. Amylase—rich foods prepared from wheat / maize to reduce bulk of weaning foods.

(c) Early Diagnosis and Treatment

1. Periodic surveillance of the population at risk.
2. Early diagnosis of any lag in growth by use of growth charts.
3. Early diagnosis and treatment of infections and diarrhoea.
4. Programmes for early rehydration in diarrhoea cases.
5. Supplementary feeding programmes during epidemics.
6. Deworming of heavily infested children.

(d) Rehabilitation

1. Provision of nutritional rehabilitation services for moderate cases.
2. Hospital treatment for severe cases.
3. Follow up care through supplementary feeding and nutrition education to prevent relapses of PEM.

Ignorance and poverty are the two main causes of PEM. Education in nutrition is necessary not only for mothers and potential mothers but for the whole community, including doctors and nurses. Recipies for weaning foods which are well within the reach of the family in agriculture, fertilisers and improved varieties of seeds are required. Only a system which provides employment and fair wages for all, linked with an educational system can prevent children dying from malnutrition. Family planning should be integrated with child health, both in the health centres as well as in the minds of the mothers. PEM has adverse effects on survival, health performance and the progress of population groups, therefore preventive action on a world wide basis is urgently needed.

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Lesson 4

VITAMIN A DEFICIENCY

Night blindness as a result of Vitamin A deficiency has been mentioned in Egyptian and Chinese, writings way back in 1500. A global survey sponsored by WHO in 1964 showed that Xeropthalmia i.e. all ocular manifestations due to Vitamin A deficiency is common in South and East Asia, many parts of Africa and Latin America. Vitamin A deficiency is closely associated with protein energy malnutrition. The peak age of its occurencc is between 2-5 years, when Kwashiorkar is very common. According to rough estimates 50,000 children may lose their eye sight every year due to vitamin A deficiency in India. Globally, it is estimated that every year about 7,00,000 children are likely to develop corneal lesions due to vitamin A deficiency and 20-40 million children are estimated to have mild vitamin A deficiency at any point of time.

Vitamin A plays an important part in many body functions and one of them is vision. Vitamin A is a component of visual purple, a pigment found in the retina of the eye.. When light strikes the retina, the visual purple changes into another pigment called visual yellow. In the dark the visual yellow again turns back into visual purple. However, this process is not 100% efficiency and a small amount of visual purple is always lost in the transformation. Thus, it must constantly be renewed from a steady source of vitamin A. When the nutrient is not present, vision is impaired and night blindness occurs, which is one of the first signs of vitamin A deficiency. In fact, the quickness of the eyes ability to adapt to darkness is sometimes used to measure the supply of vitamin A in the person's body.

INCIDENCE

1. **Age** : Vitamin A deficiency is preponderant in children. There is a progressive increase in the prevalance up to the age of 12-13 years. The corneal lesions, however, are rarely seen in the children about the age of 6 years. A great majority of the cases of careal xerophtholuia occur between 1 & 3 years.
2. **Sex** : Xerophthalmia is more frequent in boys than in girls though the incidence of kerotomalacia is similar in both sexes.
3. **Socio-economic factors** : Children from rural and tribal families belonging to low income group are more vulnerable to vitamin A deficiency. Families cannot afford animal foods which are rich in preformed VTA because of their poverty.
4. **Seasonal effects** : The seasonal charges in vitamin A deficiency are related to times of harvest. Studies indicate that the highest prevalance is observed in the months of May-June. There appears to be another peak in November and December. The peaks appear to be associated with total food intake and the consequent variation in the growth of the children.
5. **Drought** : The extent of vitamin A deficiency is more during drought due to non-availability of leafy regetables because of shortage of rainfall. The prevalance is higher in areas which are chronically drought prone.
6. **Regional Differences** : National Nutrition Monitoring Bureau (NNMB) surveys indicate that in the states of Andhra Pradesh, Gujarat, Karnataka, Orissa, Uttar Pradesh and West Bengal, the prevalance of xerophthalmia is higher.

Causes of Vitamin A Deficiency

1. **Inadequacy of the diet or a low dietary intake of vitamin A or its precursors, the caroteniods**—Deficiency occurs when the intake of Vitamin A or its precursor, carotene is poor. Sometimes food taboos and ignorance also leads to avoidance of certain beneficial foods.
2. **Impaired absorption of vitamin A**—It can be a major cause of deficiency. Failure to convert dietary carotene into biologically active vitamin can take place as a result of severe liver disease, diabetes, hypertension and body reserve may be depleted, as during pregnancy and lactation.

Absorption is also impaired in a condition where a major portion of the instestines has been surgically removed or in a condition called steatorrhea. Excessive intake of mineral oil is another cause of malabsorption of vitamins A, as much of it gets excerted in the faeces.

Lack of fat in the diet leads to impair absorption of fat soluble vitamins in the diet. Bile helps In the absorption of vitamin A and carotene. When there is an abstruction in the bile duct or low fat intake, absorption of vitamin A is seriously impaired, the conversion of carotene into vitamin A in the intestinal mucosa is helped in the presence of insulin and thyroxine. An absence of these harmones can be detrimental to vitamin A production in the body.

When the absorption of vitamin A is poor, the reserve in the liver is used up which in turn can *lead to a deficiency.*

3. **Infections**—It has been confirmed that the lowering of serum Vitamin A levels is directly related to infections. The drop in the vitamin A level is further aggravated if the infection is febrile. The degree of fever being directly proportional to the decrease in the Vitamin A level. Certain parasites e.g. Ascaris lumbrioides consumes a large amount of this Vitamin.
4. **Deficiency of Proteins and relationship with Protein Energy Malnutrition**—For the efficient coversion of carotene to vitamin A, and for the absorption, storage, transport and utilisation of Vitamin A certain amount of protein supply is essential. Protein metabolisin has a bearing on the transport of Vitamin A as this Vitamin is transported in serum in combination with a protein known as retinal bound protein (RBP). The synthesis of this protein is lowered in protein deprivation.

Foods which provide proteins also provide Vitamin A. Thus there is a direct relationship between PEM and Vitamin A decifiency. Children suffering from Kwashiorkar may fail to absorb Vitamin A given orally unless their protein status is improved.

5. **Poor Maternal Nutritional Status**—The maternal nutritional status directly affects the foetal stores as well as the amount of vitamin A that is secreted in breast milk. The foetal stores of babies belonging to mothers with poor nutritional status is much lower than their counterparts belonging to mothers having adequate nutrition.
6. **Poor Weaning Practices**—Delayed weaning with little or absolute non inclusion of green leafy vegetables can lead to Viamin A deficiency.
7. **Lack of awareness**—Due to lack of awareness, the community does not make use of the primary health care services like diarrhoea - control, immunization, vitamin A supplementation and other basic health services.
8. **Low purchasing power** : Low purchasing power of the communities and their inability to meet the dietary requirements ever after spending 80-90% of their income on food

is an important factor for vitamin A deficiency. Animal foods like eggs, milk & liver provide preformed vitamin A, but these cannot be afforded by poor communities and they rely on plant foods which provide only provitamin A.

Indentification of Deficiency

Vitamin A has several functions in the body. These are

1. It plays a crucial role in normal vision.
2. It is needed for the health of the epithelial celis viz. mucous membranes, skin.
3. It is connected with growth especially skeletal growth.
4. It is anti-infective i.e. protects the body against microbial infections.

It is thus obvious that a deficiency of vitamin A will hinder any one or all the processes mentioned above. The commonly affected part of the body as a result of Vitamin A deficiency are the eyes. Night blindness (fig 1) or inability to see in dimlight is the earliest symptom of Vitamin A deficiency. It is noticed that the child, stumbles or gropes for objects in dim light. Xeropthalmia literally means "dry eyes". The term is now used as a descriptive term for the ocular manifestations of Vitamin A deficiency in man. The presence of one or more of the following eye changes should be considered as incidence of Xeropthalmia.

(a) *Conjuctival Xerosis*—The Conjunctiva appears muddy and wrinkled instead of the usual transparent and clear. It becomes dry and unwettable. This is the first clinical sign of vitamin A deficiency.

(b) *'Bitot's Spots*—There are extension of the xerotic process. These are greyish, triangular, foamy, rough and raised patches on the conjunctiva. They are frequently bilateral. When Bitot spots are observed along with conjunctial xerosis, they are regarded as indicative or Vitamin A deficiency (fig. 2)

(c) *Carneal Xerosis*—This stage usually follows conjunctival Xerosis. The corneal surface also becomes dry and hazy, similar to ground glass. Corneal involvement should be considered a medical emergency.

(d) *Corneal ulceration with Xerosis*—If the ulcer progresses, there may be perforation of the cornea along with iris (eye muscles) prolapse. These unless are characteristically circular and sharply demarcated.

(e) *Keratomalacia (fig. 3)*—It consists of softening of the entire thickness or a part of the cornea. This process is rapid and if not treated promptly, it may lead to necrosis (scar formation) and destruction of the eye ball resulting in blindness.

The WHO reports the clinical symptoms of vitamin A deficiency affecting the eye and classifies the signs into primary and secondary signs.

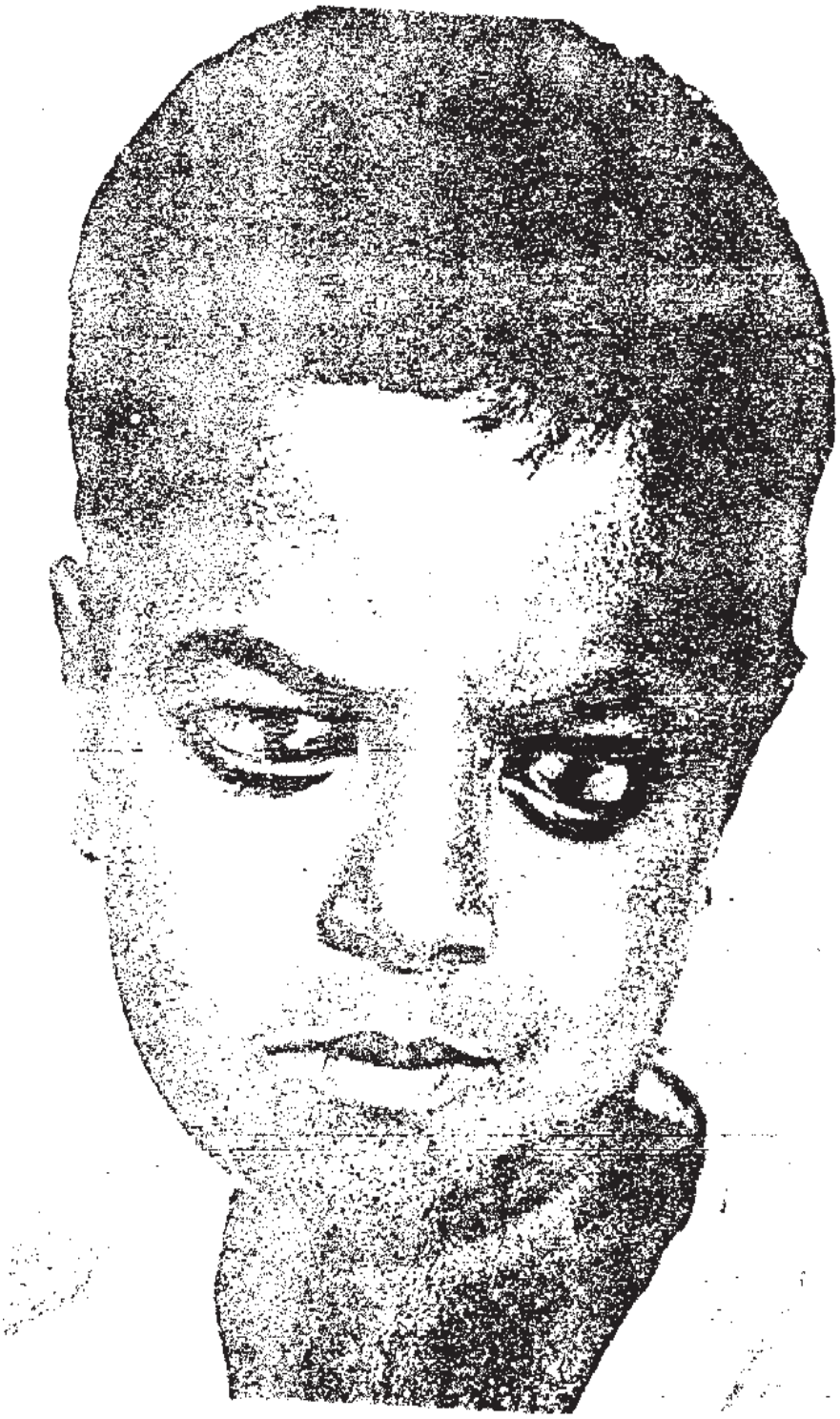


Fig. 1 : Blindness due to vitamin A deficiency.

Fig. 2 : Photograph of an eye with Bitot spot on the Conjunctiva

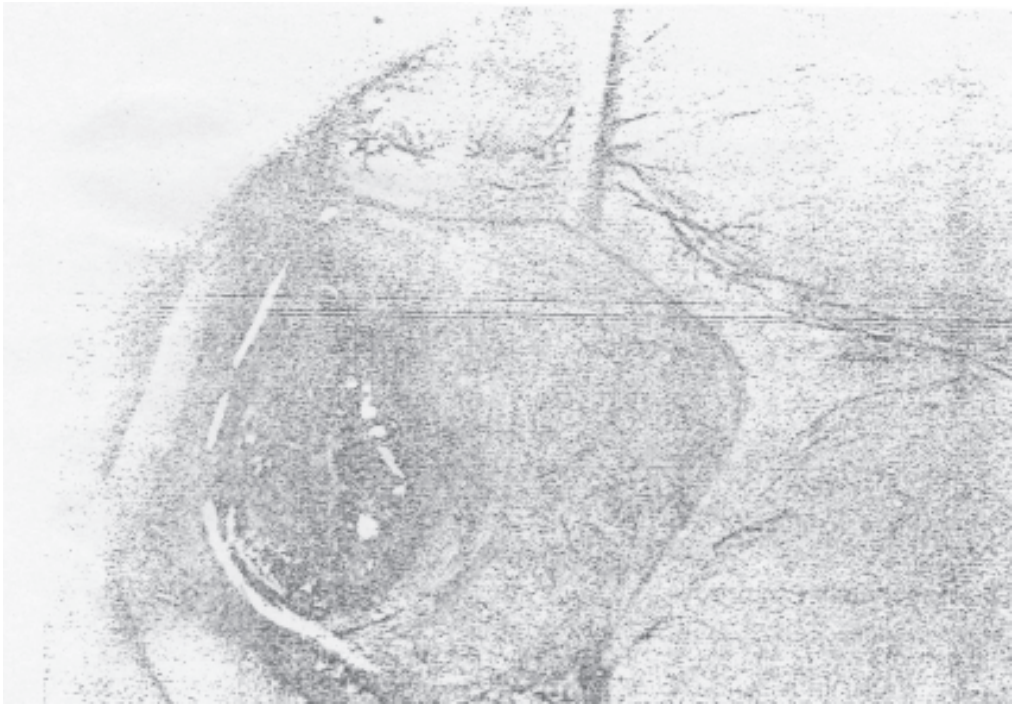


Fig. 3 : Photograph of an eye with keratomalacia

Theprimary signs according to increasing order of severity include:

X_1A Conjunctival Xerosis.

X_1B Conjunctival Xerosis with Bitot Spot.

X2 Corneal Xerosis.

X3 Corneal Ulceration.

X_3B Keratomalacia.

Uptodate the X_3A stage there are possibilities of reversing the damage caused by administering massive doses of vitamin A. However, once the X_3B stage sets in, irreversible changes occur.

The Secondary signs include:

X_1N Night Blindness

XB Bitot Spot only

XF Typical fundus changes in the eye

XS Corneal scars attributed to Xeropthalmia.

The Secondary signs are not pathognomonic signs of vitamin A deficiency i.e. there is no direct cause and effect relationship. Only when the secondary signs are observed in association with the primary signs, are they indicative of Vitamin A deficiency.

Besides affecting the eyes, in Vitamin A deficiency the mucous memberances of the nose, throat, trachea and bronchi become rough and dry resulting in bacterial infections.

In the alimentary canal, there is thickening and dryness of the tract resulting in diminished secretion of digestive juices, impaired absorption and increased liabilities to infections and diarrhoea. Due to changes in the epithelium, the skin becomes dry and scaly, the condition is known as follicular *Keratosis, toad skin* or *Phynoderma.*

Growth and reproduction affected by various forms of *Vitamin* A has been confirmed in animals studies. *'Bone development* may be showed if the vitamin is lacking during the rapid growth period. A liberal, apply of this vitamin, especially during prenatal life and childhood is important. Vitamin A is important for tooth formation. Like other epithelial cells, the enemel forming cells are affected by the lack of the vitamin. Fissures may be present on the enamel layer and the teeth tend to decay.

It has been shown in human beings that in acute infections, the serum levels of vitamin A and liver reserves decrease. This has been demonstrated in patients with pneumonia, tonsilitis and other febrile diseases. This indicates an increased need for the vitamin during infections or possibly increased destruction of the vitamin. A correlation between the amount of Vitamin A in the diet and the prevalence tuberculosis has been noted in human beings. Kidney and bladder disorders also arise as a result of epithelium abnormalities.

Identification of Vitamin A deficiency is also done by the biochemical criteria. Plasma levels if Vitamin A are indicative of the deficiency. 30 mcg to 50 mcg retional per 100 ml serum are considered acceptable, 20-30 mcg retinal/100 ml are indicative of deficiency and values below 10 mcg, 100 ml serum are considered very low. Plasma levels of<10 mcg/100 ml in more than 5% of the population at risk which is 0-5 years has been suggested for the community diagnosis of Xeropthalmia and vitamin A deficiency. Similarly if more than 2% of the children between 0-5 years show Bitot's spots Conjunctal Xerosis, it is regarded as a criterion for community diagnosis of Vitamin A deficiency.

Treatment

The oral administration of large doses of vitamin A is recommended method of treating of all stages of xerophthalia including corneal lesons. Immediately on diagnosis, an oral doses 2,00,000 I.U. of oil miscible vitamin A should be given to children in the age group of 1-6 years. In case of those with persistent vomiting and diarrhoea, an intramuscular injection of 1,00,000 IU of water miscible vitamin A can be substituted for the oral dose. This is followed by another dose of 2,00,000 IU one to four weeks later. In case of infants under the age of 12 months, half the dose of vitamin A is given.

For the prevention and treatment of secondary infections, antibiotics are of great value.

Prevention

The prevention and control of this deficiency can be done through the following methods

(1) Improving the dietary intake

The regular intake of green ieafy vegetables like spinach, methi, amaranth, cabbage; colored vegetables like carrots and pumpkin; fruits like mango and papaya, improves the serum vitamin A levels. These plant products contain Beta-carotene, the precursor of Vitamin A. The carotenes are converted to retinol mainly in the walls of the small intestine. Animal foods such as milk, egg yolk, butter, cheese, whole milk, fish are direct sources of retinol, but are relatively expensive. A daily intake of 100 gm leafy vegetables is practical way of meeting the daily adult requirement. Feeding children with 30 gms green vegetables for 12 weeks maintains adequate serum levels of the next 24 weeks period. The use of red palm oil should be encouraged wherever available. Red palm oil besides being rich in carotene pigments, the fat aids the carotene absorption in the diet. Breast feeding including feeding of colostrum must be encouraged. However, improvement in the dietary intake involves intensive nutrition education and is a long term approach.

(2) **Administration of a single prophylactic dose of Vitamin A**

The administration of 2 lakh IU of retinyl palmitate in oil by mouth every 6 months to pre-school children (1 to 5 years of age) has been found to be effective in reducing the incidence of ocular signs in preschool children by about 70-75%. A fat soluble preparation of the vitamin given orally is recommended rather than parenteral injection in oil, as the injected carrier fat is poorly absorbed. For injections, a water soluble preparation is preferred. The National Programme is now extended to cover children 6-11 months and a priority is now being given for children 6 months to 3 years. For 6-11 months infants one dose of 1 lakh IU is administered orally at the time of measles immunization.

(3) **Fortification**

Fortification with Vitamin A has been used as a technique to prevent blindness. Mother diary fortifies milk with vitamin A. Certain oils and Vanaspati are also fortified with this vitamin. Some countries like Guatemala, Panama, Costa Rica are using sugar fortified with vitamin A to prevent blindness. Trials have shown that tea leaves can be fortified with vitamin A and it is stable. Similarly, salt can also be used as a vehicle for fortification.

(4) **Dose for Lactating mothers**

To improve the quality of breast milk, 1 lakh I.U of water soluble Vitnmin A is given to the mother as soon as the baby is born. However, this method is not in practice and may not be very helpful too, as the Vilamin A may be all used up to replenish the mothers stores.

Lesson 5

ANAEMIA

Anaemia is said to be present when the concentration of haemoglobin (Hb) in the blood fails below the normal level. Hb is a vital biomolecule for the maintenance of normal health of an individual. Normal Hb levels vary with age, sex and physiological status. The WHO scientific group defined Nutritional anaemia as a condition in which the Hb 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. It is theoretically possible for anaemia to occur when there is a deficiency of any one of the substances which are known to be essential for red blood cell production i.e. proteins, amino acids, ascorbic acid, iron, folic acid, vitamin B_{12}, riboflavin, nicotinic acid, pyridoxine, pantothenic acid, copper and cobalt. Yet, it has been observed that deficiency of iron plays role in precipitating the anaemic condition followed by the nutrients like folate and vitamine B_{12}.

In India and other developing countries nutritional anaemia is primarily due to iron deficiency. It is a major public health problem involving hundreds of millions of people throughout the world. Anaemia occurs with greatest frequency at the peak periods of iron requirement which are infancy, menstruating females and during pregnancy. During these crucial periods the requirement for iron is increased.

Studies conducted by the National Institute of Nutrition have shown that 63 per cent of India's children below three years and 45 per cent from three to five years suffer from iron deficiency anaemia-moderate or severe in 10 to 15 percent cases.

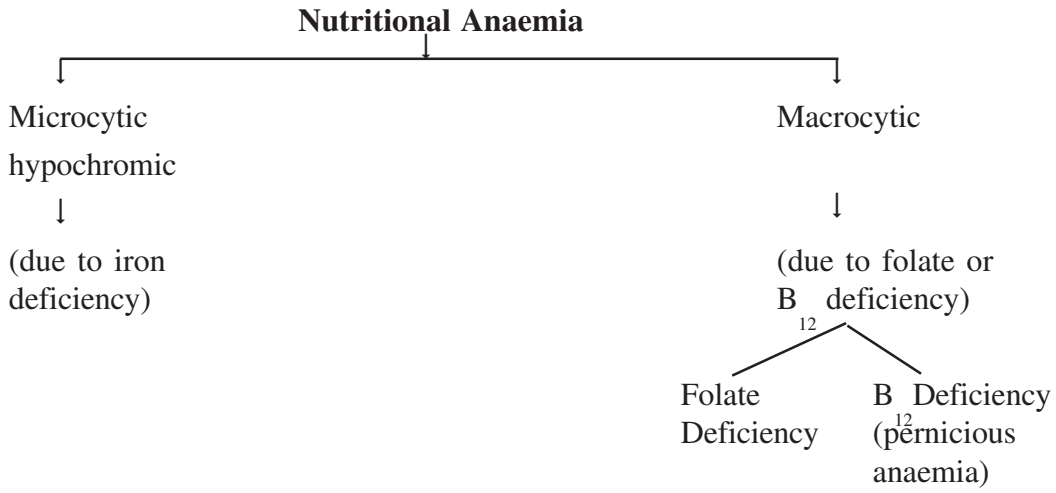
There are wide ranges of Hb concentrates in healthy persons and adult men have slightly higher values than adult women and children. The table gives figures below which the anaemic condition is said to exist. (WHO, 1972).

	Age	**Hb (g/100 ml)**
Children	6 months—6 years	11
	6 years—14 years	12
Adults	Men	12
	Women	12
	Pregnant women	11
	Lactating women	12

There are three main causes of anaemia :

1. Reduced production of erythrocytes (red blood cells) and Hb.
2. Loss of blood from the circulation, due to internal or external haemorrhage.
3. Haemolysis i.e., increased destruction of the red blood cells.

We will deal primarily with the first category of anaemias which is Nutritional Anaemia. In nutritional anaemias, the activity of the bone marrow which is the production site for the red blood cells is limited. The life of a red blood cell is 120 days. The bone marrow replaces them at such a rate that their number remains constant. When there is a deficiency of one or more essential nutrients involved in erythropoiesis (red blood cell production), the Hb concentration cannot be maintained and anaemia results. Nutritional anaemia is broadly classified into 2 types:



Causes of Anaemia

Iron Deficiency Anaemia

Iron deficiency anaemia is prevalent throughout the world. The incidence of anaemia is quite high in this country even in the upper classes, particularly among young children and women during their reproductive period. Anaemia is highly prevalent even among the men in rural areas because of hookworm infestations. The Bhils subsisting on maize are found to show a higher incidence, perhaps because of the low iron content of maize as compared to other grains. Although the infant at birth is born with adequate stores of iron, these are largely used up in the first six months and thereafter the child becomes anaemic, if the diet does not include enough iron. Infants born of anaemic mothers are found to become anaemic much earlier. Breast milk contains twice as much iron as bottle milk, so that bottle-fed infants need even more of supplements.

Causes of Iron deficiency Anaemia

1. Low dietary intake

The consumption of a merger diet is an important cause of anaemia, particularly where the economy is restricted. Quite often ignorance may lead to iron rich foods not being included in the diet.

II. Low availability of Iron

It also happens that even when the dietary intake of iron is adequate, anaemia exists. This is due to the poor absorption of iron from the diet. The cereal based diets, consumed in India contain high amounts of phosphates, phytatcs, oxalates. These from insoluble salts with iron and thus depress its absorption. Tea drinking also inhibits iron absorption in the body. Iron from animal foods, which is in the form of haem iron is better absorbed man non-haem iron from vegetarian sources. There are some nutrients which favour the absorption of iron like calcium, vitamin C and proteins, but their quantities are limited in the Indian diet. These nutrients increase the availability of non-haem iron in the diet. In some persons gastric secretions of hydrochloric acid are absent which favours iron liberation. This condition may also lead to anaemia.

III. Increased Losses from the body:

(a) Menstrual losses

(b) Pregnancy and lactation

(c) Haemorrhages

(d) Sweat losses

(e) Parasitic infestation

(f) Malabsorption syndrome.

(a) **Menstrual losses**—The menstrual blood loss varies between 10-151 ml of blood per period. The amount of Iron lost from the body is about 30 mg. and to meet this loss, 1.0 mg of iron has to be absorbed daily.

(b) **'Pregnancy and Lactation**—The loss of iron involved in a normal pregnancy, delivery and lactation for six months is approximately 900 mg. iron. Basal iron requirement is 280 mg., expansion of red cell mass is 570 mg., transfer to foetus 200-370 mg, iron content of placenta and cord, 34-170 mg, blood loss at delivery — 100-250 mg. Overall 500-600 mg of additional iron is required during entire pregnancy—daily requirement of 4-6 mg. of iron. Plus breast feeding also results in loss of another 100-180 mg of iron.

(c) **Haemorrhages** could be acute or chronic. There may be sudden loss of large volume of blood or there may be repeated loss of small amounts of blood like in peptic ulcers, haemorrhoids etc.

(d) S**weat losses** of iron are particularly significant in the tropical climates, especially when muscular work is performed.

(e) **Parasitic infestation**—Hook worm infestation is an important cause of anaemia, particularly in the tropics. Blood loss through this parasite ranges from .03 ml to. 2ml/ day/worm. Malaria parasite also destroys the red blood cells leading to the anaemic condition and later also lowers the iron absorpfon in the body.

(f) **Malabsorption syndrome**—Diseases of the gastro-intestinal tract like tropical sprue, coelic disease result in the anaemic state, gasitis. Gastric surgery, myxoedema and acute infections also result in the anaemic state.

Folic Acid Deficiency

Folate deficiency is observed in large sections of the population. It may occur at any age but pregnant women, infants and young children are affected more frequency. A high incidence of folic acid deficiency has been noted in elderly patients, correlated with poor intake of milk, fresh fruits and vegetables.

The causes of folate deficiency in man are:

(1) *Destruction of the vitamin during processing and cooking*—These procedures may bring about as much as 95% loss in the folate content. Pasteurisation of milk also destroys the folate to some extent. Storage of milk in transparent glass bottles leads to loss of folic acid, therefore, milk should be stored in opaque bottles.

(2) *Insufficient absorption of folate:* This arises as a result of a failure to absorb folic acid in certain gastro-intestinal disorders. Moreover adequate amounts of vitamin B_{12} is also required for the absorption of folate. For folate metabolism, certain vitamin B_{12} dependent enzymes are required. Therefore, it could be an indirect lack of vitamin B_{12}.

(3) *Drugs:* Certain group of drugs used for epileptics e.g. primidone, phenylhydraintone, phenobarbitones lead to increased folate excretion and megaloblastic anaemia. Alcohol and oral contraceptives also impair folate absorption.

(4) *Promature Children:* As the foetus acquires the folate from the mother only in the last term of pregnancy, premature children have a very low folate nutritional status.

Vitamin B_{12} Deficiency

Vitamin B_{12} must be bound to intrinsic factor, produced by parietal cells of the stomach before it is absorbed in the terminal ileum. Inability to produce intrinsic factor results in pernicious anaemia. The red cell count is often less than 2.5 million and a large proportion of the cells are macrocytic. The anaemia occurs chiefly in middle aged and elderly persons and may be a genetic defect.

Vitamin B_{12} is found only in foods of animal origin therefore strict vegetarians who avoid dairy products, eggs, meat, are at a risk of developing this deficiency. However, the vegetarian population derives their supply of vitamin B_{12} is uncommon.

The causes of vitamin B_{12} deficiency are:

(1) *failure in secretion of intrinsic factor by the stomach*—This is often a congenital defect in which condition the parietal cells of the stomach do not secrete the intrinsic factor leading to pernicious anaemia. Extreme cases of ulcers, where the stomach lining gets erroded also leads to mis condition. Coeliac disease, tropical sprue, other mal-absorption syndromes precipitate vitamin B_{12} deficiency.

(2) *Parasites*—Fish tape worm known as Diphyllobothrium latum is selective in drawing only vitamin B_{12} from the body.

(3) *Poor foetal stores*—Vitamin B_{12} is similar to the fact soluble vitamins, as it can be stored in the liver. When an infant is born with poor foetal stores, the risk of developing this deficiency is greater.

Identification of Anaemia

Anaemia give rise to the same general features whatever the cause. Anaemia often develops slowly. The patient may unknowingly reduce his physical activity. Lowered haemoglobin levels result in decreased ability to carry oxygen to the cells and to return carbon dioxide to the lungs for exhalation, (haemoglobin acts as a carrier of oxygen from lungs to tissues and of carbon dioxide from tissues to lungs or exhalation). With less oxygen and more carbon dioxide in the cells, body processes become sluggish and efficiency is lowered. A person in this condition is usually listless and dispirited. The severity of clinical features are dependent not only on the degree of anaemia, but on the rapidity of development.

The common symptoms are general fatigue, breathlessness on exertion, giddiness, headache, sleeplessness, the skin becomes pale, palpitations are felt, there is anorexia, a tingling sensation is felt in (the fingers and toes (paraesthesia). In addition to these, general features, signs of nutritional deficiency are seen. The tongue has a smooth glazed appearance due to the atrophy of the papillae and mucous membrance. Changes in the nails are observed. First, there is brittleness and dryness, later there is fattening and thinning, and finally spoon shaped nails (koilonychia) result. In general there is loss of efficiency and impaired general health. Psychiatric symptons are associated with low levels of vitamin B_{12} only.

The deleterious effects of anaemia are observed in (a) Pregnancy (b) work capacity of the individual (c) Resistance to infections.

The public healths importance of severe anaemia in a pregnant, woman is an increased maternal morbidity (sickness) and mortality (death) rate and increased risk for the foetus, as Hb concentration is definitely correlated with the birth weight of the infant. Anaemia lowers the capacity to do work, especially sustained physical activity for a continous period. This occurs as

Hb, the Oxygen carrier in blood is reduced. Iron deficiency anaemia in particular lowers the resistance to infections; as certain iron containing enzymes are reduced in the body.

The blood picture in the anaemic state gives indication as to the type of anaemia. In iron deficiency anaemia, the red blood cells are pale (hypochromic) and small (microcyctic) as there is insufficient iron for the formation of Hb. If the maturation of the red cells in the bone marrow is impaired due to the lack of folatc or vitamin B_{12}, the cells which enter the blood stream are irregular in shape and size but on an average, larger than normal (macrocytic). However, this is usually referred to as megaloblastic anaemia after the immature precursor of the red blood cells, the megaloblast. A measurement of Hb does not distinguish between these types of anaemias. For this two additional measurements are required. The first is to the packed cell volume (PCV) obtained by centrifuging blood and reading the height of the column of packed cells. The ratio of Hb/PCV gives the mean corpuscular Hb concentration (MCHC) in gms/100 ml. If the value is below 30, the red cells are lacking in Hb and the anaemia is hypochromic i.e., iron therapy is required. The ratio of PCV/RBC count gives the mean corpuscular volume (MCV). A value of 95 indicates the larger than normal size of the erythrocytes, suggesting either folate or B_{12}, deficiency.

Treatment

Iron deficiency anaemia—Once anaemia has developed, it is both unwise and uneconomic to try and correct it by dietary means alone. The administration of one 200 mg tablet of ferrous sulphate thrice daily for 30 days brings about a mean rise in Hb of 4 grns/100 ml. i.e., a rise of 1 g/100 ml/week. Ferrous sulphate is the iron preparation of choice. Ferrous gluconate, ferrous succinate and ferrous fumarate are equally effective but more expensive. Treatment should be continued for approximately 2 months after the Hb level has returned to normal in order to restock to body's depleted iron stores.

Parenteral iron therapy — this is often used when iron deficiency anaemia is detected in the last trimester of pregnancy or when patient does not take oral iron preparation and anaemia is serve. The advantages of parentical iron therapy are :

a. minimisation of hospital stay.

b. total amount of iron needed can be adnisitered in a short time.

c. no problem of Gastro Intestinel intolerance.

Folate deficiency anaemia—Folic acid in a dose of 5-10 mg daily is effective. Patients with Hb<5g/100 ml need blood transfusions to tide them over until the folic acid has acted. The patient should be given a well balanced diet and continue on it. The diet should include some good sources of folate every day.

Vitamin B_{12} deficiency anaemia (Pernicious anaemia)—Cyanocobalamine should be given in a dosage of 1000 mcg intramuscularly twice during the 1st week, then 250 mcg weekly until the blood count is normal. Folic acid should never be used for the treatment of pernicious anaemia, as it does not prevent the development of neurological complications but may precipitate them. Within 48 hours of the first injection of Cyanocobalamine, the bone marrow shows a striking change from megaloblastic to normal size of the red blood cells. Regular dose of cobalamine should be continued for life. A dose of 1000 mcg given every 4-6 weeks maintains the Hb level. The diet should be light, easily digested, rich in proteins, iron and vitamin C.

Prevention of Nutritional Anaemias

Iron Deficiency

Prevention of anaemia solely depends on a satisfactory diet giving an adequate supply of iron for the normal expansion of the tissue mass and blood volume. Of all nutrients, it is the most difficult to provide in diet. The anaemia is better understood if we know the individual iron requirements and the sources supplying it. Protein foods are concentrated sources of iron. Organ meats such as liver, lean muscle meat of all kinds and dried legumes, dark green leafy vegetables, dried fruits, egg yolk, whole grain enriched cereals and breads and jaggery are all goods sources of iron. Milk, cheese and ice-cream are poor sources of iron. In general fresh fruits and vegetables are of great value because of their vitamin C content which facilitates iron absorption. Cereals do not contribute much to the iron intake because of the phytates and phosphates. Thus education regarding the good sources of iron which can be afforded as well, is essential. Regular consumption of vitamin C to promote iron absorption & discouraging the consumption of foods like tea & tamarind that inhibit Fe absorption.

Besides, the incidence of anaemia can be reduced by the use of iron supplements, especially for the vulnerable groups. Research work carried out at the National Institute of Nutrition, Hyderabad, has shown that in pregnancy anaemia can be prevented by intake of iron tablets during the last 100 days of pregancy. Distributions of tables containing iron and folic acid to pregnant woman was, therefore, included as a preventive programme by the Government of India.

According to the supplementation programme, people found to have no iron stores in the body are given tablets. For pregnent women tablets containing 60 mg/100mg of elemental iron 500μg of folic acid is given daily for 100 days. The same is given to talking mothers and I.U.D. accepters and preschool children receive 20 mg. elemental iron + 100 mg folic acid daily.

Another method would be of foods with iron. The type of iron used for fortification must be one that is readily assimilated, does not cause undesirable changes in the vehicle or the food, and is stable under locally prevailing storage conditions. At present ferrous sulphate, ferrous ortho-phosphate, iron sodium pyrophosphate are the most commonly used forms for fortification. The vehicle of fortification should be one that is already being consumed in adequate amounts by the people in need. Salt is a likely substance for iron fortification. Besides salt in other countries cereals, sugar, infant foods have .aso been fortified.

Besides this, removal of parasites from the body needs immediate attention and an integrated approach is required so that repeated infestation does not occur from the environment.

In general the preventive measures should be such as to increase the absorption of iron from the diet and at the same time reduce the losses from the body.

Folate Deficiency

(1) *"Education* regarding the folale rich foods such as green leafy vegetables, yeast, foods of animal origin. Education in connection with the cooking methods which should be followed in order to retain the folate is important. Most baby foods available in the market are low in folate, therefore specific education should be given as how to improve it.

(2) *Supplementation*—500 μg of folate should be given to all pregnant women from the 2nd trimester of pregnancy and continued upto 6 months of lactation.

(3) *Combat associated problems*—Tackling the problem of malaria help in the prevention of folate deficiency.

Anaemia in infancy

Most of the time the anaemia is due to iron deficiency. The following factors explain the of anaemia in early life.

(1) *Prolonged milk, feeding* — Milk is a poor source of iron and without supplements of iron containing foods, anaemia develops.

(2) *Low Birth weight*—At full term the baby has a Hb of 20 g/100 ml. After birth hemolysis (red cell break-down) occurs and by the 8-10 week the Hb drops to 10 gms/100 ml. The iron is set free and is stored in the liver, to be utilised during milk feeding. Premature infants have a small blood volume and hence smaller stores of iron to tide them through milk feeding period. Moreover, their rate of growth is greater and hence the requirement of iron in increased, making them prone to anaemia.

(3) *Nutritional anaemia in the mother* — As a result of the mother's anaemic condition, the child's stores of iron are inadequate.

(4) *Infection* — Common infections depress the bone marrow function of producing red blood cells.

(5) *Malabsorption* may sometimes cause anaemia.

Treatment—Medicinal iron, in a dose of 6 mg/kg is required. Ferrous sulphate mixture for infants diluted with water 3 times a day is the usual dose, depending on the age.

Prevention

(1) Prevention and treatment of anaemia in pregnant women.

(2) Treatment of infections in infants.

(3) Provision of supplementary foods rich in iron from 4-6 months onwards.

(4) Administering prophylactic doses of iron to low birth weight infants from the 3rd month onwards. One paediatric tablet contains 20 mg iron and 100 mg folic acid is to be given daily for 100 days every year.

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Lesson 6

IODINE AND FLUORINE DEFICIENCY DISORDERS

Iodine Deficiency Disorders

The term 'goitre' is used to denote enlargement of the thyroid gland of whatever kind. Simple goitre is said to be present when the gland is visible and palpable, but no symptoms of either hypothyroidism or hyperthyroidism are felt by the subject. Goitre is endemic in many parts of the world. When goitre occurs in a significant number of people living in a particular area, it is considered endemic. If the prevalence of goitre is found in more than 2% of the population, the area is considered as goitre region. Prevalence of goitre in more than 5% is very young girls calls for public health intervention, as the area is considered as endemic goitre region. Simple goitres do not usually affect health, but they sometimes result in complications which may have serious consequences such as hypothyroidism, cretinism or deaf mutism. The complications rarely occur and more likely to be encountered in-regions where endemic goitre is prevalent. There is no doubt that environmental rather than hereditary factors determine the prevalence of most simple goitres-especially dietary factors, of which iodine deficiency is the major one.

The most obvious consequence of iodine deficiency is goitre, but recent studies have indicated that there is a much wider spectrum of disorders, some of them so severe as to be disabling. They include:

a) hypothyroidism
b) retarted physical development and impaired mental function
c) increased rate of spontaneous abortion and still birth
d) neurological cretinism, including deaf-mutism; and
e) myxoedematous critinism, including dwarfism and severe mental relardation

To express it more clearly, the term "endemic goitre" is now replaced by the term **Iodine Deficiency Disorders** (IDD) to refer to all the effects of iodine deficiency on human growth arid development which can be prevented by the correction of iodine deficiency.

Causes of Deficiency

1. **Lack of Iodine**

The lack of iodine in the diet is the major causative factor leading to goitre. Ocean foods such as sea-weeds, sea fish, shell fish are very good sources of iodine. Fresh river water fish are poor in iodine. Water and soil contain some amount of iodine. Most green leafy vegetables especially spinach contains a fair amount of iodine. Tea is also a good source of iodine.

2. **Presence of goitrogenic substances in food**

The identification of natural inhibitors of the thyroid gland in the causation of goitre is also well established. The goitrogenic properties are found in cabbage, turnips, seeds of cabbage, brassica seeds, groundnuts, cassava, soyabeans. The goitrogenic substances can be divided into 3 classes

Class-I These substances inhibit the uptake of iodine by the thyroid gland. eg. : Throcyanate, Isothipaganate cyanogenic glysosides.

Class II These substances interfere with organification of iodine molecule. An example such as inhibitor is thiooxazolidone (commonly called goitrin) which is activated by the enzyme thio glucosidase. Cooking and boiling destroys the goitrogenic substance.

Hardness of Water

Certain minerals like calcium, magnesium, flourine found in hand water effects the functioning of the thyroid gland and may lead to goitre.

4. **Dirty Water**

Certain observation led to thinking that faecal bacteria can produce a goitrogenic substance. Eight villages on the foot hills of the Himalayas deriving their water supply from a single source was studied. The incidence of goitre was highest in the lowest village where the pollution in the water progressively increased. The neighbouring villages were entirely free from goitre as it had an independent water supply. However, the correlation between goitre and bacterial counts in water have not been confirmed.

5. **Genetic basis of goitre**

In areas where endemic goitre exists, endemic cretinism is also present. There is some evidence, though not with certainly that, parents with goitre give birth to children who also suffer from goitre.

Identification

The problem of IDD is far more greater than that of goitre (fig : 1) and critinism. It is a national problem with grave socio-economic consequences.

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 Naga Hills in the east, extending about 2400 km and effecting 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 of iodine deficiency and the associated IDD in parts of Madhya Pradesh, Gujarat, Maharashtra, Andhra Pradesh, Kerala and Tamil Nadu. More and more new areas are being identified. Even areas near the sea coast like Bharuch district in Gujarat and Ernaculum district in Kerala are found goitre affected. In short no slate in India can be said to be entirely free from goitre. About 140 million people are estimated to be living in goitre endemic regions of the country. In the sub-Himalayan goitre belt of India done, nearly 55 million are estimated to be suffering from endemic goitre, with an average goitre prevalence rate of about 36 per cent. The spectrum of IDD is shown in Table I.

Table 1

The spectrum of iodine-deficiency disorders in approximate order of increasing severity.

Disorders	Levels of Severity
Goitre	— Grade I
	— Grade II
	— Grade III
	— Multinodular
Hypothyroidism	— Varying combination of clinical signs
Subnormal intelligence	
Delayed motor mile stones	

Mental deficiency	variable security
Hearing defects	
Speech defects	
Strabismus (squint)	— Unilateral
	— Bilateral
Neuron-muscular weakness	— muscle weakness in legs, arms, truck
	— Spastic diplegia
	— Spastic quadralagia
Endemic cretinism	— Hypothyroid cretinism
	— Neurological cretinism
Intrauterine death (Spontaneous abortion, miscarriage)	

Fig. : 1 Photograph of a man suffering from goitre.

Treatment

A simple goitre in a non endemic area already requires treatment. If the goitre becomes disfiguring, iodine therapy is seldom effective but thyroxine 0.2-0.3 mg. per day may be given. This inhibits the production of the thyroid stimulating hormone secreted by the pituitary gland and so, reduces this size of the thyroid gland. If there is no response to thyroxine and the goitre continues to be disfiguring, thyroidectomy (partial removal of thyroid gland) should be considered, especially if the size of the goitre obstruct the trachea.

Results of studies show that sodium iodide is effective in both prevention and cure of endemic goitre. When iodine is taken in forms other than food, careful supervision is essential. In goitrous regions, periodic checking of thyroid gland by physical examination is helpful so that curative treatment may be started before the goitre develops to any extent.

Prevention

There is evidence to show that iodine supplementation is effective as a means of eradicating endemic goitre and its complications.

1. **Iodised Salt** i.e., common salt fortified with small quantities of sodium or potassium iodate has been widely used in reducing the prevalence of goitre. The range of iodization depends on the degree of relative stability of salt, the amount of salt consumed by the population and the degree of goitre.

   The range of iodisation varies between one part of potasium iodide added to 1,000 to 130,000 of sodium chloride (common salt). The normal level of iodisalion is 1 part potassium iodide in 25,000 to 50,000 parts of salt. It was observed that there was 49.90% reduction in goitre due to iodisation of salt. The choice of iodine salt varied in different parts of the world. Potassium iodide is used in America and Western countries. Potassium iodate, is found to be better adapted for fortification in our country than iodide since it is a more stable compound, especially in areas where moisture and high temperatures prevail. Daily consumption of 10g of iodised salt (25 ppm KI) provides about 150µg of iodine consumption.

2. **Iodised oil**—In areas where salt Iodization cannot be employed, intramuscular injection of iodised oil has been used for preventisng endemic goitre. It is cheap, long acting and relatively free from side effects. Iodized poppyseed oil for injection (Lipiodol) as well as for oral administration (Oriodol) containing 37% of iodine has been successfully used. The dose recommended is 1.2 ml for adults and smaller doses for infants and children to be given once in every 3 years.

3. **Iodised bread**—Potassium iodate was added to bread with successful results. The prevalence of goitre in schoolchildrcn decreased.

4. **Iodine Tablets**—Sodium and potassium iodide tablets were distributed amongst children in Ohio with encouraging results.

FLUROSIS

Flurosis is a condition caused by an excess intake of flourine. Flourine is often called a two-edged sword. Ingestion of large amounts is associated with dental and skeletal flurosis and indequate amount with dental caries. Minute quantities of fluoride protect the teeth and even slightly raised levels produce harmful effects.

The main source of fluoride to man is drinking water, which is about 0.5 mg/litre in this country. In flurosis-endemic areas, the natural wastes have been found to contain as much as 3-12 mg of fluoride per litre.

Causes

The main cause of fluorsis is high fluoride content of drinking water. It has been found that flurosis occur only in areas with drinking water fluorid content about 1-2 ppm. With a longer time of exposure, these is likely to be greater accumulation of fluoride. Males are predominantly affected. The disease is more common in farmers and heavy manual workers especially those who carry heavy loads on their heads.

Areas where fluorosis is found are by and large rocky and sandy ones with hot and dry ultimate and low rainful. Further, the disease does not involve wide zones and has a patchy distribution. The villages affected often depend on well water for drinking purposes.

Villages with deeper wells are less severely affected.

Considerable amount of fluorides may be taken in with foods grown on fluoride rich soil. Similarly sea food, tea and cheese are rich in its fluoride content. Sorghum based diets have been found to result in higher retention of fluoride as compared to rice based diets.

In India fluorosis was initially seen in the Southern States & later in North Indian district of Bhatinda and Raibarily. Recently a new syndrome known as ‘Genu Velgum’ was discoursed in the areas of endemic fluorosis. It has been reported to occur in certain villages of Andhra Pradesh.

Nutrition plays a crucial role in susceptibility of persons chronically exposed to excessive intakes of flouride to its toxic effects. The effects of protein, calcium and vitamin C undenutrition have been stressed. Protective actions of the above nutrients on toxic manifestations of flouride ingestion has been reported.

The intake of certain other elements might either mitigate or potentiate the manifestations of chronic flouride toxicity. Silicon is essential for normal metabolism of bone & connective tissues. But its excessive intake can be deleterious to health. High intake of silicate aggravates the severity of dental and skeletal flourosis & enhances the retention of flouride in these tissues.

Molybdenum—High intake of molybdenum has been found to promote the retention of flouride in the bones of animals.

Copper—Copper deficiency can aggravate some clinical signs of flurosis. In endenic zones of flourosis, a primary deficiency of this element can exist due to its suboptimal intake. In addition, secondary deficiency of copper can also occur due to exessive intake of both flouride & molybdenum. The latter elements are known to deplete the copper pools of the body.

Dietary practices also can add to the fluoride intake. Parboiling of paddy enhences the fluoride content of rice significantly, the increase being directly proportional to the flouride content of the water used.

Clinical features or Symptoms

In milder from there is involvement of teeth only—dental fluorosis, while in severe cases, the entire skeleton is involved-skeletal fluorosis.

1) **Dental fluorosis :** It is the earliest and the most sensitive index of fluoride over intake during the period of teeth eruption. Usually the teeth of secondary dentition are involved, but in very severe cases, those of primary dentition may also be affected. The teeth loose their shiny appearance and chalkwhile patches appear on them. This is known as "mottled enamel" and is an early sign of dental fluorosis. The white patches later become yellow and sometimes brown or black. In severe cases, loss of enamel is accompanied by 'pitting' which gives the tooth a corroded a appearance. Mottling is best seen on the incisors of the upper jaw. Dental motting is an irreversible change and both boys and girls are equally affected.

2) **Skeletal fluorosis :** In older people, the disease affects the bones, tendons and ligaments. This is known as 'skeletal fluorosis'. This is followed by pain and stiffness of the back and later of joints of both limbs and limitation of neck movements. This loss of movements is because of the calcification of inverteberal, which renders the vertebral column rigid. This abnormal calcification also results in narrowing of inverted foramena and spinal canal and compression of vertebral disc. Skeletal fluorosis has been reported to be a public health problem of considerable magnitude in several districts of Andhra Pradesh, Haryana, Karnataka, Kerala, Punjab, Rajasthan and Tamil Nadu.

3) **Genu Velgum (or knocked knee syndrome)** : Recently scientists working at National Institute of Nutrition. Hyderabad found a new form of fluorosis called Genu velgum. Young and adobscent people were found to be the chief victims of the syndrome with higher preponderance in boys. These subjects exhibit typical signs of dental and skeletal fluorosis. In addition, they suffer from extensive osteoporosis of the limb bones. It is found in districts of Andhra Pradesh & Tamil Nadu. It was observed that this syndrome was seen among people where staple diet was sorghum.

Prevention & Control

The tolal amount of fluoride ingested determines the development of fluorosis. Therefore, it has been suggested that nutritional factors may modify the clinical course and severity of the disease. This also means that curtailment (reduction) in some way of the total fluoride intake may modify the severity of the clinical manifestations. Dietary survey carried out in Audhra Pradesh where fluorosis is endemic suggests that inadequate intakes of nutrients like proteins, ascorbic acid and calcium may aggravate the disease. In order to reduce the severity, these nutrients should be in sufficient amounts in the diet. Foods high in fluoride content should be avided by the population at risk. Communities need to be educated against indiscriminate use of fluoride rich pesticides and fertilizers, which are known to cause substantial increase in fluoride levels of foods. Paddy should be parboiled, if necessary in safe water only. It is also necessary to enlighten the people about the inherent risk of using fluoridated dental preparations newly dentrifices, mouthrinses, gels & tablets without adequate supervision.

Among the public health approaches suggested to control the problem are:

1) **Defluoridation:** Defluoridation using defluoridation agents to bring down the fluoride levels drinking water. The National Environment Engineering Research Institute, Nagpur

has developed a simple technique known as 'Nalgonda Technique' for defluoridation of water. 1st involves additions of two readily available chemicals, vis. lime and alum in sequence followed by flocculation, sedimentation and filtration. Lime powder is added first (30 mg/1) and mixed well with water. Generally the lime dose is l/20th of the alum dose. Alum (500 mg/1) is then added and the water is stirred for 10 minutes. The contents are allowed to settle for one hour. The settled water will contain fluoride within permissible limits. The technique is suitable for both domestic and community water treatment.

2) **Deep Wells :** Replacement of present wells with deep bore wells because in lower levels the fluoride content of water is lower. Deep drinking of wells may even give fluoride free water.

3) **Use of canal & dam water :** In Andhra Pradesh analysis of water smaples collected from dams and canals showed that their fluoride contents were much lower than that seen in well water which is presently being used for drinking purposes.

References

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Lesson 7

DIET IN FEVERS

Fever is an elevation of body temperature above normal which results from an imbalance between the heat produced and eliminated from the body. Fever may occur in response to an infection, inflammation or a number of other causes brought about by exogenous agents like bacteria or fungi or endogenous factors like antigen-antibody reaction, malignancy or graft rejection.

Fevers may be classified as acute or chronic.

- *Acute fevers:* Acute fevers are of short duration but the body temperature may rise to even above 104° F. Fevers accompanying infections like chicken pox, tonsilitis, influenza, pneumonia, typhoid, malaria are few examples of acute fevers. Malaria, though an acute fever is also called a recurrent fever because of repeated episodes of high fever.
- C*hronic fever:* These are of long duration. The temperature may remain low but fever continues for a longer period of time, even several months as in the case of tuberculosis. Thus, chronic fever is one which has slow, gradual onset and is low in severity.

Metabolism in fevers

With a rise in body temperature above normal (98.4° F or 37° C), the following metabolic changes occur inside the body. These changes are in proportion to the elevaton of body temperature above normal and the duration of fever.

(i) There is a 7% increase in BMR with every 1° F increase in body temperature or 13% increase with every 1° C rise in body temperature true. This change is more significant in patients suffering from acute fever.

(ii) Glycogen and adipose tissue stores decrease significantly because of increased energy expenditure. Thus more energy is required.

(iii) The rate of protein catabolism increases depending upon severity of infection and duration of fever. There are increased losses in long continuous fever than in short duration fevers. Protein breakdown is especially marked in fevers such as typhoid, malaria, poliomyelitis and tuberculosis. This leads to increased nitrogen wastes and places an additional burden on the kidneys.

(iv) There is loss of body fluid in the form of excessive sweat and urine formation.

(v) There is increased loss of minerals like sodium, potassium, chloride etc. through sweat, urine and vomiting leading to electrolyte imbalance.

(vi) The absorption of nutrients like protein, minerals and vitamins decreases.

(vii) The above changes are accompanied by a loss of appetite resulting in low intake of food which leads to loss of weight.

Acute fever - Typhoid

Typhoid fever is an infectious disease caused by a bacteria Salmonella typhi. The infection is transmitted through the faecal-oral route i.e. by consumption of food, water or milk contaminated with intestinal contents. It may affect all age groups but commonly occurs in children.

Body Changes in Typhoid Fever

There is loss of tissue protein which may amount to as much as 250-500g of muscle tissue a day. Body stores of glycogen are quickly depleted and the water electrolyte balance is disturbed.

The intestinal tract is highly inflamed and irritable and diarrhoea is, therefore, a frequent complication which interferes with the absorption of nutrients. Ulceration in the intestines, i.e., *Peyer's patches* may be so severe that haemorrhage and even perforation of intestine may occur.

Treatment

Treatment of typhoid involves:

(i) Rest in the bed is mandatory for all patients

(ii) At all times patient should be kept warm

(iii) Antibiotics are given to cure the infection

(iv) Diet is modified to bring the patient to eat food and recover the losses and maintain thereafter.

Dietary Management

During high fever, there is an aversion towards foods in the form of anorexia, nausea and vomiting. The diet should be planned with the following objectives:

i) to maintain adequate nutrition

ii) to restore positive *nitrogen balance* and reduce the burden on kidneys

iii) to provide relief to symptoms as and when present.

iv) to correct and maintain water and electrolyte balance

v) to avoid irritation of intestinal tract as may occur in typhoid.

To achieve the above objectives, the nutrient intake may be modified as follows :

Energy

Since, there is a rise in body temperature, BMR may be increased by as much as 50%. Therefore, it is recommended to increase the energy intake by 10-20%. Initially, during the acute stage, a patient may be able to consume only 600-1200 kcal/day, but the energy intake should be gradually increased with recovery and improved tolerance.

Protein

Since there is excessive destruction of tissues, the protein intake should be increased to $1^1/_2$ to 2 times the normal, i.e. 1.5 to 2 g protein/kg body wt/day. For efficient utilization of protein, energy intake should be adequate. To minimize the tissue loss, protein foods of high biologic value such as milk and eggs should be used liberally as they are most easily digested and absorbed.

Carbohydrates

A liberal intake of carbohydrates is suggested to replenish the depleted glycogen stores of the body. Well cooked, easily digestible carbohydrates like simple starches, glucose, honey, cane sugar etc. should be included as they require much less digestion and are well assimilated.

Fats

Fats are required mainly to increase the energy intake. However, due to the presence of diarrhoea, fats only in the emulsified form like cream, butter, whole milk, egg yolk should be included in the diet, as they are easily digested and well tolerated by patients. Fried food which are difficult to digest should be avoided.

Minerals

There is excessive loss of electrolytes like sodium, potassium, and chloride due to increased perspiration. Salty soups broths fruit juices, milk etc. should be included to compensate for the loss of electrolytes.

Vitamins

As infections increase the requirement for vitamin A and deplete tissue stores of vitamin C, there is a need to increase the intake of both these vitamins. With the increase in energy requirements the need of B group vitamins also increases. So, vitamin supplements may have to be given for some time.

Dietary fibre

The symptoms of typhoid include diarrhoea and lesions in the intestinal tract, all forms of irritants have to be eliminated from the diet. All harsh, irritating fibre should, therefore, be avoided in the diet, as it is a mechanical irritant.

Fluid

In order to compensate for the losses through the skin and sweat and also for ensuring adequate volume of urine for excreting waste, a liberal intake of fluids is very essential. A daily fluid intake for 2.5 to 5 litres is desirable. Fluids may be included in the form of beverages, soups, juices, plain water etc.

Consistency

A high energy, high protein, full fluid diet is recommended in the beginning. Small meals are given at frequent intervals of 2-3 hours. Sufficient intake of fluids and salt should be ensured.

As soon as the fever comes down, a **bland, low fibre, soft diet,** which is easily digested and absorbed should be given to the patient. Well cooked, well mashed, sieved, bland, semisolid foods like khichdi, rice with curd, kheers, custard may be given. Bland, readily digested food affords physiological rest to the alimentary tract. In the beginning, small quantities of food at 2-3 hours interval will provide adequate nutrition without overtaxing the digestives system at any one time.

Foods to be used in restricted amount or avoided

- High fibre foods like whole grain cereals and their products e.g. whole wheat flour and cracked wheat, whole pulses and pulses with husk,
- All raw vegetables and fruits excluding papaya and banana.
- Fried fatty foods such as samosas, pakoras, halwas, ladoos etc.
- Chemical irritants such as condiments, spices, pickles, relishes, chutneys and strongly flavoured vegetables like cabbage, capsicum, turnip, raddish, onion and garlic.

Foods to be included

- Plenty of fluids like juices and soups.
- Milk and milk based beverages.
- Low fibre foods such as refined cereals and their products, dehusked pulses, well cooked fruits, vegetables in soft and puree form and potatoes.
- Foods providing proteins of high biologic value e.g. eggs, soft cheeses, tender meats, fish, poultry etc.
- Plain gelatin based desserts, sugars, honey, jam.

A Day's Sample Diet Plan for a Typhoid Patient

Personal Data

Age	8 years
Socio-economic	MIG
Food habits	Non-vegetarian

Recommended Dietary Allowances (ICMR, 1990)

	Normal	**Modified**
Energy	2190 kcal	+10% = 2,420 kcal
Protein	54 g	+50%=81g
Vitamin A	600 mg retinol	
Vitamin C	40 mg.	

Chronic fever—tuberculosis

Tuberculosis is a chronic infectious disease and is one of the major causes of illness and death in the underdeveloped countries as well as the deprived sections of developed countries. Malnutrition resulting from poverty and ignorance combined with unhygienic living conditions and poor sanitation makes an individual susceptible to the infection. It is caused by a bacteria Mycobacterium tuberculosis. The bacteria most often affect the lungs, leading to pulmonary tuberculosis.

Body Changes in Tuberculosis

Pulmonary tuberculosis is accompanied by wasting of tissues, exhaustion, cough, expectoration and fever. It is characterized in the early stage by a marked rise in body temperature, flushed face, increased circulation and respiration, constant fatigue, loss of weight, cough and a general run down condition. If the temperature rises above 39% C, the metabolic rate may increase 20-30% above normal. In the acute stage, the disease is quite similar to that of acute fevers. The chronic phase is accompanied by low grade fever and therefore, increase in metabolic rate is not marked as in typhoid. However, due to the long duration of illness, wasting of body tissues may be considerable and there is a noticeable loss of muscle tissues.

Treatment

Rest, antibiotic therapy and fresh air along with nourishing food are the four factors necessary to provide recovery from tuberculosis.

Dietary Treatment

Chronic fever leads to increased tissue breakdown and malnutrition. Therefore, the objectives of dietary treatment are:

i) to reduce mortality by providing nutrients required by body's immune mechanism,

ii) to prevent or control weakness and loss of weight, and

iii) to accelerate convalescence.

To achieve the above objectives, the following nutrient modifications are made.

Energy

As the patient with chronic tuberculosis is undernourished and underweight, energy needs are increased in order to minimize weight loss and achieve desirable weight. Therefore, energy intake needs to be increased by 300-500 kcal/day above the normal intake.

Protein

In fevers of prolonged duration like tuberculosis, there is a considerable wasting of body tissues. Serum albumin level is often low. Therefore, it is essential to increase the protein intake. Protein foods of high biological value should be included in the diet. About 1.2 to 1.5g of protein per kg body weight per day must be given.

Carbohydrates and Fats

Enough carbohydrates should be included in the diet to meet the increased energy requirement. An abundant amount of carbohydrate helps in sparing proteins for tissue building. Too much fat should be avoided as it frequently causes gastric upsets and diarrhoea.

Minerals

A liberal amount of calcium should be included in the diet to promote the healing of tuberculin lesions. Therefore, some amount of milk should be included in the daily diet. Iron supplementation may be necessary, if the patient suffers from haemorrhages.

Vitamins

The diet should provide liberal amounts of all vitamins. Since the conversion of Beta carotene to retinol in the intestinal mucosa is a adversely affected, the diet should provide as much ratinal as possible by giving some amount of milk and milk products, eggs and meat. Tuberculosis being an infectious disease results in increased urinary loss of ascorbic acid. As ascorbic acid helps in healing, additional amounts of ascorbic acid are therefore, recommended in the diet.

For proper absorption of calcium, enough of vitamin D should be included, as the energy needs increase. In addition, prolonged use of chemotherapeutic agents used in treatment of tuberculosis may have an adverse effect on the utilization of certain B-group vitamins.

Consistency and Feeding Pattern

During the acute stage of illness when fever is high, a high protein, high energy full fluid diet as used for acute fevers is given. As improvement occurs, this diet is progressed to a soft and then a regular diet. Most patients have a poor appetite. Meals should be made simple, easily digestible, well prepared and tempting to encourage the patient to eat. All meals should have cereal-pulse combination with some amount of animal protein e.g. khichdi with curd, sweet dalia with milk, Pushtik roti and curd etc. Cheap sources of vitamin C, such as guavava, and sprouted pulses must be given. Seasonal vegetables must be simply provided.

A Day's Sample Diet Plan for a Tuberculosis Patient

Personal Data

	Age	:	28 years
	Sex	:	Female
Socio-economic status		:	Low middle income group
Food habits		:	Non-vegetarian

Recommended Dietary Allowances (ICMR 1990)

	Normal		Modified
Energy	2225 kcal	:	+ 25-50% = 62 - 75g
Protein	50 g	:	
Vitamin A	600 mg retinol		
Calcium	400 mg		

Meal	Menu	Amount
Early morning	Tea	1 cup
Breakfast	Buttered Toast	2
	Boiled egg	2
	Milk	1 cup
	Orange	1
Mid-morning	Milk	1 glass
	Poha	1 Bowl
Lunch	Vegetable Khichiri	1 Bowl
	Curds	1 medium bowl
	Mix Vegetable	1 medium bowl
	Fruit cream	1 medium Bowl
Tea	Tea	1 cup
	Paneer sandwich	2 slices with paneer & tomato
Dinner	Idli Sambhar	Sambhar with 2 idlis
	Curd Fruit	1 medium bowl
	Boiled Potatoes	1 larges

Lesson 8

DIET DURING DIARRHOEA

Diarrhoea is a common illness and a leading cause of death in young children. In developing countries, children suffer an average of three episodes per year during the first five years of life. An estimated 4 million diarrhoea related deaths occur annually among young children throughout the world.

What is diarrhoea?

The number of stools normally passed in a day varies with diet and age of the person. In diarrhoea, the stool contains more water than normal. They may also contain blood and mucous, in which case the diarrhoea is called dysentery. Therefore, we can say that diarrhoea is the frequent passage of loose watery and informed stools which may also contain blood or mucous.

It occurs due to an abnormally rapid passage of food along the alimentary tract, preventing complete digestion and absorption. The number of stools may vary from several per day to one every few minutes. The affected individual becomes weak and malnourished. His body capacity to work as well as the resistance to disease is lowered considerably. In children physical and mental state may also be affected. Diarrhoea, if neglected can lead to dehydration due to excess loss of water and electrolytes from the body. This may prove fatal, particularly in infants and young children.

Etiology

Diarrhoea is generally not a disease by itself but symptom of an underlying functional or organic disease.

In **organic diarrhoea,** there are lesions in the mucosal linning of the intestines. It can be caused by

- Bacterial infection as in typhoid and Bacillary dysentery continued through food or water.
- Parasitic or Protozoal infection like amoebiasis.
- Malabsorption syndrome like celiac disease.
- Other diseases like viral hepatitis, tuberculosis, liver, cirrhosis etc.
- Carcinoma of the intestine.

Functional Diarrhoea results from an increase in the neuromuscular activity which may be brought about by any of the following :

- Inflammation of mucous membrane by physical, chemical or bacterial agents as in food poisoning.
- Sensitivity to a particular food as in allergies.
- In nutritional deficiencies such as PEM, Vitamin A deficiencies, pellagra etc.
- In emotional disturbed state as in fear, anxiety or tension.

Diarrhoea may be either acute or chronic in nature.

Acute Diarrhoea

Acute Diarrhoea is characterized by sudden onset and frequent passage of watery and unformed stools. The patient may even pass several stools in an hour. Other symptoms include

abdominal pain, cramps, weakness and sometime fever and vomiting. Acute diarrhoea lasts for only 24-48 hours. Due to danger of dehydration, replacement of water and electrolytes is of prime importance and meeting the nutritional requirements become secondary.

Chronic Diarrhoea

Chronic diarrhoea persists for a longer time even several weeks and the patient may pass 4-5 unformed stools in a day. The rapid passage of food through the intestines does not allow sufficient time for absorption of nutrients and thus nutritional deficiency symptoms may develop. Therefore, meeting the nutritional needs and providing an extra allowance to compensate for the nutrient losses becomes the major objective of the treatment.

How does diarrhoea cause dehydration ?

The body normally takes in water and salts it needs through drinks and food (input). It normally loses water and salt through stools, urine and sweat (output).

When the bowel is healthy, water and salts pass from the bowel into the blood. The water and salts can then be used again by the body. When there is diarrhoea, the bowel does not work normally. Less water and less salts pass into the bowel. Thus, more than the normal amount of water and salts is passed out of the body, in the stools. This large than normal loss of water and salts from the body results in dehydration. The more diarrhoea stools a person passes, the more water and salts he or she loses.

Dehydration occurs faster in infants and young children, in hot chimates and when a person has fever.

Treating a child who has Acute diarrhoea

In acute diarrhoea emphasis is laid on restoration of lost fluid and electrolytes.

The most important factors in the treatment of diarrhoea are :

- To prevent dehydration from occurring, if possible
- To treat dehydration quickly and well if it does occur; and
- To feed the child

Dehydration can usually be prevented in the home if the child drinks more fluids than usual as soon as the diarrhoea starts. A child should be given one of the fluids recommended for home treatment. Food-based fluids, for example gruel, soup, rice, water can be used.

Treatment of dehydration - Oral rehydration therapy (ORT)

If dehydration occurs, the child should be taken to community health worker or health centre for the treatment. The best treatment for dehydration is Oral Rehydration Therapy (ORT) with a solution made with rehydration salts (ORS). It is a simple, inexpensive and effective treatment. The composition of oral rehydration salts (ORS) which are available in a dry packed form, as per the standard WHO/UNICEF formula is given in Table I.

Table 1

Formula of Oral Rehydration Salt

Salt	Amount
Sodium Chloride	3.5 g
Sodium Hydrogen Carbonate	2.5 g
Potassium Chloride	1.5 g
Glucose	20 g

To be dissolved in 1 litre of clean drinking water

The oral rehydration should be started as the child passes even one watery stool, and should be given as frequently as possible.

Other fluids which may be given along with ORS are coconut water, whey water. Cereal/ rice konjee, weak tea. barley water and pulse water.

While the child is ill with diarrhoea, he or she should be frequently offered small amount of nutritious easily digestible foods. Feeding during diarrhoea episode provide nutrients the child needs to be strong and to grow, and helps prevent weight loss. The extra fluids given to the child do not replace the need for food. After the diarrhoea has stopped an extra meal each day for a week will help the child regain the weight loss during the illness.

What foods to be given ?

1. **Fluids** - Give the recommended home fluid or food based fluids such as gruel, soup, or rice water.

   If an infant is breast-fed and try to do so more often than normal (at last every 3 hours). If the infant is not breast-fed than diluted milk is the ratio of 1:1 is given every 3 hours.

2. **Foods** - Weaning starts when the child is 4-6 months old. Give a child of above this age foods with highest amount of nutrients and calories. Fresh fruit juice and banana are useful as they help replace potassium.

Dietary Management of chronic Diarrhoea

The objectives of dietary treatment in chronic diarrhoea are

- to meet the nutritional requirements.
- to replenish water & electrolyte losses.
- to provide extra nutrients to compensate for losses, and
- to reduce residue to minimum levels.

The dietary modifications in chronic diarrhoea are :

Energy - Energy requirements are increased by 10-20% to meet the losses during diarrhoea as well as overcome weakness and loss of weight.

Protein - An increase of 50% in protein intake is essential to build up body tissues and replace the tissue breakdown which has occurred.

Carbohydrates - The intake of carbohydrates should be increased to meet the high energy requirement. However, the fibre intake is kept to a minimum (l-2g/day), to give rest to the intestines.

Fats - Due to increased mobility of the intestines, fats are not completely digested and therefore their intake needs to be restricted. At the same time, some amount of fat is essential to meet the increased energy needs. Emulsified fats like butter and whole milk may be given as they are easy to digest. Coconut oil may also be include in the diet.

Minerals - The absorption of calcium and iron is lowered during diarrhoea. Therefore, calcium and iron rich foods should be included in the diet.

Vitamins - In areas where vitamin A deficiency is common, foods that are rich in Vitamin A are recommended. In any case the absorption of all vitamins is affected during diarrhoea. Vitamin B should also be given in good amount as they are not synthesied immediately after diarrhoea.

Fibre - Low fibre foods should be given, as high fibre diet will irritate the intestine. Some low and high fibre foods are mentioned below.

High fibre food - Whole pulses and cereals, green leafy vegetables and other vegetables like beans, drumsticks, lotus stem, fruits like apple, guava, peas, cherries etc.

Low fibre food - Refined cereals like bread, semolina, washed pulses, vegetables like potato, pumpkin, bottle gourd, fruit juices and fruit like banana and papaya, sugar & oils.

Foods allowed in Restricted Amounts or Avoided

Whole cereals and pulses, raw vegetables and fruits, fried foods nuts, milk & milk based beverages, foods with lots of sugar.

Foods Allowed

Washed pulses and refined cereal, well cooked vegetables, banana, papaya, milk products such as curd, paneer, egg, chicken, fish.

Prevention of Diarrhoea

1) Breast feeding should be encouraged as it is clean and contains substances that protects against diarrhoea. Besides, unhygienic bottles and teets are a source of contamination with microorganisms causing diarrhoea.
2) For weaning purposes, fresh utensils and fresh and properly washed vegetables should be used.
3) Drinking water should be clean with a covered container. It possible, water can be boiled.
4) Mothers hand should be washed after defecation, after cleaning a child who has defecated, after disposing of an infants faeces, before preparing food and before feeding the child.
5) Children's hand should be washed after they defecate and before they eat.
6) Pet and other animals should be kept in pens or otherwise prevented from entering the house, areas where food and water are kept or places where children play.
7) Infants and young children cannot use a latrine. Their faeces should be promptly collected and placed in latrine of buried.
8) Careful attention should be given to hygiene, which includes the building, maintenance and consistent use of latrine by all members of the family.

A Day's Sample Diet Plan for a Chronic Diarrhoea Patient

Personal Data :

Age	:	5 Years
Sex	:	Male
Socio-economic Status	:	MIG
Food habits	:	Non-vegetarian
Pathological Condition	:	Chronic Diarrhoea

Menu Plan

MEAL	MENU
Breakfast	Semolina Porridge
	Boiled egg
	Bread and butter
Mid- Morning	Orange juice
	Biscuits
Lunch	Spinach Khichdi
	Curd
Mid-afternoon	Banana custard
Evening Tea	Paneer & Tomato Sandwich
Dinner	Lentil & Tomato soup
	Moong Dal & Rice
Bed - Time	Phirni

Lesson 9

DIET IN CONSTIPATION

Constipation is a physiological disosder of alimentary canal. It may be defined as the retention of the faeces in the colon beyond the normal length of emptying time. It is characterised by infrequent, incomplete and difficult evacuation of hard, dry stools. Infrequent or insufficient emptying of the bowel may lead to malaise, headache, coated tongue, foul breath and lack of appetite. These symptoms disappear after satisfactory evacuation.

Causes of Constipation. A number of factor leads to constipation,

i. Irregular eating time practices

ii. Change in ones usual routine due to illness, in travel, nervous tension, depression, anxiety, worry or excitement

iii. Faulty dietary habits like inadequate intake of water or fluids, use of highly refined foods,

iv. Lack of sufficient rest

v. Poor muscle tone of small intesline due to lack of exercise and sedentary lifestyles.

vi. Difficult or painful evacuation due to fissures or haemorrhoids

vii. Intake of drugs or medicine

viii. Poor personal hygiene - urge to defecate may be neglected leading to irregular times of evacuation

ix. Excessive and continuous use of laxatives and regular enemas

There are three types of Constipation

i. Atonic constipation

ii. Spastic constipation

iii. Obstructive constipation

(ii) Atonic Constipation is more common. The main reason for atonic constipation are lack of fluids in the body, especially after perspiration, lack of roughage which contributes to the lack of cellulose, deficiency of vitamin B, which produces poor muscle tone, irregular evacuation habits and use of purgative agents. Due to any of the above causes muscular tone of the intestine is affected and peristaltic action is reduced. Bacterial action on stagnated food is more and symptoms of constipation develops.

(ii) Spastic Constipation, on the contrary, occurs due to excessive muscle tone of the colonic muscles. The movement of the food is very irregular and often causes pain in the lower abdomen. Irritating foods, excessive use of purgatives and mental stress produce this type of constipation. Spastic constipation occurs as a complication of some other disease. Excessive use of alcohal, tea or coffee also produce this.

(iii) Obstrutive Constipation is due to maliganancy or stricture of the colon.

Use of castor oil or other purgatives in infancy is a common practice in our country. It contributes to constipation in later life.

Treatment Should attempt at the following

1 Correction of faulty food habits.

2 One should follow regular bowel movements habits

3 The patient should be advised to take exercise for the development of abdominal muscles. Regular physical exercise is beneficial especially for sedentary workers.

4 Mental worry and anxiety should be avoided.

5 Consume a high fibre diet.

6 Ensure an adequate intake of fluids.

Modification of Diet

A well balanced diet with B group vitamins and liberal fluid intake is recommended. Fibre or cellulose content of the diet must be high. High fibre foods like whole grain cereals & legumes, green leafy vegetables and other vegetables and other fibres foods should be eaten is large amounts. Bland cooking is preferred. In spastic constipation high roughage is harmful. Fat containing foods are useful for some because of their lubricating effect and stimulating action of the fatty acids on the mucous membrane. A normal diet with light mental attitude and proper exercise or physical activity is recommended for a constipation patient.

For spastic constipation, a soft bland diet is recommended. Small meals prevent stagnation of food mass in intestine. Vitamin B supplements are essential. Daily 8 to 10 glasses of water must be ingested to help stool formation.

Foods to be Included : Whole cereals, whole legumes, fruits like guava, apple, oranges, bananas, grapes and green vegetables.

Foods to be Avoided : Highly refined foods and foods which leave very little residue such as maida, meat, rice and candies.

Constipation among children :

Continuous usage of castor oil or purgatives by infants and children to clean the bowels affect the muscle tone of the intestine. This leads to constipation and interfere seriously with the absorption of fat soluble vitamins - vit A & K. Instead of purgatives lots of water, fruit juices and fruits like banana, apple or guava or other fibrous fruits are better.

In cases of chronic constipation, inert laxatives like hydrophilic colloids i.e. isabgol & agar agar may be helpful, because in the presence of water they swell up ands increase feacal volume.

A Day's sample Diet plan for a constipation Patient

Personal Data

Age	-	42 Yrs
Activity	-	Sedantry
Socio-economic status	-	Middle income group
Food habits	-	Non Vegetarian
Pathological Condition	-	Constipation

Menu Plan

Meal	MENU
Early Morning	Hot water with lemon juice.
Breakfast	Wheat dalia Omelette Brown bread Tea
Mid Morning	Apple
Lunch	Chapati Rajma Curry Carrots & Pea subzi Spinach Raita Cucumber & tomato salad
Evening Tea	Coffee Guava, papaya, sweet potato & cucumber chaat
Dinner	Sweet corn chicken soup Vegetable noodles Sweet & sour paneer & vegetables Orange and Cabbage and salad greens
Bed Time	Milk

Lesson 10

OBESITY

Obesity is the most common nutritional disorders of the affluent countries as well as the high socio-economic groups of our country. Obesity can be defined as generalized accumulation of excess fat in the body. In clinical learns, it is defined as a condition of excess body weight i.e. when a person is 20% or more above the ideal weight. Overweight term is at times used to refer to individuals with body weight 16-20% an excess of ideal body weight.

Excess of fat is as much a hindrance as carrying a load of the same weight day and night. It gives rise to breathlessness on moderate exertion. Obesity predisposes to diseases like angina pectoris, coronary thrombosis, high blood pressure, diabetes, gall bladder diseases and osteoarthritis of weight bearing joints. Surgery life expectancy dismisses with excess weight.

Assessment of obesity

(i) **Standard Weight for Height Measures:** These are based on weight ranges associated with least mortality rates and serve as only a rough guide for estimating desirable weight. They may not be the representative of the total population and so must be used with discretion.

(ii) **Body Mass Index:** This is a measure of relative body fatness to evaluate the risk factors associated with obesity. It is based on weight (in Kg) and height (in meters) with minimum clothing and no shoes.

$$BMI = \frac{Wt\ (kg)}{Ht.\ (Mt)^2}$$

BMI value indicative of Obesity are given in Table 1

Table 1

Grading of Obesity by BMI

Grade	BMI (kg/m2)
Not obese	25
Grade I	25-29.9
Grade II	30-40
Grade III	>40

(iii) **Skin Fold Thickness:** Measurement of skin fold thickness has been frequently used as a measure fat deposited under the skin. (Sub-cutaneous). As the obesity increases, the thickness of fat increases. Sub-cutaneous fat is about 14% of body weight in man and 18% in women. The thickness of fat is measured at various sites with skin calipers. The limitation of using skin fold measurement is, that, there is no agreement on the number and sites that best reflect the actual body fat content.

Etiology

Obesity is either due to excessive energy consumption or decreased energy output resulting in positive energy balance. The main contributing factors can be discussed as follows:

1. **Physiological Factors**

(i) *Age and sex:* The normal physiology of growth and development during the life cycle contributes to accumulation of adipose tissue. Critical periods for the onset of obesity are early childhood and adulthood; when no diet adjustments are made for decreased activity. In women obesity normally occur during pregnancy and after menopause.

(ii) *Endocrinal Factors:* Obesity normally occurs at puberty, pregnancy and at menopause. It clearly suggests the role of harmons for the occurrence of obesity. Obesity is also associated with hypothyroidism or myxoedema. Crushing syndrome or over creation of the adrenal gland leads to obesity in which excess fat is laid on the trunk and usually not on the limbs. Intake of oral contraceptives may lead to obesity, as these are hormonal in nature. Endocrine factors, however, contribute to less than 5% of the total cases of obesity.

2. **Genetic Factors**

The role of genetics in obesity is not will understood, neither the genes responsible for it have been identified. However, it has been observed that the chance of obesity in a child increases, if both the parents are obese. However, the dietetic habit of a family rather than hereditary factors may be responsible for obesity.

3. **Psychological factors**

These may be the etiological as well as the aggravating factors for obesity. In some cases eating is a compensatory mechanism to attain self satisfaction, when there has been failure or frustration is life. Tension, anxiety and the humiliation associated with being obese may further make a person resort to food for emotional satisfaction.

4. **Eating Habits**

Over eating is the prime factor in obesity. Any excess of ingested calories over energy expenditure is stored as fat. Nibbling in between meals, eating at night due to insomnia and consumption of refined, starchy and fatty foods contribute to a high energy intake. An extra slice of bread or a banana provides 50-100 calories and such slight excess, amounts to a considerable accumulation in the course of time. From a variety of foods, an obese person usually chooses fried potatoes, while a thin person prayers vegetable salad.

5. **Physical Activity**

Obesity is most common after the age of 35 years. Most of us do more physical work and also take more exercise before this age than, in later life. With the proceed of middle age, promotion to executive jobs involves longer hours at the desk with less physical work. Food consumption either remains the same or may even increase with the improved economic status.

6. **Social factors**

Social pressures and need for eating out and attending parties is a common cause of excessive intake of food and energy.

Consequences of Obesity

As already discussed obesity leads to mechanical disabilities, predisposes to renal, metabolic and cardiovascular diseases and reduces life expecting, as well as psychological factors.

a) **Psychological Factor**

These may be the etiological as well as the aggravating factors of obesity obese people after feel humiliated and unhappy. This further leads to eating for solution. Thus it's a vicious cycle where psychological factors leading to obesity and obesity leading to psychological factors.

b) **Mechanical disabilities**

The extra load of the body may cause flat feet and arthritis leading to pain in hips, knees and spine. It also results in varicose veins and abdominal hernias. Fat is deposited under the diaphragm and on the chest. This interferes with the normal respiration and predispose to bronchitis. It also causes carbon dioxide retention and drowsiness.

Predisposes to other diseases

Obesity contributes to a number of health related problems namely hyperlipidemia, hypertension, diabetes, pulmonary and renal problems and pregnancy complications. Alteration in lipoprotein levels have been observed in obese people, which in turn is related to increased risk of developing coronary atherosclerosis. Certain cancers such as cancer of gall bladder, biliary tract, ovary, breast and cervix cancer in women and cancer of colon and prostate in men have been associated with obesity.

Obesity is also associated with complications in pregnancy. The frequency of toxemia and hypertension has been shown to be greatly increased and duration of labour is usually longer in overweight women. The chances of caesarean delivery are increased with increased body weight.

Reduced Life Expletory

Obesity, because of its association with several other diseases and risk factors decreases life expectancy.

Treatment

Successful weight loss can be brought about through a combined nutrition and behavioral approach. The food plan should be well balanced and suited to the particular needs of the individual, together with constant exercise for effective results.

Dietary Management: The objective of the dietary modification in the treatment of obesity ares

a) to bring about a gradual weight loss. A low calorie diet is designed to reduce the weight by 3-4 Kg per month till the ideal body weight is reached.

b) To main a desirable weight and a good nutritional status. A moderate diet is designed to maintain the ideal weight for the three to six months.

c) To control faulty food habits - A revision to the usual diet with such a mental reorientation that foods of high calorie value are taken in small judicious helpings

Energy

The level of energy is adjusted to individual weight reduction requirements. A decrease of 1000 Kcals daily is required to lose about 1 kg a week and reduction of 500 Kcals brings about a weight loss of about 1/2 kg. a week.

An average low calorie diet prescribed for women is 1000-1200 K cals. and for men is 1500-1800 Kcals. per day. A drastic restriction of energy is not advisable as it leads to hunger, nervous exhaustion, weakness and inadequacy of other nutrients. The patient is checked every week for weight loss and a weight loss of 3-4 kg is expected by the end of the month. Table 2 gives the value of energy needs per kg IBW based on activity. It can be used for energy intake of any individual.

Table 2

Energy requirement (Kcals/kg Ideal Body wt./day)			
Activity	**Obese**	**Normal**	**Under Weight**
Sedentary	20-25	30	35
Moderate	30	35	40
Heavy	35	40	45-50

Protein: Normally a slightly higher protein diet gives a feeling of satiety and also helps to maintain a good nutritional status. Approximately 20% of the total energy must be provided from protein. These should include good dietary protein in the form of low fat milk, lean meats and whole pulses.

Fat: Energy provided by the fat should be 20% or less. Fat used should be unsaturated to avoid heart related ailments. Fried foods are strictly prohibited.

Carbohydrates: Carbohydrates should provide about 60% of the energy. These should mainly be complex carbohydrates like starches and dietary fibes. Use of simple carbohydrates like glucose and sugar should be avoided

Minerals and Vitamins: Adequate minerals and vitamins should be provided by the diet. The diet should include ample fruits and vegetables as they are low in energy, but a good some of vitamins and minerals.

Fibre: To avoid constipation, the diet should have enough fibre by taking bulk producing vegetable and fruits.

Fluids: Enough fluid in the form of water and other liquids should be given to avoid constipation. It also gives a fuller stomach feeling.

Foods to be used in restricted amounts or avoided

1. High carbohydrate foods such as bread, cakes, cookies, dried fruits, rich pulses, potato, sweet, honey, syrups, jams, puddings, chocolates, sugar etc.
2. Carbonated and matted beverages, alcoholic drinks and sweetened fruit juices.
3. High Fat foods like butter, ghee, fried foods, ice-creams, processed cheese, potato chips, nuts, pastries, rich salad dressings etc.

Exercise: Aerobic exercise consists of activities that are sustained long enough to draw on body's fat reserve for fuel while increasing oxygen intake. Lean body mass burns fat in presence of oxygen. Aerobic exercise helps in reducing weight by suppressing appetite and thus reducing food intake and by increasing BMR and hence energy expenditure. It also lowers set point and reduces body fat while retaining lean body mass.

A sample Diet

A Day's sample Diet for An obese female aged 39 years having a sedately life style with a weight 65 kg and height 5' 3".

Menu :

Meal	Menu	Amount
Early Morning	Tea without sugar	1 cup
Break fast	Skimmed Milk,	1 cup
	Boiled egg.	1
	Toast without butter	1
Mid-day	Watermelon	1 cup
Lunch	Chappati	1
	Thin Arhar Dal	$^3/_4$ katori
	Skimmed milk curd	1 katori
	Salad without dressing	1 plate
Tea	Tea without sugar	1 cup
	Sprouted Moong	$^3/_4$ katori
Early Dinner	Chappati	1
	Chana Dal with	1 katori
	Bottle ground	
	Fruit salad	1 plate
	Kheer with sugar free	1 katori

Lesson 11

DIABETES MELLITUS

Diabetes Mellitus is a chronic disease that has affected mankind thoughout the world. The records of the ancient civilization of Egypt, India, Japan, Greece and Rome describe the symptoms of this disease.

It is a metabolic disorder *characterised* by the decreased ability or complete inability of the tissues to utilize carbohydrates, accompanied by changes in the metabolism of fat, protein, water and electrolytes. This defect is due to deficiency or diminished effectiveness of hormone insulin. The insulin affect may be a failure in its formation, liberation, or action since insulin is produced by the B-cells of the Islets of Langerhans in pancreas, any reduction in the number of functioning cells, will decrease the amount of insulin that can be synthesized.

Prevalence:

Diabetes Mellitus is an important public health problem all over the world. In India the prevalence of diabetes in India is about 2.1% in the urban population and 1.5% in the rural population. This is about 2% of the total population in our country. Besides, there is an equal number having undetected diabetes. Urbanisation and increasing prosperity have raised the prevalence rates.

Type of Diabetes

Diabetes Mellitus (DM) is of there types:

i) **Insulin Dependent Diabetes Mellitus (1DDM) or Type I :** It usually occurs is much younger adults, normally prior to the age of 20 yrs. This disease usually has an abrupt onset and the abnormality of carbohydrate metabolism is severe due to lack of endogenous insulin to control blood glucose levels. Insulin therapy is a must for these patients. The patients are usually obese and may even be wasted and underweight. This type is difficult to manage.

ii) **Non-Insulin Dependent Diabetes Mellitus (NIDDM) or Type II :** This type normally occurs in middle mostly after 4 yrs. of age, but may occur before also. The onset is gradual and there is some amount of endogenous insulin. Majority of the patients are obese, and improve on weight loss and can be maintained on diet therapy. The treatment is usually assisted by oral hypoglycemic drugs.

iii) **Malnutrition Related Diabetes Mellitus (MRDM) :** In this case Diabetes Mellitus is secondary to other conditions such as pancreatic disorder, protein deficiency in early years etc.

AT RISK OF DIABETES MELLITUS

Besides the known cases of DH, there are others who may be at risk of developing DM. Some of these risk factors are discussed below:

(i) **Impaired Glucose Tolerance (IGT) :** Patients suffering from impaired glucoses tolerance are regarded as 'at risk'. Risk factor should, therefore, be minimlised in such patients to prevent development of diabetes.

(ii) **Gestational Diabetes Mellitus (GDM) :** It is diagnosed when glucose intolerance is detected for the first time during pregnancy. It does not apply to women known to be diabetic before conception.

(iii) **Latent Diabetes :** Subjects who have normal glucose tolerance but had a temporary period of abnormal glucose tolerance earlier. Such a period of glucose intolerance might have been precipitated by stressful situation like infection or trauma.

(iv) **Potential Diabetes :** Subjects with potential abnormality of glucose tolerance like children, twins, siblings of known diabetics; women with bad obstetric history or with a history of having delivered infants of high birth weight; ethnic groups with a high predisposition for diabetes and obese subjects with a strong family history. These subjects have a potential abnormality of glucose tolerance and therefore, are at a risk of developing diabetes.

Etiology

The Main causes are:

(i) Heredity: There is a familial tendency to develop diabetes but the specific biochemical defect and the mode of inheritance has not been identified. The tendency of diabetes is believed to be inherited as a Mendelian recessive, characteristic. If both parents are diabetic, all their children will be diabetic. If one parent is a diabetic and the other is a diabetic carrier, half of their children will be potential diabetics. If one parent is a diabetic and the other a non-diabetic and also a non-carrier, none of their children will be diabetic, but they all will be carriers. If these children and their children consistently marry non diabetics, the carrier tendency will eventually disappear. However, environmental and other factors play a role in unmasking an underlying diabetics genotype and determine whether a person with a genetic predisposition actually develops the disease or not.

(ii) **Age:** The disease may occur at any age but about 80% of the cases occur after the age of 50. Diabetes is thus mainly a disease of the middle aged and elderly.

(iii) **Sex:** In the younger age groups, diabetes is more commonly seen in males than in females. In middle age, women are more affected and the chances increase with pregnancy.

(iv) **Obesity:** There is a strong association between diabetes and obesity but it is uncertain whether obesity is the result or the cause of diabetes. The majority of middle-aged diabetics are obese, while only a minority of obese people develop diabetes. Whether an obese individual develops diabetes or not is probably dependent on genetic factors. Obese people are also less physically active than normal weight individuals which increase the risk of diabetes.

(v) **Dietary factors:** A high intake of sugar has been related to obesity and may predispose to diabetes as seen in Indians in South Africa. A low intake of fibre due to consumption of refined foods is associated with high prevalence of diabetes in prosperous communities. The specific effect of fibre in reducing the risk of diabetes is, however, not clearly defined.

(vi) **Infections:** Infections, particularly staphylococcal, may unmask latent diabetes due to production of hormones that are antagonist to insulin. There is increasing evidence of Type I diabetes following viral infection, which brings about an autoimmune reaction that destroy the B-cells of pancreas and impairs insulin secretion.

(vii) Stress: Physical injury or emotional stress may be a cause of unmasking latent diabetes by eliciting Adrenocortical response. Surgical operation and severe infections may, therefore, be contributing factors in precipitating the disorder.

Secondary Diabetes- A minority of diabetes occur as secondary to some other disorders. These are due to diseases (i) which destroy pancreas and lead to impaired secretions and release of insulin or (ii) Abnormal concentration of certain hormones in the circulation which are insulin antagonist e.g. Growth Harmone, Adrenocortical, Hormone, Adrenaline, Thyroid Hormone.

Clinical Pathology

The normal blood glucose level is 70 to 120 mg/100 ml. when this level is lowered, the cells starve and die due to lack of energy while in case of hyperglycemia, the body's life sustaining water, electrolyte and acid base balances are disturbed. To maintain a normal blood sugar level, a balance is kept between the

- entry of blood glucose from dietary carbohydrates, protein, fat and liver glycogen, and
- removal of blood glucose to prevent a rise above 120 mg/100 ml.

Insulin, The hormone secreted by the Beta-cells of the Islets of Langerhans of the pancreas helps in regulating blood glucose in the following way :

- Insulin facilitates transport of glucose across the cell membranes through specialized insulin receptors, which are located on insulin-sensitive cells in the adipose tissue. The receptors have been found to be fewer in obese diabetics and their number increases with exercise and weight loss.
- Insulin brings about glycogenesis i.e. conversion of glucose to glycogen for storage in the liver.
- Insulin brings about conversion of glucose to fat (lipogenesis)
- Insulin inhibits lipolysis and breakdown of protein.
- Insulin increase protein synthesis by promoting uptake of amino acids.
- Insulin brings about glycolysis i.e. oxidation of glucose for release of energy.

Metabolic Changes in Diabetes Mellitus

In diabetes, insulin secreted by the pancreas is either insufficient or ineffective. This may be due to a primary disorder of insulin secretion or due to insulin resistance because of a receptor defect in the target tissues. As a result, glucose cannot be oxidized propertly through the main glycolytic pathways in the cells to furnish energy and its leveling in the blood rises, leading to **hyperglycemia.**

When the glucose concentration in the blood exceeds the renal threshold or the capacity of renal tubules to reabsorb glucose from the glomerular filtrate, glocosuria i.e. presence of glucose is the urine, occurs. In most of the people this occurs when blood glucose level is about 180mg/ 100 ml. Increased concentration of glucose increases the osmolarity of the glomerular tubules. This increases the volume of urine causing polymeric and nocturia. Loss of water lead to excessive thirst i.e. polydipsia. Due to high blood glucose levels, the extra cellular fluid becomes hypertonic and water comes out of the cells ultimately leading to dehydration.

Impaired utilization of carbohydrates leads to intra-cellular lack of glucose. This brings into effect two compensatory reactions so as to provide metabolic substrate. Both lead to loss of body tissue and wasting despite of food intake being normal or even increased.

Symptoms

Onset of diabetes or an uncontrolled state, the following symptoms observed

1. Polyuria or increased urination

2. Polydipsia or increased thirst as a result of excessive water loss.
3. Polyphagia or increased hunger due to a failure to utilize food for nourishment of the body.
4. General weakness.
5. Decreased resistance to infection especially staphylococcal infection and T.B.
6. Deceased ability of wound healing due to a high blood sugar and poor fluid balance.
7. Dehydration as a result of excessive water and electrolyte loss from the body, causing dry furred tongue and cracked lips.
8. Vulvitis or balnitis due to an infection of the external genitalia by a fungus (Candida) which flourishes on skin and mucous membranes having a high concentration of glucose.
9. Ketosis or ketoacidosis i.e. accumulation of ketone bodies in the blood as a result of increased lipolysis. Breathing may be deep and rapid and the breath has acetone smell. Ketosis, if not controlled, may lead to coma and finally death.
10. Degenerative changes in advanced cases include peripheral neuritis, retinitis, atherosclerosis with associated diseases of coronary arteries and vascular changes in kidneys causing nephropathy. Associated symptoms are failing or blured vision, pain, numbness of the limbs and proteinuria.

Biochemical Changes

Laboratory tests show:

(i) Hyperglycemia or elvated blood sugar levels

(ii) Glycosuria or presence of sugar in the urine as a consequence to hyperglycemia

(iii) Ketonemia or elevated levels of ketone bodies in the blood

(iv) Abnormal glucose tolerance tests.

Dietary Management

Basic objective in the care of a diabetic person is to maintain and prolong a healthy and productive life. The aims are:

- To maintain optimal nutrition so as to provide for adequate growth development and maintenance;
- To achieve and maintain a desirable body weight;
- To provide relief from symptoms;
- To maintain normal blood sugar levels and
- To prevent, delay or minimize the onset of chronic degenerative complications.

Diet therapy has a major important role to play in realising these aims. Success or failure of the treatment depends to very large extent on patient. Self-discipline and self-control under the guidance of the doctor and nutritionist are needed for sound management. The patients have to ultimately treat themselves and therefore, educating them on the objectives of treatment is extremely important. Counseling the patient so as to make him understand the problem, reassurance, proper instructions teaching means of self-care and monitoring go a long way in controlling the disorder.

A therapeutic diet plays an important role in the treatment of diabetes. The diet may be used alone or in combination with insulin injections or oral hypoglycemic drugs.

The diet plan of an individual is based on height, weight, age, sex, physical activity and nature of diabetes. The following are the essential considerations:

1. Determining energy requirements.
2. Distribution of energy in terms of carbohydrate, fat and protein.
3. Determining the type of carbohydrate, fibre and type of preparations.
4. Distribution of carbohydrate.

Determining energy requirements

The energy value of the diet and its proportionate distribution needs to be calculated for each diabetic patient individually. In case of IDDM or Type 1, the kilocalories prescribed are based on needs for normal growth and development, physical activity and maintenance of desirable body weight.

In case of NIDDM or Type II, as the majority of cases are obese, the kilocalories adjustment is made to achieve weight loss.

This can be done in the following way:

a) Calculation of ideal body weight (IBW) on the basis of weight and Height.

b) Determining energy intake on the basis of kcal prescribed per kg IBW according to activity and whether the individual is normal weight, obese or underweight.

Distribution of energy in terms of carbohydrate, fat and protein

The ratio of carbohydrate, protein and fat in the diet should be such that it maintains blood glucose and reduces the risk of cardiovascular diseases.

a) The protein allowance is essentially the same as that for a normal individual and may vary from 1.0 to 1.5 g/kg of desirable body weight. Proteins should provide about 15-20% of the total energy in the diet as compared to proteins providing normally about 10% of total energy. Slightly higher than normal protein are given, as in poorly regulated diabetes large quantities of nitrogen are excreted in the urine. Another reason for prescribing higher protein is that the metabolism of protein to glucose is slower and hence there is less available glucose. Children require enough protein to meet growth and development needs.

b) The carbohydrate allowance is estimated on the basis of the patient's bloods sugar, urine analysis, and available insulin. A moderate restriction of carbohydrate calories is made to about 55-70% of total calories. An amount less than 100g carbohydrate per day is not advisable as it leads to ketosis. On the other hand, more than 300g carbohydrate per day may overburden the metabolic capacity. Not only the total amount of carbohydrate but the type of carbohydrate and its distribution between different meals is extremely important and depends on the type of treatment being followed.

c) The fat allowance of the diet is calculated so as to provide the rest of energy. Total fat intake is lowered to 20% or less of the day's energy intake in case of obese, adult diabetics. As diabetic patients have an increases risk of atherosclerosis, the total amount of fat must be restricted. At the same time, polyunsaturated fats should be given rather than saturated ones.

d) Mineral and vitamin requirements of patients with well controlled diabetes do not differ much from those of normal subjects. Neuritis, a frequent complication in diabetic patient is prevented by liberal intakes of B-group vitamins especially thiamine; Vitamin

and mineral supplements are not needed if the diet is well planned and nutritionally adequate. Alcohol may be allowed only in moderation at the same time, its energy value and the carbohydrate content must be taken into account. Beer contains about 20 to 60g of carbohydrate per litre. Sweet wines and cider have a high sugar content, while whisky and gin although free of carbohydrates, provide about 70 kcals for every 30ml.

Type of carbohydrate and amount of fibre and types of preparations

More of carbohydrate must be given as complex starches rather than simple sugars as they breakdown more slowly to releases glucose. The presence of fibre in complex carbohydrate like grains, vegetables and other starches increases intestinal transmit time, delays gastric emptying and slow glucose absorption. Soluble fibre present in oats, barley, fruits and legumes has been shown to lower fasting blood sugar and glycosuria and improve sensitivity to insulin. Some of the high fibre foods are whole cereals like buck wheat (kootu), barley, ragi and oats, leafy vegetable, beans whole pulses and legumes. Refined foods like maida, suji arrowroot, sago, juices, etc. are low in fibre and should be avoided. Supplementation or cereals with gram is beneficial. For example using three part of whole wheat flour with one part of gram flour in making of chapattis; one part rice with one part whole pulse to make idli, dosa, khichri and two part suji with one part of pulse to make upma. The rise of blood sugar after a meal does not merely depend upon the amount of carbohydrate ingested but also on the rapidity of absorption which varies with the fibre content, phylate, lactins and enzyme inhibitors.

Meal Intervals

In all diabetics, the amount and the time of food intake particularly the carbohydrate, should be conferred to prevent fluctuations.

Foods not Allowed

Glucose, sugar, honey, all sweets, chocolates and candies.

Foods to be avoided or restricted

Potato, yam, arbi, sweet potatoes, Mangoes, grapes, bananas, alcoholic beverages, fried goods, poories, pakoras, dalmoth, mathris, deep fried vegetable, dry fruits, rich salad dressings cakes and pastries

Foods to be used freely

Green leafy vegetables, tomatoes, cucumber, reddish lenous, clear soups, black coffee and tea without sugar, butter milk, pickles without oil.

A Day's sample Diet for a Patient with NIDDM

Plan a day's diet/menu for a 49 years old men, this built 5'7" weighing 64 kg and suffering from Type II DM. He is a sedentary worker and a vegetarian.

Personal Data

Age	-	49 years
Sex	-	Male
Work	-	Sedentary
Eating Habits	-	Vegetarian
Physiological condition	-	Diabetic Mellitus Type II

Recommended Dietary Allowances

Energy

i)	Estimate IBW for heigh	=	48 kg + (2.7 x 7 kg)
		=	48 + 18.9
		=	67 kg.
ii)	For IBW estimate energy	=	67 x 30
	for Normal adult	=	1910 cals.

The patient's weight is 3 kg less than required but for a diabetic patient 10% less weight is preferable. There for no adjustment for weight gain will be made.

Protein	20% of energy	=	0.2 x 1910	=	95g
Fats	20% of energy	=	0.2 x 1910	=	45.5g
Carbohydrates	60% of energy	=	0.6 x 1910	=	285 g
Calcium		=	400 mg		
Thiamine		=	0.95 mg		

Menu Plan

Menu	Food stuff	Amount
Early Morning	Tea without sugar	1 cup
Breakfast	Cracked wheat porridge	1 medium bowl
	Boiled egg	1
	Toast with Paneer	1
	And tomato	
	Tea without sugar	1 cup
Mid Morning	Guava/Apple	1
Lunch	Chappati	3
	Arhar Dal	$^3/_4$ katori
	Bottle gourd vegetable	1 katori
	Salad cucumber, tomatoes.	1 plate
	onion, raddish	
Mid day	Tea without sugar	1 cup
	Idli/sandwich	2
Dinner	Chappati	2
	Cauliflower	1 katori
	Spinach & Paneer	1 katori
	Curds	1 katori
	Salad, cucumber, corn.	1 plate
	Tomato	
Before Bed	Milk without sugar	

LESSON 12

HYPERTENSION

Hypertension or elevation of blood pressure above normal is a symptom that accompanies may cardiovascular and renal diseases. It can involve many organs and systems including the heart, endocrine glands, kidney and central and autonomic nervous system. It has been clearly shown to increase the risk of developing stroke, coronary heart disease, congestive heat failure, peripheral vascular disease and nephrosclerosis.

Blood pressure is a continuous or graded phenomenon and the risk of hypertension increases speedily with blood pressure level - either systolic blood pressure (SBP) or diastolic blood pressure (DBF). Therefore, any definition of elevated blood pressure is arbitrary and serves to classify people into risk categories. Table I shows the classification of blood pressure in adults-18 years & above.

Table 1

Classification of Blood Pressure in Adults 18 Yrs. & Older*

Blood Pressure Range	Classification
Diastolic Blood Pressure	
< 85	Normal Blood Pressure
85 to 89	High Normal Blood Pressure
90 - 104	Mild Hypertension
105 - 114	Moderate Hypertension
> 115	Severe Hypertension
Systolic Blood Pressure (when DBP > 90)	
140	Normal Blood Pressure
140 - 159	Borderline isolated systolic hypertension
> 160	Isolated systolic hypertension

*Adopted from JNC (1988)

Etiology

More than 90 percent of the people do not have identifiable cause of elevated blood pressure and are said to have *'Primary',* or *'essential'* hypertension. Rest of the people with hypertension do have an identifiable cause and are said to have *'secondary'* hypertension. *Secondary hypertension* may be due to

- Renal diseases
- Use of oral contraceptives in women
- Endocrine diseases such as hypothyroidism, hyperaldosteronism. Factors responsible for primary hypertension are diet related or lifestyle related and can be linked as risk factors, which are discussed below.

1) **Age** : Systolic blood pressure rises steeply from infancy to adulthood and levels off once adult height is reached. Studies have shown that SBP increases on an average by about

20mm Hg between ages 20 and 60 an additional 20mm Hg between the age of 60 and 80. Diastolic blood pressure rises approximately *10 mm Hg* between the age of 20 and 60 and gradually declines thereafter.

2) **Sex** : Higher prevalence of hypertension has been found among males from adolescence till 45 years of age. After this age, mean blood pressure values are higher in women.

3) **Heredity** : A genetic predisposition to hypertension is generally there for humans, although it has not been determined whether it is via a single gene or is a polygenic inheritance.

4) **Social and Cultural Influences** : People moving from rural to more urbanised way of live usually show an increase in blood pressure. Little is known how these factors influence.

5) **Exercise & Activity** : Some studies suggest that increase physical activity and exercise have a long term hypertensive effect, either independently or in association with diet. However, the data is not very conclusive.

6) **Dietary Factors** : Some of the dietary factors such as total energy, dietary fat, dietry fibre, sodium, potassium, alcohol and caffeine have been linked with high blood pressure.

   a) Energy: Increased energy intake has been associated with increase blood pressure.

   b) Dietary Fat: The ratio of polyunsaturated to saturated fat (P/S ratio) in the diet may affect blood pressure. Increasing the P/S ratio to 1 or more with app. 25% of energy as fat, has been associated with lowering blood pressure in hypertensive patients. This effect is independent of sodium balance or body weight.

   c) Dietary Fibre: Several studies have suggested that increased dietary fibre intake may lower blood pressure. Vegetarians who diet are usually high in fibre generally have lower blood pressure but a number of other differences in vegetarian and non-vegetarian diets may also account for this variation.

   d) Sodium: Since 1920, studies of isolated populations have suggested an association between sodium chloride intake and blood pressure. Deprivation of sodium has led to fall in blood pressure in hypertensives.

   e) Potassium: It has been suggested that potassium salt supplementation in hypertensive subjects lowered their blood pressure, especially the ones sensitive to sodium chloride.

   f) Calcium & Magnesium: High dietary calcium intake has been associated with lower blood pressure in men. A number of studies have also suggested that low dietary magnesium level may be associated with higher blood pressure and large doses of magnesium salts may lower blood pressure.

Alcohol: A strong association exists between moderate to heavy alcohol consumption and hypertension. The effect of alcohol consumption on blood pressure appears to be more pronounced in the elderly compared to the young.

Caffeine: Caffeine present in coffee can produce a short term significant increase in both SBP and DBF, but its long term affect is not clear.

Symptoms

Most patients with hypertension have no symptoms and the condition is discovered in the course of a routine medical examinations. Common symptoms observed are the headache, dizziness, impaired vision, failing memory, shortness of breath, pain and gastrointestinal disturbances. Extent of the symptoms depend on the elevation of the blood pressure and the length of the time it has been present.

Treatment

Patients suffering from secondary hypertension should be treated for the underlying cause.

Patients suffering from primary hypertension generally do not have any overt symptoms and continue to lead a normal life. Hypertension is a chronic, life long condition and may lead to the development of CHD and stroke. Therefore, the treatment is directed towards controlling the blood pressure within the normal limits by, the use of drugs diet and behavior modification. In mild to moderate hypertension, this may be achieved by the diet restriction, controlling weight, cessation of smoking, alcohol restriction, regular physical activity and moderation in life.

However in serve hypertension, there is danger of the involvement of other vital organs, therefore, drugs such as diuretics and Beta blockers are used along with the above measures to reduce the elevated blood pressure.

Dietary Management

Objectives of Diet Management are

i) To achieve a gradual weight loss in overweight and obese individuals and maintain their weight slightly below the normal weight,

ii) To reduce the sodium intake,

iii) To maintain adequate nutrition .

To achieve the above objectives the diet modified is as follows :

i. Energy : Hypertension is often lowered with weight loss in obese patients. Even a 5 to 6% weight loss is sufficient to produce a substantial fall in blood pressure in overweight persons with mild hypertension. Even normal weight hypertensive persons benefit with slight reduction in weight. Therefore, energy intake should be adjusted in such a manner so as to bring about weight loss and maintain it slightly below the normal level.

ii. Protein: Protein should contribute about 15% to 20% energy in a low energy diet and thus it is recommended that the diet of a hypertensive patient should have about this much protein. Vegetable protein from legumes and pulses are high in protein and fibre and low in sodium, should be included in the diet. Animal protein are rich in protein as well as fat, therefore, should be used judiciously.

iii. Carbohydrate: Simple Carbohydrates like sugar and glucose should be avoided. Complex carbohydrate from whole legumes, pulses, vegetable and fruits should be included. As such, carbohydrate should account for 60-65% of the energy.

iv. Fats: Fats should not provide more than 20% of the energy in the diet. Since it is important to raise the P/S ratio to 1 or above, saturated fat should be avoided and should be replaced by unsaturated fat.

v. Sodium: Sodium restriction is affective in lowering blood pressure when used alone. However, except for the patient with mild hypertension, the level of sodium permitted is so low as to be impractical for most. The primary means of treating hypertension is through diuretics and a mild restriction of sodium is recommended.

vi. Potassium & Calcium: An adequate amount of potassium is an essential part of hypertension treatment. It can be achieved by including sufficient amounts of food rich in potassium such as milk, fruits and vegetables. Some studies have suggested that

optimum intake of calcium has a beneficial effect in hypertension, hence, calcium rich food like milk, leafy vegetable should be included in the diet.

A. SAMPLE DIET IN HYPERTENSION

Plan a day's diet for a hypertension female 50 Years old weighting 65 Kg and is 5'2" tall. Her blood pressure is 150/90 mm Hg. She has been advised to reduce her weight. She needs a sedentary life and is a vegetarian.

I. **Persona Data**

Age	:	50 Yrs.
Sex	:	Female
Weight	:	65 Kg
Height	:	5''2'
Activity	:	Sedentary
Food Preference	:	Vegetarian

II. **Recommended Dietary Allowances**

a) Energy Requirement

(i)	Estimate IBW for height	:	100lbs + (2x5 lbs)
		=	110lbs = 52 Kg
(ii)	for 1BW, estimate energy	=	52x30 K cal/Kg
	required	=	1560 Keals.
(iii)	Estimate weight above 1BW	=	65 Kg - 52 Kg
		=	13 Kg
(iv)	Estimate energy required to	=	1560+(8.8 Kcals above 1BW)
	maintain present weight	=	1560+115 = 1675 Kcals.
(v)	Estimate energy required to	=	1674 - 500
	loose weight	=	1174 Kcals

This will result in a weight loss of approximately $^{1}/_{2}$ Kg/week.

B) Approximate distribution of energy from Protein, Fats and Carbohydrates

Protein (20% of energy) = $0.2x1174/_{4}$ = 59g

Fats (20% of energy) = $0.2x1174/_{9}$ = .26g

Carbohydrates (60% of energy) = $0.6x1174/_{4}$ = 176 g

C) Sodium = 500mg to 1000mg

Menu

Menu	Food Stuff	Amount
Early Morning	Tea without sugar	1 Cup
Breakfast	Wheat Poriddge	1 Bowl
	Toast without Butter	1
	Tea without Sugar	1 Cup

Mid Day	Fruit	1 Bowl
	Orange/Watermelon	
Lunch	Chapati	1
	Chick Pea Curry	$^{3}/_{4}$ Katori
	Pumpkin Vegetable	1 Katori
	Curd from skimmed Milk	1 Katori
	Salad (Cucumber, Tomato,	1 Plate
	Lemon, Onion)	
Evening Tea	Tea without sugar	1 Cup
	Moong Sprouts	1 Bowl
Dinner	Chapati	1
	Spinach & Paneer Veg	$^{3}/_{4}$ Katori
	Bottle Gourd Vegetable	1 Katori
	Fruit Salad	Papaya/Apple

Lesson 13

FOOD ADULTERATION

In any country, the food industry is a major industry with a very large turnover. In India, except among the very rich, the money spent on food accounts for a large portion of the family budget. In low income group families, money spent on food may be as high as 80% of the income and about 55% and 30% in the middle income group and high income respectively. Food supplies are dwindling in relation to population in the case of pulses, oils, milk, meat and eggs. At the same, the demand for foods other than foodgrain is increasing with industrialisation. Due to export possibilities, as in the case of spices, tea and Coffee, home market is at time strained. Thus in case of both foods, which are in short supply and foods which are abundantly produced, there is a real or artificial gap between supply and demand. When that happens, there is a great temptation for the shopkeepers for either adulterating the food sold or selling food of inferior quality.

What is adulteration?

The word "adulterated" implies an element of deceit. It means according to the definition in chamber's Twentieth century Dictionary "to debase, falsify by mixing with something inferior or spurious.

Any food article is considered to be adulterated if its nature and quality are not upto the, standards, which it professes to have, implicity of explicitly. Adulteration may be intentional or incidental, former is a wilful act on the part of the adulterator, intended to increase the margin of profit, food contamination is usually due to ignorance, negligence or lack of proper facilities.'

A third category is the contamination of food with harmful microorganisms during production, and handling.

When is food deemed to be adulterated?

In order to protect the health of the consumer, the Govt. of India promulgated the Prevention of Food Adulteration Act (P.F.A. Act) in 1954. The act prohibits the manufacture, sale and distribution of not only adulterated foods but also foods contaminated with toxicants and misbranded foods. A central food laboratory established under the act is located at Calcutta, for the purpose of reporting on suspected products. More recently, the central Food Technological Research Institute, Mysore has been recognised as another laboratory for the testing of adulterated foods for the Southern Regions. "A Central Committee for Food Standards" has been constituted under the act for the appointment of Food Inspectors by the state governments and their powers have been defined. The state government will set up food laboratory and will appoint Public Analyst with adequate staff to report on suspected foods.

In this unless otherwise requires:

(i) "adulterant" means any material which is or could be employed for the purpose of adulteration.

(ii) "adulterated"—an article of food shall be deemed to be adulterated-

  (a) If the article sold by the vendor is not of the nature, substance or quality demanded by the purchaser and is to his prejudice, or is not of the nature, substance or quality which it purports or is represented to be;

(b) if the article contains any other substance which affects, or if the the article processed so as to affect, injuriously the nature, substance or quality thereof;

(c) if any inferior or cheaper substance has been substituted wholly or in part for article, so as to affect, injuriously the nature, substance or quality thereof;

(d) if any constituent of the article has been wholly or in part abstracted, so as to affect injuriously the nature, substance or quality thereof;

(e) if the article had been prepared, packed or kept under instanitary conditions, whereby it has become contaminated or injurious to health;

(f) if the article consists wholly or in part of any filthy, putrid, rotten, decomposed or diseased animal or vegetable substance or is insect-infested or is otherwise unfit for human consumption;

(g) if the article is obtained from a diseased animal.

(h) if the article contains any poisonuous or other ingredient which redcrs it injurious to health;

(i) If the container of the article is composed, whether wholly or in part, of any poisonous or deleterious substance, which renders it contents injurious to health;

(j) if any coloring matter other than the prescribed in respect thereof is present in the article, or if the amounts of the prescribed coloring matter which is present in the article are not within the prescribed limits of variability;

(k) if the article contains any prohibited preservative or permitted preservatives in excess of the prescribed limits;

(1) if the quality or purity of the article falls below the prescribed standard or its constituents are present in quantities not within the prescribed limits or variability, which renders it injurious to health;

(m) if the quality or purity of the article falls below the prescribed standards or its constituents are present in quantities not within the prescribed limits of variability, but, which does not render it injurious to health;

Provided that, where the quality or purity of the article, being primary food, has fallen below the prescribed standards or its constituents are present in quantities not within the prescribed limits of variability, in either case, solely due to natural causes and beyond the control of human agency, then, such article shall not be deemed to be adulterated within the meaning of this sub clause.

This act came into force of 1st June, 1955 and the rules have been amended in 1968, 1973 and 1976. The standard which prescribed the minimum requirements for all types and categories of foods—are amended when required by the Central Committee of Food Standards (CCFS).

Common Adulterants in Food Material

The prevention of food adulteration act makes it obligatory for merchants and caterers to distribute foods coming up to specified standards. The term adulteration includes foods of inferior quality (e.g. grains), food contaminated with other substances (e.g. ghee), as well as those from which valuable factors have been removed (e.g. milk). It is also obligatory to make specified addition to certain foods (for example, stabilizers for ice creams and til oil for hydrogenated oil), and to specify the addition in the case of others (e.g. coloring matter, sweetening agents etc.)

Despite the Act, the adulterated food continue to be sold on a large scale. Housewives and others, concerned with the purchase of foods must be aware of the qualities expected in different foods and the ways by which the same can be ascertained.

(i) **Cereals and pulses** : These being seasonal corps are usually for 6-12 months. During this period degenerative changes may take place affecting their nutritional value because of their liability to be subjected to insect infestation. The changes occuring in them during this process can be summarised as follows:-

1. Loss in weight with decrease in the ratio of weight volume. This happens with insect infestation.
2. As uric acid is the main end product of protein metabolism in insects, contamination with uric acid increase proportionately with the storage period and insect infestation.
3. Decrease in organoleptic acceptability.
4. Increase in hydrolytic rancidity due to the deterioration of fat in the grain.
5. Deterioration in the quality of gluten in wheat and its products.

In most cases, the inferior quality of grains is apparent by visual inspection and tasting.

The permitted limit for *uric acid is* 20 mg. per cent, but market samples often contain more than this. Uric acid content was found to be as high as 40 mg. per 100 g. In some wheat samples, 64 mg. per 100 g. in maida samples, 50 mg. per 100 g. in bengalgram flour samples and 69 mg. per 100 g. in backgram dal, samples of balahar used in school lunch programmes have been found to contain as much as 180 mg. per cent.

The addition of *exrantous matter* like grains stone, marble chips and other filth to grain is also widely practised, specially in times of scarcity. These can be seperated by visual inspection from a known weight of grain and weighed. The permitted limit is less than 4%. Samples of bajra containing as much as 20-25% of extraneous matter have been found.

fungal and pesticide contamination of grains is also becoming common. Pesticides are sprayed in high doses and their residues are toxic to man. Sometimes grain is tended for seed and, therefore, heavily sprayed with pesticide is sold for human consumption resulting in death and disability.

Wheat flour, suji, and maida are expected to have a *gluten content* of at least 7%. As wheat contains 10-12% gluten, the permitted limit gives scope for adulteration.

Suzi is sometimes contaminated with iron fillings which can be detected by combing with a magnet.

Kesari dal (Lathyrus) is mixed witivoiher pulses. This is a staple food of the low income group in manys parts of Madhya Pradesh, Uttar Pradesh, Bihar and Bengal. This plant grows with very little effort and yields abundant crops, even under adverse conditions.

Common pulses adulterated with lathyrus are:

1. The whole pulse like black masoor, black bengal gram with whole kesari dal,
2. Split pulses such as arhar and chana dal with kesari dal.
3. It is mixed with besan, hence in all preparations made out of besan.

Intake of more than 30% of the calories may result in a disease called Lathyrism, P.F.A. has banned the use of Kesari dal in all form.

(ii) **Milk :** It *is* subjected to more adulteration than any other commodity. The readiness with which water can be added and fat removed, plus the fact that the demand is greater than supply, encourage adulteration.

The composition of milk is dependent on a number of factors viz. the breed, the season, the time of the day, the individuality of the cow, age and feed of the cow, estral period, the part of udder from which milking is done and the period of lactation. Still some reasonable minimum standards can be expected and have been set.

The adulteration of milk consists of either addition of *extra* water or the *removale* fat or both. Sometimes extraneous substances like *groundnut* milk are added. The selling of diluted buffalo milk as cow milk after coloring it, is a common practice (Cow's milk has a slight yellowish tinge not found in buffalos milk).

The constituents of milk bear a constant relationship with each other and this has led to the formulation of standards such as the percentage of dry solids, protein-fat ratio and lactose - protein ratio, which help in detecting abnormalities in milk sample resulting respectively from addition of water, removal of fat or addition of extraneous matter like groundnut milk.

Milk has a specific gravity of 1.029 to 1.035, which can be measured by lactometer by the housewife also. Milk adulterated with water when set as curd, can also be used as a criterion. Milk adulterated with other substances become yellowish and frothy and does not form good curd and it forms sour taste.

Khoa may contain maida, which can be detected by testing for the presence of starch.

The following standards have been prescribed by law for milk and milk products.

Milk Product	Standards Specified
Skimmed Milk	At least 8.5% non-fat milk solids.
Toned Milk	8.5% non-fat solids and 3% fat.
Butter	Milk fat not less than 80%.
Condensed skimmed Milk	At least 20% milk solids.
Chana	At least 15% milk fat.
Cream	Not less than 23% milk fat.
Ice-Cream	Not less than 36% by weight of milk solids.
Khoya	Not more than 10% moisture and not less than 20% fat.

(iii) **Salt, Sugar and Jaggery:** These may contain *extraneous matter* (insoluble matter) like dirt, marble chip, chalk, dust, dirt etc. The same can be measured by dissolving the substance in water and filtering the solution. The residue can be dried and weighed. By law, such insoluble matter should be less than 1% in the case of salt 2% in the case of jaggery and nil in the case of sugar. Sometimes salt and Sugar absorb *moisture* and become heavy. According to law, the moisture content of salt and sugar should not exceed 6.0 and 1.5% respectively. The same can be determined by weighing the sample before and after drying. *Metanil yellow* colour is added to jaggery to give it a bright appearance. This is a non-permitted colour due to its toxic effects on man, *Washing Powder* may be added as an adulterant to bura.

(iv) **Spices:** Turneric and chillies are colored with *lead pigments* in order to give them a bright color and the appearance of a product of good quality. Small quantities of lead

are deposited in the skeleton and the progressive accumulation of the same over the years may result in their release into the blood stream and consequently serious liver damage etc.

Metanil yellow which is a carcinogenic agent is also used for coloring turmeric powder. It is sold as Kesar color and used for the coloring of sweets and beverages, very widely and openly, although it is prohibited by law.

Hing (as afoetida) is adultered with *foreign resins.* Pure hing dissolves in water to form a milky white liquid and burns with a bright flame on being ignited.

The *essential oils are extracted* from cloves and cardamons, leaving behind a product without its full fragrance. Cinamon powder, which is cheaper, is passed off as nutmeg.

Mustard seed is often adulterated with *sand* which may be as much as 10% or more in some sample. Mustard seeds are adulterated with a toxic seed i.e. argemone seed. *Argemone seed is* similar to mustard seed, except that it has a tail on one end. These can be identified under a magnifying glass.

Crops of *clay, grit, chaf, straw* are found in coriander (whole) and cumins.

Garam masala and other masala's contain *more salt* than prescribed. Salt being cheaper than other expensive spices like black pepper, cinamon etc. leaves a margin for profit. Salt is not harmful, but the quality of these masalas go down.

(v) **Coffee, Tea and Cocoa:** The quality of tea is affected by malpractices such as addition of *sawdust, exhausted (leaves, foreign* etc. Microscopic example of tea sample discloses a great deal about its purity. Other estimates which should be made, when a thorough checkup is desired are moisture, total soluble and insoluble ash and nitrogen. Exhausted tea leaves when used, are colored to make the appearance acceptable. The coloring matter used, may be harmful for health.

In case of coffee, the raw seeds do not lend themselves to adulteration, except by way of mixing inferior seeds with superior ones. But, when roasted and ground, they are liable to adulteration of various kinds. The most common is the roasted seeds of *dates and tamarind.* Other powder which may be added, is *chicory powder.* The important constituents of coffee are caffeine, sugar, cellulose, protein and small quantities of fat and essential oils.

The ash content of the powder is used as an index of purity. The ash content of coffee is less than 4-6%, of which about half is water soluble, while adulterants like chicory contain a higher amount of insoluble ash.

In the case of cocoa, there is a temptation for the manufacturers to use the finely ground sell as an adulterant, sago flour or starch coloured with red oxide or iron is also used. Microscopic examination is useful in selecting the presence of shell.

(vi) **Eggs:** The shells of fresh egg have a glazed appearance. The air space between the contents and top of the shell is not more that 1/4" wide. The eggs should feel heavy for its size. When opened, the white should be clear and yolk, yellow and firm, without the signs of embryo or a blood spot. Eggs must be discarded, if increase in air space, unpleasant colour, discoloration of yolk, watery white mould infestation on the shell and blood stains are evident. Fresh egg sinks in water while spoilt egg floats. This test of efficiency is improved by using a salt solution containing 2 ozs of salt per pint of water.

(vii) **Meat:** The quality of meat is determined by its tenderness, juiciness and palatibility. It is almost a universal practice to hang the carcass overnight after slaughter of the animals. Thus meat sold to the public is generally hung for 15-24 hours at room temperature. The carcasses become rigid a few *hours* after the slaughter of the animal. This change is a accompanied by a drop in pH from 6.5 to 5.7 and again increases to 6.1 when stored for 20 hours.

Inspection of the meat is necessary to ensure that the meat is from a healthy animal in a sound condition at the time of slaughter.

According to the report of the committee appointed by FAO/WHO in 1955, the following steps should be take to ensure a safe meat supply:

1. Examination of animal before slaughter.
2. Examination of carcass and parts of carcass immediately after slaughter.
3. Removal of all unfit and diseased material.
4. Adoption of environmental conditions to prevent the contamination of edible parts.
5. Hygiene of meat handler.
6. Transport and distribution of meat under proper conditions.

Meat from diseased animal or if unfit due to hygienic conditions of improper handling by the handler, is designated adulterated.

(vii) **Vegetables and Fruits:** The quality of vegetable varies considerably and although some variation such as those in tenderness, color and moisture content are of no serious concern from the stand point view of hygiene, their working quality and acceptibility are affected. Vegetables are prone to mould and worm infestation. Vegetables and fruits often contain the eggs of intestinal parasites, if they have been exposed to contaminated soil and must be thoroughly scrubbed and cleaned before consuming.

(ix) **Other Foods:** It is particularly important to ensure the purity of prepared foods sold in restaurants and sweet meat shops and rcady-to-eat foods such as candy, dates, raisins etc. Sweet-meats may contain *non permitted colours* like metanil yellow in laddoos and jalebis. Non-permitted colours are used by ice-cream, ice-ball vendors. The syrup used for decorating the ice-balls may contain saccharine. Many a times Khoa preparation like burfi, gulab-jamum contains *excess of maida.* The *aluminium sheets* are used to decorate the sweet-meats in place of silver foil, which may result in poisoning from aluminium. Food poisoning often results because of the *Bacterial contamination,* particularly sweets prepared from milk; *copper poisoning* may result from the use of insufficiency tinned copper brass vessels. Milk and prepared foods should be kept in containers made of galvanised iron. Various preparation like pani-puri, bhel-puri, chaat etc. may contain *harmful Bacteria,* resulting in gastro-intestinal infections. A number of snacks bought by school children may be adulterated. Example of some of them are given in Table I.

Table I

Possible Adulterants in some school snacks

Name of the snack	**Adulterants**
1. Am papad	dust, insect exereta, harmful becteria
2. Ice candy	(a) Metanil yellow

3. Ice candy	(b) Rhodamin 'B'
4. Karachi Halwa	(c) Orange II
5. Rasgulla	(d) Blue VRs
6. Colored Sweets	(e) Auramine
7. Sugar coated saunf	(f) Malachite green
8. Churan	Saw-dust, red-poisonous color, excess lead
9. Supari	Orange B, Auramine, Dust, Other nuts, fragments of insects
10. Toffee, Biscuits	Talc or soap stone, asbestos
11. Groundnuts	Fungus & Fungal Toxins

(x) **Packed Foods:** Many hazards may result from packaging material used for packaging the foods. Polythylene, polyvinyl chloride and altered compounds are used to produce flexible packaging material. While this method of packaging is very convenient, it may contain noxious thermal breakdown products, which could be injurious to health. Further, temperature used for heat sealing, or sterlisation may result in the formation of toxic residues. It has been observed sometimes that in foods like pickles, the acid and oil could attack the plastic packaging material and creates a health hazard.

Tin is used for packaging various materials like oils, fats, beverages, biscuits and all types of processed foods stuffs, which is normally well tolerated. Yet there are many cases of tin poisoning due to ingestion of contaminated fruit juices. Tin poisoning may result (a) when the canned food is acidic; (b) when the food contains nitrates, oxalates copper or certain sulphur compounds; (c) when moist food is left in the can after opening.

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Lesson 14

ILL EFFECTS AND DETECTION OF SOME COMMON ADULTERANTS

Adulteration of food articles is rampant in the country and has to become a grave menace lo the health and well being of a community. It makes a heavy dent in the already low nutritional standards and the benefits of many public health programmes, on which large sums of money are spent. The consumer is not a producer himself and has to depend upon the market. He is blissfully ignorant of the insidious hazards of consuming adulterated foods, which may be adulterated at all stages, from production to retail. Foods of excellent nutritive value to begin with, may become unfit for human consumption during storage or handling, Insect infested grains, rancid fats and contaminated milk and sweets are example. An average consumer is mostly guided by the price, visual appearance and claims of the advertisement. He has still to grasp the idea of a good quality in his mind.

Ill Efects of Adulteration

Main ill effects of adulteration may be classified into three main categories.

(a) Consumer is penalised by paying for the adulterant along with the food.

(b) Consumer is getting less for his money's worth i.e. the nutritional content of the adulterated food may be less than the pure food, as in case of milk adulterated with water.

(c) Lastly, but not the least, are the numerous health hazards that can adversely affect the consumer. These are discussed in detail below.

Health hazards: Numerous health hazards may occur due to adulteration, which could be intentional or incidental. Intentional adulterants are added to increase the margin of profit. These could also be due to contaminations with bacteria or fungi during storing and handling.

(a) Health Hazards due to Intentional Adulterants:

(i) *Sand, Stones, earth, marble chips and otfur fifth* may be found in food grain, pulses, spices etc. Stones and sand if present could have an adverse effect on the teeth and soft linning of the digestive tract. Filth in any form is a health hazard, as it may contain millions of disease producing micro-organisms.

(ii) Talc and Chalk power are usually added to wheat flour, powdered spieces which are white and to other such foods. Being undigestible by the human system, these can effect the normal digestion.

(iii) Water and excess moisture are added to milk curd, paneer, butter and other products. It is not a health hazard unless the water is contaminated, and if so, it may result in gastro-intestinal disturbances.

(iv) Asbestos is often used in filtering fruit juice and soft drinks. It is a natural fibrous silicate. It has been found that these fibres which are very fine and can only be seen through an electron microscope, are present in clear juices. These are known to cause gastro-intestinal cancer.

(v) Sugar and Sweeteners: If sugar is added in pure form to honey, then no health hazard is involved. Toxic sweeteners like cyclonates, dulcine or saccharine may be present, where not permitted as in sweetmeats or in excess of the permissible amounts. Fruits are injected with saccharine to make them more sweet in taste.

Additives are colorings, preservatives, flavourings, anti-toxicants, emulsifying and stabilizing agents, anti-microbial agents. The amount of these additives, if exceeds the minimum amount permissible, may have harmful effects, varying from minor gastro-ailment to cancer.

(vi) *Mineral oil* mixed with edible oil, when taken in sufficient amount may cause gastro-intestinol disturbances and vomiting. These are petroleum derivates and much cheaper than edible oils. These have been known to adversely effect the absorption of vitamin-AO.

(vii) *Rancid Oil* is sometimes mixed with edible oil for increasing the margin of profit. Food stuffs cooked in this oil may loose its vitamin content.

(viii) *Argemone Seeds:* These seeds look very much like mustard seeds and mixed with mustard seeds as an adulterant. Argemone seed is different from mustard seed by possessing a little tail at one end. When oil is extracted from adulterated mustard seeds, it is highly toxic because consumption of argemone oil can lead to loss of eyes sight, heart disease, epidemic dropsy resembling wet beri-beri.

In 1971 in kagaznagar (West Bengal), hundreds of families including young children, became victims of epidemic dropsy due to adulteration by argemone oil. Even in 1973, these was a report from Hari Nagar, New Delhi. This made the Union health Ministry issue a warning to public at that time, against buying mustard oil from unreliable sources.

(ix) Kesari Dal (Lathyrus) kesari dal would be mixed as an adulterant with the other dals.

(a) The whole pulses like black masoor, black bengal gram with whole Kesari dal.

(b) Split pulses such as arhar and channa dal with kesari dal.

(c) It is mixed in besan, hence all preparations of besan.

When more than 30 per cent of the total calories consumed is contributed by this dal, it can cause lathyrism, a from of crippling paralysis of both lower limbs, mainly in boys and men in the age group of 5 to 45 years. If a diet consists of 40% or more of lathyrus, then this disease manifests itself in 2-4 months. The disease starts with stiffness of the knee joints and legs with pain around the knee and ankle joints. Within 10 to 30 days of the onset of the symptoms, paralysis of the lower limbs set in. The patient gradually becomes crippled as his knees are bent and stiff.

(x) foreign seeds, barks, leaves or other foreign matter:

(a) Foreign seeds may be colored to look like genuine food material. These are added to increase the bulk, as most of these have no value as condiments. As such they may not be harmful, but addition of toxic colors to it could be harmful. Cumin seeds, cardamons, poppy seeds, black pepper etc. are adulterated with these foreign matter, e.g. the seeds of wild grass which resemble cumin seeds are added to it. Fenugreek and aniseed may similarly be adulterated with excess of other edible seeds. Black pepper may contain dry papaya seeds, cardamom may contain damaged seeds or substitutes.

(b) Roasted tarmarind and date seeds are powdered and mixed with coffee or cocoa.

(c) Chicory seeds are powdered and mixed with coffee powder.

(d) Thick bask of cassia is added to cinamon as an adulterant.

(e) Foreign leaves or exhausted tea leaves are colored. These and saw dust have been mixed with fresh tea leaves.

(f) Exhausted bark or seeds of coffee or spices are suitably dried and colored and substituted for fresh ones. In such cases the sample will be deficient in its volatile oil content.

(g) Powdered bran and sawdust are often present in atta, suji and ground spices.

(h) Strips of jute, paper or foreign material may be suitably dyed to pass off as saffron.

(i) Resin or gum is generally used as an adulterant in asafoetida.

(j) Cinamon powder which is cheaper, may be passed of as nutmeg powder.

(k) Refind starch or mashed potato being cheap are used in milk and milk products like khoya, burfi, ice-cream, rasgulla, butter, ghee, cheese. Rice starch or starch from tapioca are added to arraroot, turmeric powder, coriander and mixed spice.

(xi) **Toxic colorings:** Color is added to increase the variety in food and also its acceptibility. Color is widely used in preperations of ice-creams, dairy products, biscuits, confectionaires, pastries, fruit products, processed and preserved vegetable, jelly, custard powder, soup powder, toffee, sweets etc. Many of the colors are not permitted, due to its harmful effects and addition of these colors is adulteration. The common non-permitted colours used are mineral pigments like lead chromate, red or yellow earth, color dyes like metanil yellow, Rhodamine B, orange I and II. Most popular is the water soluble metanil yellow. It is widely used to color pulses and spices, sweet-meats, (jelabies, ladoos, karachi halwa) and aerated waters. These colorings are carcinogenic in effect. Intake of these colors produces various abnormalities of eyes, bone, spin, lungs, ovaries, testes etc. The non-permitted coal dyes are generally found as adulterants in the followings foods :

Metanil yellow	:	Ice Candy, faluda, ice balls, pulses.
Orange II	:	Karachi Halwa
Rhodamine B	:	Red Chilly powder and churan
Blue VRS	:	colored sweets
Auramine	:	sugar coated saunf and supari
Malachite green	:	Coconut

(b) Health hazards due to Metal Contaminants:

Most metals are toxic when present beyond a specific concentration. Its poisonous effect is neutralised in the beginning, when they combine with proteins. With the increase in concentration, symptoms such as vomiting, nausea, stomachache may arise.

(i) **Arsenic:** It is present in small amounts in air, water and oil and slowly finds its way into food. Arsenic pesticides are the main source of arsenic contamination of foods. For example fruits such as apple and grapes sprayed with lead arsenate, if eaten without washing, could be harmful. The quantity of arsenic allowed in food products various from 0.1 p.p.m. (in milk) to 5.0 p.p.m. (in pectins, spices) depending upon the food. When arsenic concentration is higher than prescribed, it can cause dizziness, chills, cramps and paralysis.

(ii) **Lead:** Human system absorbs lead not only from foods, but also from water and dust in the air. It is absorbed more easily from liquid foods than solid. Lead is present is varying amounts in food stuffs such as shellfish, herbs, edible gelatin, pectin, food colorings, tea, baking powder, custard and peaches. In addition to this, food may be contaminated by.

* Exposure to leaded dust in air.

* Contant with Equipment packing i.e. tin plate containing lead.

* Lead pipe through which drinking water is obtained.

* Use of turmeric powder with lead chromate.

* Spraying and dusting of fruits and vegetables with lead containing pesticide.

* Use of synthetic dyes.

The quantity of lead allowed in food products varies from 0.5 p.p.m. (in concentrated soft drinks) to 10 p.p.m (in liquid pectin and chemicals not otherwise specified), depending on the food product. If build up of lead in blood increase beyond the critical blood level of 40 to 80 mg. per 100 ml of blood, mental disturbances and behaviour disorders may be produced followed by paralysis, convulsions and permanent brain damage. Milk has been recommended as a good antidote for lead prisoning.

(iii) **Mercury:** It is present is traces in the form of its compounds in all water and food. The effluents from many industries now a days have high concentrations of mercury and human beings and animals consuming crops grown with such water or fish from such areas could develop mercury poisoning. Intakes of about 0.0033 mg/kg. of mercury and methylmercury per person are harmful. Such intakes of mercury could affect the brain with the patient becoming blind or deaf. Convulsions with intense plain is also one of the symptoms of mercury poisoning.

(iv) **Tin:** Various kinds of food come in close contact with tin in the process of preperation and storage. Beverages and canned food stuffs normally contain very small amount of tin which are well tolerated, yet, there are many cases of tin poisoning due to ingestion of contaninated fruit juices. A variety of containers made of this plate are also used for storing oils, fats, beverages, biscuits and all types of proceed foods. Tin poisoning may result.

* When the canned food is acidic.

* When the food contains nitrates, oxalates, copper or certain sulphur compounds.

* When moist food is left in the can after opening.

The maximum tin content allowed in any canned food stuff is 250 p.p.m. High tin content in foods (about two to three grams) may cause severe headache, vomiting, vertigo, photophobia, abdominal pain, dehydration and retention of urine.

(v) **Copper:** It is normally present in food stuffs in traces, but contaminated food causes diarrhoea, abdominal pain and vomiting. Acute poisoning by copper salts brings constriction in throat and an astringent taste with thirst within 5-10 minutes.

(vi) **Aluminium:** Sweetmeats were generally topped with their silver foil. Now-a-days very often this aluminium foil is used. This can cause undesirable reactions in the digestive tract, interfering with the normal functioning.

(vii) **Cadmium:** It is a silvery soft, white metal, used for plating utensils and electric cookers. It is resistant to alkalies, but dissolves in the acid present in fruit juices, wines, soft drinks. The salts of cadmium thus formed, dissolve in food and can be harmful to health, 15-30 minutes after ingestion of contaminated food.

(c) **Health hazards due to Pests, and Pesticides:**

Pests such as rodents and insects once get into food, can cause heavy damage due a high degree of filth in the form of excreta, bodily secretions, insects fragments and hairs as well as disease bearing and spoilage micro-organisms.

(i) **Rodents:** They usually include rats and mice. A single rat voids 10,000 dropings and 4 litres of urine annually, besides constantly shedding some of its coat made up of 5 lakh hairs. The kidneys of these rodents have an organism, which causes disease of liver (spirochaectal jaundice). This is usually transmitted to man through food or water mixed with rat urine. Some bacterias are found in rat urine, which resist ordinary cooking temperatures. Rat alone may cause, as many as 35 diseases. Their rate of reproduction is very high, which creates a major problem.

(ii) **Insects**: Insects such as beetles, weevils is, bores, enter in foods like cereals, pulses and their flours, when kept in warm, humid climate. They practically eat up the whole grain or food and in this process uric acid and a offensive odour and taste develop in the food.

(iii) **Pesticides**: At times, food is directly mixed with the pesticides, although it is against law. Organic pesticides such as DDT/BHC, Malathion, Pyrethrum are used, but these leave behind a residue (the unchanged chemical or its derivative) on the food. These are toxic depending on the nature of the pesticide. The maximum concentration of pesticide residue, that is permitted in or on the food is laid down by PFA. The maximum permissible residues allowed for DOT, and malathion is 3 p.p.m. For pyrethram it is 10 p.p.m. Many time, pesticides residue could be much higher than this figure and could the be toxic. Acute poisoning by DDT and BHC are rare, but continued consumption of these may lead to chronic poisoning.

(d) **Health Hazards due to Packaging**

The most common packaging material in the food industry is PVC (polyvinylchloride) and PE (Polyethylene or plastic), to produce flexible packaging material. While this method of packaging is very convenient, it must not contain any noxious thermal breakdown products, which could be injurious to health. Furthermore temperature used for heat sealing, or sterlisation should not result in formation of toxic residues. It has been observed that in foods like pickles, the acid and oil could attack the plastic packaging material and create a health hazards. To avoid such incidents, it is essential that only food grade plastic packaging material be use for packaging foods.

(e) **Health Hazards due to Bacterial and Fungal Contamination**

Improper and unhygenic methods of storage, handling, and preparation may result in contamination of food material with numerous bacterias and fungi. This has a direct and immediate impact on the public health. The foods that are commonly contaminated are milk and milk products, meat and meat products, poultry, eggs, fish, raw vegetables, cereal-based products. Food serves as a vehicle to transport these micro-organism to man. In the host, they multiply and during the process of growth, some microbes produce toxic metabolities, which may remain in the food even after organism dies. The symptoms of pathogenic bacterial contamination may be nervous disturbances or gastro-intestinal disorders.

Simple tests for detection of food adulterants

The adulterants used are so similar to natural food-stuffs that it becomes very difficult for a common man to detect them. A few simple tests can be conducted to detect the presence of adulterants.

(a) **Cereals and Pulses**

(i) **Stone chips in Rice:** Place the rice grains on the palm of the hand and gradually immerse the hand in water. The stone chips will sink.

(ii) **Inorganic and Organic Matter:** Inorganic matter consists of matter like sand, gravel, dirt, pebbles, stones, lumps of earth, clay or mud. Organic matter could be chaff, straw, edible seeds. These may be used as an adulterant in rice, sugar, pulses, wheat, jowar, mustard, edible seeds etc. Such matter could be observed and removed physically. Presence of these substances is allowed to the extent of two to five percent, depending on the food product. Beyond this limit, food is considered adulterated.

(iii) **Infested Grain:** The grain may become infested by pests and rodents during storage. These can be observed and removed physically. Presence of infested grain is allowed upto one percent, beyond which the food produced is considered adulterated.

(iv) **Kesari Dal (Lothyrus) in Arhar, Masoor and Ghana Dais**: Kesari dal has a characteristic wedge shape. There are two varieties. One is small and resemble masoor dal. The larger is the size of arhar. The husked kesari dal is mixed with arhar and chana dals. The unhusked one is mixed with black masoor or bengal gram. Kesari dal can be separated by visual examination.

It can be tested chemically also. Add 5 ml. of normal hydrochloric acid to a small quantity of dal in a glass. Keep the glass in simmering water for 15 minutes. Development of pink colour indicates the presence of kesari dal.

(v) **Ergot in Bajra:** Ergot seeds are lighter than bajra seeds. If a small quantity of grains are put in 20% salt solution of water in a glass, ergot seeds will float to the surface, while bajra will settle down.

(vi) **Argemone seeds with Mustard seeds:** Argcmonc seeds are small and black in colour, resembling mustard but not uniformly smooth and round. They can be identified under a magnifying glass.

(vii) **Coal tar dye in Roasted Gram:** This would be detected by visual observation.

(vii) **Iron fillings in Suzi/Semolina:** By drawing a magnet over the sample, iron fillings will cling to the magnet, thus revealing their presence.

(b) **Milk and Milk Products**

(i) **Water in Milk:** The specific gravity of milk lies between 1.029 to 1.035. With the increase in fat content, the specific gravity decreases and conversely as the non-fat solids increase, the specific gravity increases. However, water added may not be detected by specific gravity, if water is added and the fat is also removed.

(ii) **Starches in Milk:** Addition of a drop of iodine solution, to starch containg milk, will turn it blue. Pure milk turn a coffee shade.

(c) **Fats and Oils**

(i) **Vanaspati in Pure Ghee:** One teaspoon of pure ghee or butter is mixed with equal quantity of concentrated hydrochloric acid in a test tube. To this 2 to 3 drops of furfurol solution is added and the test tube is shakcd well and allowed to stand for five minutes. Appearance of pink colour in the lower layer of acid means that vanaspati is present in pure ghee/butter as an adulterant.

(ii) **Mashed potatoes, sweet potatoes and other starches in Butter:** Iodine test is performed i.e. addition of a drop of iodine solution turns the butter blue.

(iii) **Cheap edible oils in vanaspati:** A solution of washing soda is added to the sample of vanaspati and shaked well. Appearance of a froth on top is taken as an inference that cheap oil has been added to vanaspati.

(iv) **Argemone Oil in Mustard Oil:** 5 ml. of test sample is heated with 5 ml. of nitric acid for two or three minutes. Appearance of red colour is an indicator of the presence of argemone oil.

(v) **Rancidity in Oils:** To 5 ml. of sample in a test tube, 5 ml. of hydrochloric acid is added. Stopper the test tube and shake vigorously for thirty seconds. Add 5 ml. of 0.1 per cent phloroglucinol solution in either. Shake for thirty seconds and allow to stand for thirty minutes. A pink or red colour in the acid layer indicates that the oil sample is rancid.

(d) Sugar and Sugar Products

(i) **Chalk or any other dust or dirt in Sugar:** Dissolve a little of the sample is hot water, Sugar will dissolve while the impurities will not.

(ii) **Jaggery with mctanil yellow:** Hydrochloric acid is added to the solutions of jaggery. Appearance of magenta r«l colour is an indicator of the presence of adulteration with the meianil yellow colour.

(iii) **Bura (Sugar powder) with Washing Soda:** With hydrochloric acid, the audlterated sample give effervescence. If dissolved in water the washing soda will turn red litnus into blue.

(e) **Spices and Condiments**

(i) **Metanil yellow colouring in Haldi, Haldi Powder:** To 2 g. of sample, add 5 ml. of alcohol. Shake and add a few drops of concentrated hydrochloric acid. A pink coloration indicates the presence of metanil yellow.

(ii) **Artificial colour in Chillies:** (i) Rub Ihc outside of a chilli with a cotton soaked in liquid paraffin. If the cotton extracts the colour and becomes red, it is an indication that the sample has added colour, (ii) Mix a small quantity or red chilly powder in ether. Take the extract and add conccnteated hydrochloric acid, A dark pink colour indicates that ihe sample has added colour.

(iii) **Grit, Clay, extraneous matter in common Salt:** Dissolve the sample in some water in a tumbler. Pure salt will completely dissolve in it, while extraneous matter will be seen in the solution or it will settle down.

(iv) **Grit in Spices:** Spices can be dissolved in carbon tetrachloride. In water, the sprinking of small quantity of the samples will result in floating of the spices while grit will settle down. A tumbler should be used for testing.

(v) **Resin or Gum in asafoetida:** Pure asafoetida dissolves in water to form a milky solution. Pure asafoetida burns with a bright flame on ignition. Any deviation will be the indicator of adulteration.

(f) **Beverages**

(i) **Artificial colourings in tea-leaves:** Deposit the tea leaves on a moistened blotting paper. Artificially dyed tea leaves will impart colour to the moistoned blotting paper immediately.

(ii) **Powdered date seeds or tarmarind Powder in Coffee:** Spinkle a little coffee powder on a piece of blotting paper and spread a few drops of potassium hydroxide solution

over this paper. If a brown colour emerges around the particles of coffee, adulteration is established.

(iii) **Starch in Coffee:** Make a decoction of the coffee. Decolorize it by adding potassium permagnate soludons. Then add a drop of iodine solutions. Blue colour formation indicates adulteration with starch.

(iv) **Chicory in Coffee Powder:** Sprinkle a small quantity of coffee powder on the surface of water in a glass tumbler. Particles of genuine coffee powder will float, but chicory powders will begin to sink within seconds. They will also leave a stain of colour in the water.

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Lesson 15

ACCIDENTAL CONTAMINATION

Food can be contaminated by accident also, through chemicals, the utensils we use or the packaging we use. It can also occur due to various microbes like bacteria or fungi. At times certain plants animals have natural poison which are harmful for the human body e.g. certain mushrooms, favabeans etc. Contamination of food accidently by any of the above mentioned sources result in food poisoning. This chapter deals with this type of food poisoning and the remedeal measures.

Food poisioning is defined as illness resulting from ingestion of food containing certain inorganic chemicals, poisions derived from animals and plants and toxic products produced by several species of bacteria and fungus. Essentially they are of three types as given in fig.1.

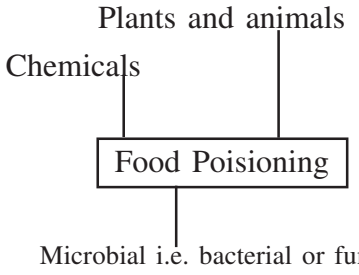

Fig. 1: Three types of food poisioning

Food poisioning is characterised by its very explosive nature of illness. The prominent symptom is gastro-intestinal upset, therefore nausea, diarrhoea, vomiting and abdominal cramping are seen, it is different from food infections like cholera and typhoid for two reasons. Firstly, the incubation period of infection is longer than food poisioning and secondly in food poisioning, the toxin is present in the food itself at the time of consumption and affects immediately; whereas in infection, food is only a carrier and the bacteria invades the body, grows and multiplies and leaves toxic products which damages the tissue, e.g. in typhoid there is inflamation and eruptions in intestine followed by high fever. Similarly, in cholera there may be discharge of mucous and blood in loose stools due to disruption in the integrity of the gut muscles. There is a possibility that micro-organisms produce the toxins and escape or are killed on their own or on cooking, but toxic residues are still present and are potent to cause food poisioning.

Let us now study, the three types of poisioning in detail. These are :

— Chemical poisioning

— Plant and animal poisioning

— Microbial poisioning (both bacterial and fungal)

1. **Chemical Poisioning**

Incubation period for this is very short. Incubation period, i.e. the time taken for the first symptom to appear after eating the unacceptable food is 10 min—2 hours.

Alcohol Poisoning—Almost every year it is heard that a group of people became blind because of unacceptable alcohol consumption due to methyl alcohol (CH_3OH) toxicity. The methyl alcohol toxicity appear when the preparation of alcohol (C_2H_5OH) is crude. Methyl alcohol toxicity appears within 18 to 48 hours after consumption. Methyl alcohol is also used to

denature alcohol i.e. make ethyl alcohol unfit for human consumption and prevent stealing of ethy alcohol. Its consumption leads to blindness, abdominal disorder and ultimately death.

Selenium (Se) toxicity : Small amount of selenium is required by the body, but excess leads to gastro intestinal disturbances, dyspepsia, anorexia, stunted growth, falling of hair. The toxicity occurs where soil is rich in selenium, therefore, plant and cattle are rich in selenium leading to exceeding the toxic level in man.

Antimony poisoning: It occurs from eating food which are looked in cheap, grey enamelled utensils or by consumptions of water rich in industrial wastes.

Cadmium poisoning (Cd): is associated with acidic liquids stored in cadmium plated metallic utensils or corrosion of water pipes, crops of cadmium rich soil, by industrial waste and environmental pollution. Also, by cigarette smoking one inhales 0.1 my cigrette. If a 70 kg man takes 25-60 meg/day toxicity can occur. It competes with zinc in body and affects growth and leads to stunting. It is a cumulative poison and is reabsorbed by renal tubules leading to renal failure. It is retained in large amounts in kidney and liver and can therefore also lead to liver damage (liver cirrhosis). Due to the effect on renal system, it can also cause hypertension and anaemia.

High calcium and high protein diet protects from cadmium toxicity. Further prevention of this toxicity requires lowering from environmental pollution and the industrial waste.

Nitrates, Nitramine, and Nitrosamine: Their toxicity leads to digestive problems, cyanosis in 2-3 days. Cyanosis in small children can lead to death. Nitrates can come from water, fertilisers, foods like spinach, cured meats. But fortunately, on cooking nitrates are converted to nitrites which is not harmful. Nitramine and nitrosamine are carcinogenic, but its occurrence has been reported only from South Africa where plantain and banana is eaten in plenty (40 mg/l00g banana). It leads to myocardial fibrosis.

Cancer is reported on use of nitrate and nitramines in cured meats.

Lead Poisoning (Pb): Lead salts are sprayed on fruits and unwashed fruits can accidentally cause lead poisoning. Some of it is received through water from lead pipes. Lead is a cumulative poison i.e. it takes some time to show signs and symptoms and accumulate in the body till it reaches toxic levels. Symptoms like anaemia, anorexia, gastroenterities are seen.

Barium carbonate ($BaCO_3$): is also called red poisoning and is accidental when it is used instead of taking soda accidentally.

Arsenic poisoning: Fruits sprayed by arsenic salts can cause toxicity, if consumed unwashed.

Flouride toxicity : Sodium flouride (NaF)—is used to kill cockroaches, and if mistaken-for salt or baking powder in the kitchen, it can lead to death. Flouride excess affects teeth and bones and can cause crippling. Excess flourides can be ingested from crops grown on flouride rich soil, toohpastes etc.

Zinc poisoning—Zinc is a trace element required in small amounts by the body, but its excess (which is rare) can cause zinc poisoning. This can occur when acidic fruits (or foods) are cooked in galvanised containers.

Mercury poisoning—It is very rare and occurs if industrial waste contaminates the water. The fish in river can get killed, and if it survives, it becomes a carrier of large amounts of mercury.

Symptoms of chemical poisoning

These are nausea, vomitting, general malaise (feeling of not being well), lassitude, abdominal cramping and in cases like sodium fluride poisoning can cause paralysis of certain muscles or death.

Safe chemicals—Aluminium, tin, stainles steel, brass (tinned only) chromium, glass are safe for cooking and storing purposes. Packaging material of aluminium foil or polyethylene terephthalate (PET) are recommended. Wax coated paper for bread and butter paper for biscuits are also used.

Prevention of chemical poisoning

Usually cure is very difficult and these usually lead to irreversible changes like paralysis or death. Precautions should be taken to prevent it—

(1) Safe utensils and storage containers should be used for handling food.

(2) Proper labelling of insecticides should be done and they should not be stored in kitchen.

(3) Children should be kept away from insecticides.

Effect of other Chemicals

Chemicals poisoning can also take place due to insecticides, fertilisers and its residues, contaminants, adulterants, preservatives and packaging materials. Artificial colors like coaltar dyes, some of which are permitted, but most especially in large can cause cancer, paralysis, indigestion and pathological lesions in vital organs like brain, kidney, liver, spleen. Colors like metanil yellow, lead chromate, rhodamirie, sudan red are popular but not permitted colors. They are used for dals, haldi, chillies etc. and can cause cancer, brain damage. Preventive measures are to give consumer education, buy from reliable shop etc.

Pesticide residue can also be toxic but pesticide benefits are more than harm; therefore their uses are only encouraged. NaFC (cochroach bait), BaCOS (rat baits) DDT, pyrethum, parathion, malathion etc. are usual toxicants. DDT, is very popular and is cumulative poison, therefore, even seen in breast milk. Malathion, parathion etc. (organophosphates) are neurotoxin i.e. affect nervous system causing anxiety, giddiness, insomnia (lack of sleep) paralysis, mental depression etc.

Food packaging should be very selective. Only materials which are recommended to hold food should be used. Aluminium foils, polyethylene terephthalate (PET) are recommended instead of polyvinyl chloride, other plastics, cardboard etc. Recycled dyed polyvinyl chloride decreases shelf life of food, discharges color in acidic or alkaline foods. Food and Drug Administration in USA, Japan, UK and West Germany recommended (PET bottles which are tightweight, unbreakable, and hygienic for packaging food products. Bread is packed in parafin wax coated paper. Cardboard caused poisoning through noodles stored in it in Japan. Special precautions are therefore required while tinning the foods, cardboard packaging and not using recycled colored PVC etc.

II. Plant and animal poisoning

Some plants and animals have natural poisons, which render them unfit for human consumption. It is for this reason that certain foodstuffs are excluded, like favabeans, snake root, shubarbs, certain mushrooms, gossypol, green potato, raw soyabean, goitrogens, kesari dal (Lathyras sativus), argemonc seeds etc. from plant kingdom and shell fish from animal kingdom.

Favabeans (Vicia faba) cause 'favism'. This is not prevalent in our country. Even smelling the flowers of this plant can cause poisoning leading to haemolysis of blood, jaundice, blood in urine haematuria.

Snake root—Usually animal feed on it and milk given by these animals affect us.

Rhubarb is a green leafy vegetable like spinach or lettuce and is rich in 'oxalic acid'. Usually its consumption leads to oxalic acid stones, but excess consumption leads to oxalic acid poisoning.

Mushrooms—Only a few type of mushrooms are edible and usually these are cultivated. The local Indian name is khumbi (button mushrooms), guchic (morels), dhingri. Some of the most beautiful varieties in color and size and extremely poisonous and can cause death like Amanita Muscarin (toxin is an alkaloid—muscarin). Muscarin causes excess salivation, blindness, anuria (no urine)vomitling, abdominal cramping, diarrhoea, dizziness, confusion, convulsion i.e. act as neurotoxin. Antidote is atropine and electrolytes and water is given to prevent dehydration.

Gossypol is yellow pigment in cotton seed cake used as protein supplement. ISI has now laid standards for cake and oil, so gossypol has to be extracted and removed. Gossypol binds with lysin and reduces protein quality.

Green potato contains solanine which can be harmful apart from giving an off taste. Salaniane is a glycosidal alkaloid, which is produced when potato is exposed to sun. 8 kg of such potatoes or 40-50 mg of solanine can lead to poisoning. Thus, its a rare feature. The symptoms appear in 8 hrs like abdominal pain, vomitting, diarrhoea. Solanine is heat stable but prevention is possible by discarding the keel and green area.

Soyabean (raw)—Soyabean has been largely advocated by nutritionists for its protein, fat and fibre content. Soya protein was textured to nutrinuggets or nutrela to give an appearances of a non-vegetarian dish and provide good amount of protein (42% by weight). Soya oil is also a good substitute for vegetable oils in the market and is rich in poly unsaturated fatty and, which is recommended for heart patient. Soya fibre prevents increase in blood cholestrol and blood sugar and is, therefore, good for heart patients and diabetes patients. But these soyabeen preparations are processed on cooked. Raw soyabean has trypsin inhibitor, which must be destroyed by heating (or cooking) so that the soya proteins are better digested. Otherwise, raw soya-diet can decrease the digestibility of proteins in the diet. Trypsin inhibitor can be destroyed on cooking, soaking and sprouting the beans.

Goiterogens: Raw cabbage has thiocyanates which can suppress thyroid gland activity, but cooked cabbage does not have this effect.

Kesari dal (Lathyris Sativus): Increased consumption of this dal can lead to lathyrism which is characterised by paralysis of limbs, the toxic principle is BOAA i.e. B oxalyl amino alanine. BOAA is water soluble, heat labile to some extent and varies from one variety to another of kesari dal (12-20% of total proteins can have BOAA).

Mode of action—It can cross the brain barrier and circulate in the blood supply of brain. It is a known neurotoxin. In animal studies the toxin had produced damage to heart, liver etc because it is a deep penetrating toxicant.

Once lathyrism sets in, it is difficult to cure. It is more common in males than females, especially in productive age group (15-45 years). Therefore, work output decreases and it is a chronic ailment. It takes 2-4 months for the symptoms to appear when 20% of total energy from diet is from kesari dal.

Symptoms—It is a neurotoxin which can also affect the skeletal system. It is, therefore, called neuro-osteo-lathyrism. The first symptom is pain and cramps in legs, (myospasms) numbness of extremeties, paralysis of limbs and uninary bladder and rectum (i.e. loss of sphincter control), and finally death. Because skeletal system is affected, walking is improper; thus called "weakling disease". Once it sets in, it is incurable, the person ultimately starts walking on four, but it takes 6 months to appear. Therefore, if diagnosed earlier, stop the intake of dal so that disease does not progress.

Prevention:

1. Identity the dal and discourage its use (looks like arhar dal).
2. Crop variety can be developed which has low BOAA.
3. Parboiling prevents the toxin.
4. Detoxify the dal by procedure given by NIN (National Institute of Nutrition, Hyderabad) Detoxify at home level by soaking the dal for 24 hours. Decant the water, put seeds in boiling water, Let it remain for 2 days, decant the water, dry it. The seeds have reduced amount of toxin. This method should be educated to the related community (mostly in Madhya Pradesh and Bihar)

Argemone seeds—Argemone is grown along with mustard and colour of the seeds is same as mustard or rye. The adulteration can be accidental or intentional because, it is very rich in oil (60.65% by weight of seeds) as compared to mustard (40% by weight of seeds).

The toxin in called "sanguanarian" and results in "Epidemic dropsy." It occurs in epidemic because contaminated mustard oil may be consumed by many people in that area. Symptoms are nausea, vomitting, diarrhoea, swelling of limbs, pitting odema, redening of exposed skin, swelling of face and eyes and cardiac arrest can take place in uncontrolled cases, leading finally to death. Occurence of symptoms is so fast that death occurs before any control is possible. In 1973, 172 persons died in West Bengal due to unintentional adulteration of mustard oil.

Prevention of dropsy is possible only by visual inspection of mustard seeds or rye seeds with argenome. From oil it is difficult to identify the toxin, nor is the toxin destroyed by heat. By commercial processing the mustard oil, the toxin can be removed (called degumming of oil), but this is not possible at home level.

Shell fish—Rivers and ponds contaminated with industrial wastes, radioactive wastes etc. can increase the toxic load of water plants and animals. Water animal like fish, shell fish, prawns and lobsters can be the carriers of chemical or radioactive toxins. Some marine animals have toxic acids and phenols which are not edible. Shell fish has saxitoxin which lead to paralysis of limbs and brain damage.

III. **Microbial poisoning**

This class of poison is produced by bacteria or fungi. In this class we will give especial attention to

- — Botulism
- — Staphylococcal poisons
- — Other microbes like Salmonella, Streptococcus fecalis, Clostridium perferinges, E. coli, B. coli etc.
- — Fungal toxins or mycotoxiiis like Ergot poisoning, Aflatoxin.

Sometimes, Nematoda infections like of Trichinella spirialis are also included, only they are infestation with nematodes (worms). They enter in human body as cysts from an infected food e. g. pork and the larvae from the cysts affect the intestinal mucosa or they migrate in blood to affect other tissues. This category is not strictly a poisoning, but is an infection giving similar symptoms of nausea, anorexia (i.e. lack of apetite) colic pain, vomitting, diarrhoea, sweating and soreness of infested muscles.

Botulism—It is caused by Clostridium botulinum. It is very rare, but is fatal. Common foodstuffs which are associated as the carrier of its toxin are sausages and preserved foods which are not prepared well like caused meat, smoked meat, home or improperly canned foods etc. Usually in non acidic foods, they grow, multiply and produce toxin.

Symptoms of botulism—First and foremost is the occurrence of dysphagia (i.e. difficulty to swallow the food). There is double vision, nausea, diarrhoea, fatigue, dizziness, headache, difficulty in speech, difficulty in breathing and finally death because of the paralysis of the respiratory tract.

The symptoms am produced in 1-3 days (incubation period), and usually by the time the symptoms appear, the toxin has already caused enough damage which is irrevocable. An antitoxin therapy becomes ineffective but other people in the community who have consumed the contaminated product can be saved.

The toxin is a neurotoxin and the bacteria are rod shaped, which can grow even in the absence of oxygen (anaerobic). Optimum temperature for their growth is 35°C (10-48°C) in 30% moisture with a pH of 5 or above (i.e. below 4.7 it cannot grow). Destruction of bacteria takes place at 121°C in 15 minutes. High salt concentration also inhibits their growth. The toxin itself is heat labile and gets destroyed in $^{1}/_{2}$—6 minutes at 80°C.

Treatment—Enema (washing the colon and rectum with soap solution) is given. The patient cannot tolerate anything and therefore does not eat anything. To prevent dehydration due to loss of water arid electrolytes in stools, vomits, electrolyte solution (oral rehydration solution, Annexure 1) is given.

Prevention—Even I mcg of toxin can cause poisoning, so it is recommended that a can in which there are bubbles of gas or have an off odour, should not be even tasted. The toxin is heat labile and gets destroyed at 80°C, but the spores of bacteria are very resistant. The germination of spores can however be prevented by keeping the pH below 4.7.

The prevention of the growth of the bacteria or the production of toxin can be ensured—

1. Use of approved heat processes for canning; i.e. pressure cooking for low acid vegetable, fruits and meats at 115.5°C 10lb pressure for 20-45 minute.
2. Reject all gassy or swollen canned foods, or if the tin is otherwise spoilt or damaged or give some offensive odour.
3. Refusal to even taste the contents of doubtful food.
4. Consume only freshly cooked food which has gone through good heat treatment.
5. Smoked meat and fish are also important carriers and therefore, sanitation should be maintained while handling, processing or serving the food. It has to be ensured that food is heated at least for 30 minutes in the corest part to 82°C. The fish should be frozen immediately after smoking and packing.

Staphylococcal food poisoning—In the commonest type of microbial food poisoning, usually type A strain is responsible.

The usual carriers are milk, cheese, cream filled bakery products (like cream rolls, eclairs), custards, ice creams, sometimes even sausages and cured meats. Usually, starch and protein encourages the production of the toxin.

The optimum temperature for growth is 98°F, but it can grow in wide range of 40-116°F (optimum temperature—98°F). The toxin production is maximum between 17—97°F. The optimum pH for growth of bacteria and toxin production is 5 and below 4.8 growth is retarded. They are aerobic organisms, thus need oxygen for their growth.

Staphylococci are present in clusters or bunches of grapes which is present in throat, nasal passages and pimples, boils and wounds etc. Nasal droppings of persons suffering with cold are rich in this bacteria.

Symptoms—These are vomitting, nausea, salivation, diarrhoea, abdominal cramps, fatigue and feeling to just lay down. In moderately severe conditions, headache, sweating, muscular cramping are reported.

The symptoms may appear within 25-30 hrs after taking infected food. Exact incubation time on susceptibility of the person and amount of food eaten. Unlike the neurotoxin producing botulism, the toxin here is enterotoxin because it affects the gut. The intestines get inflammed within hours, provided the conditions are favourable.

Treatment—Recovery is quick i.e. almost 1-2 days. This can be facilitated if the patient is given electrolyte solution (ORS, Annex. 1) and almost no solids are given. Ispaglula husk has been recommended by dietitians because this dietary fibre is soft, non irritant and viscous. It acts as a stool binder and absorbs the toxins. No other medicine should be given because toxin gets washed out as diarrhoea or vomitting. The only precaution is against dehydration for which ORS is recommended.

Prevention—To prevent staphyococcal infections, following precautions must be taken-

1. *General sanitation*-food handling should be done under sanitary conditions especially in restaurants, hotels, canteens etc.
2. *Proper storage*-all perishable foods should be stored under refrigeration, otherwise they make excellent media for the growth of the organisms. Similarly frozen foods should always be in frozen form.
3. *Cooking*-foods should be properly heated and cooked before consuming.
4. *Personnel management*-all the personnel in catering should not have sinus infections or infected wounds, boils or cold; and people suffering from it should not be allowed to handle the food.
5. *Pasteurization* of milk destroys the organism, but if toxins are produced before hand, than can still cause poisoning as the toxin is not heat labile. To kill toxin, the food should be boiled for 20-30 minutes or more.

Other bacteria causing food Poisoning are Salmonella, Streptococcus fecalis, Clostirdium perfringes, E. coli, B. coli etc. Most of these bacteria should be ingested in million to cause poisoning.

Salmonella—The food poisoning is called salmonellosis. The foods that act as carriers are meat, sausages, processed meat, eggs of infected poultry,,raw vegetables like salads. The incubation period is 7-12 hours and the symptoms are nausea, vomitting, diarrhoea, abdominal pain, fever, muscular weakness which last for 2-3 days. Usually there is complete recovery. Prevention is same as for other microbial poisoning.

Streptococcus fecalis—Usually animal food are involved like sausages, meat products that are not properly processed. Incubation period is 5-12 hours. Prevention is same as for other bacterial poisoning.

Clostridium perfringes is the bacteria that forms spores and is present is soil, water, milk, sewage and the intestines of man and animal. Foods involved are meat, fish, poultry and milk. The incubation period is 10-12 hrs. Usually symptoms are nausea, abdominal pain, diarrhoea, but vomitting is not usual. Although stools are offensive, the recovery is rapid.

Mycotoxins

Aparts from bacteria, the toxins of various fungus are also harmful. These toxins are called mycotoxins e.g. aflatoxin, ergot toxin etc.

Aflatoxin is produced by the fungus—Aspirgillus flavus. This fungus usually grows in water (30-37^{o} C), damp (moisture above 16%) conditions on ground nut, maize, cotton seed or its cake (i.e. defatted cotton seeds), and also on wheat, rice, jowar or soyabeans. Aflatoxin affects the liver of the animal or human being, it can cause liver cirrhosis marked with jaundice and ascites followed with cancer and death. If breast milk has aflatoxin, child can have infantile cirrhosis.

Prevention—Foods should be stored in dry conditions, preferably in tins which can be fimigated with antifungal agents. Doubtful food articles (i.e. with grey-green fungus) should not be consumed lasted. Pressure cooking does not destroy the toxin, but heating under alkaline conditions gets it destroyed Hand-picking and discarding infected seeds (groundnut, maize, rice etc. in) can reduce the contamination. Now by genetic approach, seeds resisting this fungal growth are grown.

Ergot toxin—the poisoning is called ergotism. Usually foods like bajra, wheat, Jowar causes ergotism, because of the fungal growth of ergot on these millets. When the food is infested, the food poisoning is characterized by diarrhoea, nausea, giddiness, vomitting, abdominal stress, headache painful cramps; all of these cap become severe.

Summary

Food poisoning occurs from very diverse sources like-chemicals, plant and animal toxins bacterial toxins and fungal toxins. Prevention is therefore specific for each source. Some toxins are heat labile, some can be reduced by parboiling (lathyrus toxin-BOAA) and some can be washed off. Some toxins are very resistent to heating processing or just washing.

General principle of management are:

1. Identify the cause of poisoning and prevent further poisoning. Treat with antidote if known.
2. Gastroenteritis and other disturbances are usually seen like nausea, vomitting. The patient requires nourishment, therefore, food should be presented aesthetically to overcome nausea. Give plenty of water to flush toxin from GIT and overcome nausea. Water can be substituted with electrolyte mixture (oral rehydration education), so that water which is lost in vomitting or diarrhoea is compensated. Water absorption is better in the presence of glucose and salt (sodium chloride); further glucose is absorbed better in the presence of salt. Therefore ORS is better accepted than plain water.

Composition of home made ORS

1 Litre of boiled cooled water (5 glasses)

5 g of sodium chloride (pinch of common salt)

25 g of sugar (handful of table sugar)

1 lemon juice to taste is dissolved and the patient can drink in 5-6 divided dose to prevent dehydration.

3. Whey, dal water, rice water (kanji) juices etc. can be given, but food should be avoided because the patient may not tolerate. Fluids as drinks, juices, soups etc. should be given at 2-hrly intervals, round the clock i.e. 24 hrs day and night in amounts not more than 150 ml per feed.
4. Soft fibre like mucilax or isabghol (ispaghula husk) give bulk to stools, reduce watering stools. It also absorb the toxin and entangles it and removes it through fcaces, instead or letting it get absorbed. This removal of toxin by fibre may not be possible for all types of toxin and by not all types of fibre. Harsh fibres cannot be given, as they can further irritate GIT.
5. The person must report to the hospital. ORS alone cannot treat or prevent further damage, some toxins are fatal and needs to be washed off by dialysis or require antidote.
6. If epidemic of adulteration, it should be made public like epidemic dropsy, methyl alcohol toxicity etc. Mass media are used like radio and televisions to make people aware within hours of its occurrence.

Selected References:

1. *Textbook of Preventive and Social Medicine;* Park & Park.
2. *Advanced Textbook on Foods & Nutrition;* Vol. II: Dr. M. Swaminathan.
3. *Foods & Nutrition for Senior Students;* Education planning Group; Arya Publications.

Lesson 16

FOOD SAFETY

Food has a well recognised function. It is essential for 'growth and maintenance of health. But food can perform this basic function only if, it is safe. It should be safe in nature and free from toxins and such other components like additives and contaminates which may interfere in assimilation of nutrients or prove to be potential health hazard.

Every food may not be safe from health point of view. When is food fit to eat? It is fit to eat, if, it is free from harmful contaminants, toxins, bacteria, etc. and after consumption the person does not feel sick.

Factors Affecting the Safety of Food

The type of food systems by which food is produced, processed, distributed, prepared and consumed is related to the stage of development, level of income and socio-culture characteristics of communities.

The food system can be broadly divided into three important categories namely:

(a) Low income rural system.

(b) Low income urban system.

(c) High income system.

Each system has its own particular food safety problems and require different remedial approach.

(a) *Low income rural system:* Since most of the food utilized in this system is produced locally, the primary areas of concern are:

(i) The frequent contamination of grains and pulses during harvesting by soil borne spores of organisms such as Bacillus cereus and Clostridium perfringens.

(ii) The contamination of food at the time it enters the home possibly through the use of non-potable water in growing or washing vegetables and fruits before eating as raw foods.

(iii) Poor practices in drying, storing, handling and preparing the staple food, and/or use of non-potable water.

The main food contamination threats appears to be raw food, non-potable water, inadequate domestic sanitary practices, (practically improper food-handling practices, especially of cooked food), and in-appropriate storage practices.

(b) *Low income urban system:* The basic areas of concern of this category are:

(i) The growth of centralized markets as primary distribution points for food supplies purchased from large number of small farms in the rural areas and transported by assembly traders to the urban areas.

(ii) The greater tendency for cereal staples to be ground, partially processed or cooked before the retail stage, and for developing separate marketing channels for these commodities.

(iii) An increase in the quantity of simple processed or prepared food for consumption inside or outside the home and mostly produced by relatively small scale processors.

(iv) A retail system consisting of multitude of small scale traders, vendors and retail outlets, each selling small quantities of raw, processed or ready to-eat foods.

(C) *High income systems:* As income rises, the percentage of available income spent on food declines. People can afford a more varied diet and are likely to purchase more semi-processed or processed foods that incorporate variety and reduce time spent on food purchase and preparation.

It is because of these reasons that the population failing in this category is more depended on industrial processing techniques. The potential problem thus arises from failures in the quality (and safety) control of processed and prepared foods by the industrial food processors or mass caterers.

Suggested ways to Food safety in General

Food hygiene covers all measures necessary to ensure the safe, whole-someness, soundness of food at all stages from its growth, production or manufacture, until its final consumption. Because there are so many sources from which disease agents may be transmitted to food and because food can become easily contaminated by these organisms, food must be protected at all points along the food chain, from the time it is produced until it is served to the consumer.

All the measured or precautions practiced in applying the principles of food protection, can be classified into 4 basic rules:

(i) Keep food clean.

(ii) Store in cool place.

(iii) Serve food hot.

(iv) Don't keep food in dirty places.

In particular, the following can ensure food safety and prevent food borne illnesses.

A. *Personal Hygiene*

"Human element" is the single most important factor in the control of food borne illness. Every individual must follow some routine habits like hand washing throughout the preparation of meals to avoid possible risks of contamination. Cuts, sores, burns and , boils should always be covered with water proof bandages because they also carry food poisoning bacteria. Food service workers in food service establishments must be free from all communicable disease.

B. *Food must be secured from Approved Sources*

Since food come from different places, one has to ensure that the incoming food is safe for consumption.

- *Water supply/potable water:* Water through not considered as food yet, plays an important role in human nutrition. When contaminated, it transmits disease through the pathogenic organisms it contains or through the utensils and equipment washed in it. Thus, it is necessary to use safe water for consumption.
- *Safe sources of food:* Most foods supports microbiological growth. The consumers should take particular care to purchase only uncontaminated foods. Milk and milk products should be pasteurized. Meat and poultry should be slaughtered and processed in sanitary conditions. It should bear the label of an official inspection agency. Canned food should be processed in authorised food processing plants.

C. *Store Foods Properly*

Foods are not usually prepared and served just after purchase. It is stored for varying lengths of time and in a variety of conditions until it is prepared for service. Thus, subjecting to contamination in a variety of ways.

- *Protection during dry storage:* Foods placed in a dry storage should be arranged and stored so that the food placed in first is used before newer incoming supplies, that it, "First in, First out". All foods should be stored off floors on pallets and shelves and, if possible, away from walls thus preventing it from being contaminated by insects or rodents or from flooding.
- *Avoid wet storage of packaged food:* Never store packaged foods like cartons or bottles of milk and juices in iced water to keep them cold. Water in which the container is stored is contaminated through use and seepage through leaks may aggravate the problem.
- Temperature has relation to food protection: Food, moisture, temperature and time are some of the requirements for the growth of organisms. There is little opportunity to control the first two and so prevention of contamination depends on controlling the temperature of food to control microbial growth.

Temperature Guide (F)

	180	
To serve hot Foods, store while Serving above 140° F (soups, gravies	170	
	160	
	150	
Meats etc.)	140	Some bacterial
	130	growth may occur
	120	
DANGER ZONE Most bacteria including food poi- soning type will grow rapidly over this temp. range.	110	Ordinary room temperatures may fall in this
	100	range. Do not
	90	store prepared foods at room temperature.
	80	
	70	
	60	
	50	Some bacterial
	40	Growth may occurs
Refrigerate Pre Pared foods to prevent bacterial growth and to prevent food spoilage. Store frozen Foods at this tem- Perature or be Low	30	To serve pre pared cold dishes keep on ice while serving
	20	(Potato salad,
	10	Chicken salad etc.
	0	Thaw or cook from frozen con- dition when Ready to use.

Microbial organisms grow best at temperature near that of human body or about 100°F (37°C). Disease organisms grow over a wider range of temperature in the danger zone where the microbial growth is rapid, i.e. 45°-140°F (4°C-60°C). Therefore food should be stored either below 45°F (4°C) or above 140°F (60°C) but not for too long.

Keep food cold so that micro-organisms do not grow very well to reproduce in numbers sufficient to cause illness. This temperature or even freezing and refrigeration does not kill most micro-organisms but only slow down or halt their growth temporarily. If temperature rises significantly, any organism present in the food begins to grow.

On the other hand, in case food held is at 140°F (60°C) or above, micro organisms are killed rapidly but are not readily affected when the temperature goes below 140°F(60°C). So after food is cooked, it should be kept hot and not just warm, until served. If it is kept "just warm" on the back of the kitchen range or on the table, rapid microbial growth may occur and outbreak of food borne illness could result.

D. ***Practice Proper Preparation of food***

- *Work with clean hands:* Hands must be washed often with warm water and soap to keep them clean. Though many foods are handled by hands during preparation, practice using sterilized handling utensils for food like forks or tongs. This is not only appealing but shows concern about health of the consumer.
- *Use clean utensils:* Clean contact surfaces of tables and equipments. Prompt and throughout washing and sanitizing between uses prevent outbreaks from occurring.
- *Clean raw fruits and vegetables:* Fruits and vegetables are grown and handled in many different conditions. Soaking followed by throughout washing in running water is usually sufficient to remove most chemical contaminants or disease organisms.
- *Avoid left overs:* Food kept for a long time between preparation and serving is more likely to cause disease. If leftover prepared foods (unserved) are to be kept, adequately protect and refrigerate them. Serve hot food again by rapidly reheating to above 140°F (60°C) to boiling temperature.
- *Practice healthy habits:* Habits of individuals are most important in providing proper protection during food preparation. Therefore, habits as placing fingers in the hair. Picking at the nose, or coughing and sneezing are to be avoided when preparing food.
- *Cook foods properly:* In cooking see that all parts of the food reach the proper temperature. The principles that apply to cooking foods also apply to reheating of food.
- *Store and display food properly:* Cold foods on a serving line must be maintained at a temperature of 45°F (4°C) or below and not that of the ice or cold plate on which the food is placed. Hot foods on a serving line must be maintained at 140°F (60°C) or above. This must be the temperature of the heating unit at which the food is held.

  Serving of foods should be done with an appropriate utensil.

  Pack foods properly. Packaged foods that have been opened once should not be offered for resale or reservice.

E. ***Observe Proper Practices for Refrigeration of foods***

1. Foods must be refrigerated at all times except during the period of actual preparation or serving. Hot foods removed from the range should be refrigerated immediately after cooling to shorten the time it is in the danger zone.

2. Thaw frozen food quickly.

   Frozen foods should be kept frozen at all times. They should be kept in the freezer of the refrigerator until thawed and newer at room temperature.

   For quick thawing, place the food in a clean plastic bag and suspend in running 70°F or less) tap water and cook promptly.

3. Arrange food in containers in refrigerator.

Store foods in shallow pans or in smaller round containers to cool Rapidly. A good rule is to make certain that the distance to the centre of the food is not more than 2 inches. This means that shallow pans or even round containers should be filled not more than 4 inches deep.

Stir the food occasionally to bring warmer parts of the food mass closer to the surface to dissipate heat rapidly.

Place containers in the refrigerator in a manner that permits air circulation around each container.

Cover all containers in the refrigerator to prevent contamination from the shelves and among the foods in the refrigerator.

Clean refrigerator thoroughly with a detergent.

F. ***Practice Hygiene at home and at commercial and Community Feeding Establishments***

Proper ventilation, lighting, floors, adequate supply of safe drinking water and water for washing drains, surface drainage, regular collection and storage of garbage in covered bins and removal at least daily, sanitary latrines control measures for flies and rats are pre-requisite and should invariably be observed.

G. ***Observe Legislation Procedures***

To support proper investigation in matters of food sanitation, ordinances and regulations are promulgated, which is discussed in the next chapter.

Lesson 17

FOOD STANDARDS

As already discussed in the proceeding chapters, adulteration of food stuffs is on the increase. It is a menace which saps the vitality of our people. The Indian consumer, in general is blissfully ignorant of the harmful hazards caused by various adulterants, and is mostly guided by the price, visual appearance and claims made by the advertisements. To safeguard the people from the health hazards posed by the practice of adulteration, it is necessary to exercise a strict check and control over the quality of foods offered for sale in our different markets. As already mentioned, according to the consumer the most important factors that determine the quality are price, color and appeal in respect to both appearance, and taste. But what really is quality? It has been stated to be the product and not the sum of all the variable involved. If any one of the desirable characteristics approaches zero, the quality and therefore, the product acceptance itself approaches zero.

To understand the quality features of a certain food stuff, it is essential to know the meaning of the word "quality". It is defined as character, kind, property, status grade of goodness, excellence. The parameters of the quality are the grades, standards and specifications laid down by the government of expert bodies constituted for the purpose. However, there may be varying and numerous qualities. An established system of quality control assures uniformity in accepted standards and thereby ensures that each food stuff is what is purports to be and what its label claims to be.

Standardization is a method by which quality control can be maintained. This is done to maintain the minimum standard necessary for foodstuffs. Standard is something that is set up and established by authority for ensuring quantity, weight, extent, value of quality.

International Food Standards

The International codex Alimentarius Commission is the principal organ of a world-wide food standards programme, under the joint auspices of FAO (Food Agricultural Organization and WHO (World Health Organization) two specialized agencies of the United Nations Organizations. The Commission's main task is to prepare an international codex alimentations, based on principles outlined in statement prepared by the commission itself.

General Principles of Codes Alimentarius

Purpose of the Codex Alimentarius

The Codex alimentarius is a collection of internationally adopted food standards presented *in* a uniform manner. These food standards aim at protecting consumer's health and ensuring fair practices in the food trade. The publication is intended to guide and promote the elaboration and establishment of definitions and reauirements for foods, to assist in their harmonization and in so doing to facilitate international trade.

Scope of Codex Alimentarius

The Codex Alimentarius is to include standards for all the principal foods, whether processed, semi-processed or raw for distribution to the consumer. The Codex Alimentarius is to include provisions in respect of food additives, pesticides residues, contaminants, labelling and presentation, methods of analysis and sampling.

Nature of Codex Standards

Codex standards contain requirements for food aimed at ensuring for the consumer, a sound, wholesome food product free from adulteration, correctly labelled and presented. A codex standards should, therefore, for any food or foods:

1. Incorporate by reference the applicable hygiene, labelling, method of analysis and other general provisions adopted by the commission; and
2. Specify in whole or in part the following criteria, as appropriate:
   (a) *Product designation, definition and composition*: these should describe and define the food and cover compositional requirements which may include quality criteria.
   (b) *Hygiene requirements*: These should include such factors as specific sanitary and other protective measures and safeguards to assure a sound, wholesome and marketable product.
   (c) *Weight and measure requirement,* such as fill of container, weight, measure, or count of units based on an appropriate method or criterium.
   (d) *Labelling requirements:* These should include specific requirement for labelling presentation.
   (e) *Sampling, testing and analytical methods:* These should cover specific sampling, testing, and analytical procedures.

HACCP

An important guideline of Codex Alimentaries Commission for the food processing companies is to follow a food quality management systems called HACCP.

HACCP has been defined as systematic approach to be used in food production as the means to ensure food safety. Seven basic principles underlie the concept and these principle include an assessment of the inherent risk that may be present from the harvest till ultimate consumption. These principles are :

1. Assess Hazards and Risks associated with growing, harvesting, raw material and ingredients, processing, manufacturing, distribution, marketing, preparation and consumption of food.
2. Determine critical control points required to control the identified hazards.
3. Establish the critical limits that must be met at each identified CCP.
4. Establish procedures to monitor CCP.
5. Establish corrective action to be taken when there is a deviation identified by monitoring a CCP.
6. Establish effective record keeping system that document the HACCP plan.
7. Establish procedures for verification that HACCP system is working correctly.

Foods Standards in India :

A number of existing food standards in India are based on the international codex alimentarius, with relevant modifications and additions wherever necessary. The most important of these are:

* Prevention of Food Adulteration Act (PFA)
* 'Agmark' Standards (JAGMark)
* Fruit Products Order (FPO)
* Specifications of Indian Standards Institution (ISI)

Quality standards are descriptions of commodities in terms of net weight, accurate size, dimensions, content and other characteristics. The following gives an idea of the kind of specific requirements laid down as quality standards:

* Establish the minimum and also maximum content of one or more components of food, e.g. cooking chocolate contains not less than 50% and more than 58% by weight of cocoa fat.
* Establish the ingredient content of food e.g. jams made of mixtures composed of 45 parts by weight of fruit constitutent and 55 part by weight of sugar.
* Establish the minimum quality of one or more ingredients of food, e.g. margarine contains no less than 80% fat.
* Describe processing requirement e.g., sealed in a container and so processed by heat to prevent spoiling.
* Identify the species of plant that may have been utilized.
* Define packing media of any foods such as water, syrup, juices and oils.
* Prescribe essentials in production e.g., bread is prepared by baking a kneaded yeast leavened dough made by moistoring flour with water or other specified liquid.
* Require label statement of the use of any food additives with explanation of use.

1. **The PFA Standards**

These precribe the minimum standards for all types and categories of food. The PFA rules were fjist introduced in 1955 and have been subsequently amended in 1968, 1973 and 1978 and this has been reinforced since June 1st 1945. The standards are formulated and revised when required, by an expert body called the central committee for food standards (CCFS). Only food does not conform to the minimum standards laid down by the PFA rales, is said to be adulterated, irrespective of whether anything has been added to or removed from the original food.

The regulation consist of many articles direction towards the protection of food adulteration. Some of these are discussed below :

1. **Section II** : This section gives some common definitions like definition of adulteration, definition of food false labels.

(a) *Definition of Food Adulteration* : This has been discussed in chapter 13 already.

(b) *Definition of Food Stuff:* All the liquid and solid products that are ingested (except water and medicine are termed as food stuffs.

(c) *False Label* **:** The following conditions define any product with false label :

(i) If the label is deceptive or not true,

(ii) If the product is sold by some another name.

(iii) If the product is the copy of some other product and the name of the product is not mentioned on the label.

(iv) If the container is such that is deceives about the amount it contains.

(v) If the lable does not mention the name of the manufacture and the products used and their standard.

(vi) The lable cannot be read easily.

(vii) If the artificial colors and chemical preservatives used are not mentioned,

(viii) If the standard of the food product is not mentioned.

2. **Section (V)** : This Section mentions the food products whose sale is prohibited in India (i) adulterated food stuff: (ii) Food products with false lable; (iii) Import of licenced food stuffs; (iv) any other food product which is not in this law.

3. **Section (X)** : This Section mentions the rights and duties of food inspectors (i) Food Inspectors can take the sample of any food product to sent it for testing and keeping the public health in mind, he can prohibit the sale of any food product with the prior permission of food officer (ii) Food Inspector can take the sample from any place of inspection (a) where the food stuff is produced; where it is stored and (c) where it has been kept for sale. (iii) When the food adulteration has been certified, food Inspector can assess the seller. He (iv) can take possession of the adulterated food stuff and send the, same for testing.

4. **Articie (XI)** : According to this article, when the food inspector takes samples for testing, then (i) then he will give notice to the seller, then the sample has been sent for testing : (ii) The sample will be sealed in three different parts : one for seller, one for testing and one for himself.

5. **Article (XII)** : According to it, any food stuff suspected for adulteration by the consumer, can be sent by him for testing, and so proved, the testing cost is reimbursed to the consumer and the seller is taken by the law.

According to PFA act, a person keeping, selling or transporting the adulterated food stuff may be jailer from three to six months and fined for a sum from Rs. 500 to Rs. 2000. If the same person is caught again, his name and offence is published in the local newspaper and the expenses are borne by the same person. If a consumer dies after consuming adulterated food stuff, then he offencer may he given life imprisonment.

2. **Fruit Product Order (F.P.O.)**

The Fruit Products Order was promulgated by the Government of India in 1946, under the Defence India Rules. In 1955, the order was revised and section III of the Essential commodities Act, which was enacted that year. This order stipulates minimum fruit and vegetable products manufactured in India. Under this act, every manufacturer for fruit and vegetable products must obtain a licence before starting production and the products should confirm to the standards prescribed under the order. The latest amendment was made in 1975.

The Central Fruit Products Advisory Committee constituted under the order is expert body which advises the Central Government on Standards and policy matters relating to the vegetable and fruit industry. Any product which deviates from the minimum prescribed standards under the order is decalred sub-standard and in case the office is repeated, the producer is liable to punishment under the provision of law. The Main objective of the F.P.O. is to ensure minimum standards for various fruit and vegetable product. The order specifies standards and sanitation and hygiene to be followed in the factories besides giving directions regarding packing, marking and labelling of containers. The inspectors attached to the Deptt. of Agricultural Marketing are empowered to collect samples and inspect the factory and send the samples to the control testing laboratory located at Central Food Research Institute, Mysore. The order has further laid down limits for the presence of poisonous elements, permitted food colors, preservatives and other food additives.

Fig. 1

The following are the products of fruit and vegetables marked with FPO :

(i) Preserved fruit (in cans and bottles)

(ii) Preserved vegetables (in cans and bottles)

(ili) Fruit juice and fruit pulp (in cans, bottles)

(iv) Fruit drinks like Rasika, Mangola etc.

(v) Jam, Jelly and Marmalade.

(vi) Squash, Syrup and Cordial.

(vii) Pickles, Chutneys and Preservers.

(viii) Dried fruit and vegetables.

(ix) Candid and Glazed Fruits

(x) Freezed Fruit, vegetables, fruit, juice and fruit pulp.

(xi) Sherbet and carbonated drinks like Cola drinks etc.

(xii) Synthetic vinegar.

Obviously, the objective of the PFA and FPO standards is to obtain a minimum level of quality for food stuffs, consistent with the minimum quality attainable under Indian conditions by the majority of farmers, processors, sales and distribution agencies. Needless to say, these can never cover excellence nor the zenith of quality. In fact these minimal requirement sometimes act as a deterrent to improvement of quality above the prescribed minimum.

'Ag Mark' Standards

This was set up by the Directorate of marketing and Inspection of the Government of India to cover the various quality levels of agricultural commodities. The Grading and Marketing of Agricultural Products Act of 1937, defines standards for the quality of creals, oilseeds, oils, butter, ghee, legumes, eggs, etc. and provides for the categorisation of commodities into various grades depending on the degree of purity in each case. The several grades are : (i) Special (Grade 1); (2) Good (Grade 2); (3) Fair (Grade 3); and (4) Ordinary (Grade 4). These standards also specify the type of packaging to be used for different products.

AgMark specification are mainly formulated on the basis of physical and chemical characteristics, intrinsic as well as acquired during processing or otherwise. The agricultural practices prevailing in the country, consumer preferences and availability of the different farm commodities are taken into consideration while framing these standards.

The benefits of Agmark standardisation are made available to public by providing for Agmarking of articles of foods such as edible oils, butter, ghee, eggs, etc. The mark gives the consumer an assurance of quality in accordance with the standards laid down. This system of grading and marking has made better buying and selling of agricultural produce possible by establishing regulated markets, the salient features of these markets are healthy market practices,

use of standard weights and measures, licencing of market functionaries, arrangement for settling disputes in regard to quality, weight and discounts.

Ag Mark certification is passed only after elaborate testing. All the Food processing Factories with Ag Mark certification appoint an expert (chemical) with the permission of Ag Mark Officers, who checks and controls the raw materials and produced food product for quality control, Ag Mark inspection can take sample from the factory or open market and can get it analysed in dependently in their food laboratory. If the testing shows that the product is below the required standard, then factory's chemical expert services are terminated and the sale of that batch of food product is prohibited.

List of Ag Mark certified Food Products :

1. Ghee
2. Butter
3. Edible Oil
4. Honey
5. Pulses
6. Ground spices
7. Wheat Flour
8. Besan

Meat Foods Product order

This makes it illegal to transport meet unless is has been prepared and processed according to the provisions of the order and carries the make of inspection. It provides for means to:

a. detect and destroy meat of diseased animals
b. ensure that the prepcration and handling of meat and meat products be conducted in a clean and sanitary manner
c. Prevent the use of harmful substances in meat foods.
d. see that every cut of meat is inspected before sale to ensure its wholesomeness.

The order also lays down rules and conditions for procedures to be adopted for the selection of disease free animals, slaughter house practices and further treatment of the meat so as to maintain the meat in a wholesome manner, devoid of pathogens.

BIS (Bureau of Indian Standards)

BIS was found in 1986 under BIS act. It comprises of members representing industries, consumer organizations, scientific and research institutes, professional bodies and ministries. Function of BIS is expert promotion of food products which are prepared and processed in safe hygienic conditions.

So far 15,000 standards have been formulated in different technological areas and these standards fall in the following categories:

1. Product specification
2. Method of Test
3. Quotes of practices & guidelines
4. Terminologies
5. Basic standard

BIS is founder member of the International Organization for standardization, ISO. It is also involved in international electro-technical commission and also has a particular status in 46th technical committee. In addition to the mandatory acts and order, agencies like ISI and Directorate of Marketing and Inspection have also laid down quality standards of foods. These are refered to as voluntary standards.

(1) AGMARK

(2) ISI

Indian Standards

Indian Standard Institution was established in 1947. The ISI is the national standards organisation for India, responsible for laying down standards in consultation with, and with the active participation of experts drawn from manufacturing units, research and technical institutions and laboratories, purchase organisations and other parties involved. Following are the objectives of Indian standards Institution:

1. To formulate standards for commodities, products, materials and processes.
2. To encourage their adoption at National and International level.
3. Certification of Industrial Products.
4. Help in production of Standard Products.
5. Distribution of information regarding standardisation.

The Indian Standards are available for vegetable and fruit products, spices, condiments, animal products and various processed foods. The standards are formulated and revised from time to tme by various committees set up by the Indian Standards Institution *(1ST)* for the purpose. The standards are evolved on the basis of physical characteristics and chemical as well as biological assessment as and when required.

The ISI certification Marks Scheme under the Indian Standards Institution (Certification Marks) Act, 1952, as amended by the Amendment Act of 1961, makes provision for making quality goods with ISI stamp. The ISI mark on any food article is a guarantee of good quality, in accordance with the prescribed Indian Standards for that commodity. Licence to use the marks are granted only to producers following ISI approved methods of production and quality control. The method of manufacture and the premises as well as the final product are all subject to inspection by the ISI personnel. For analysis, the samples are sent to the independent laboratories if the product is found to be of inferior quality, the manufacturer is warned and the sale of the products of that batch is prohibited. ISI has its own laboratory at I.T.O., New Delhi and Ghaziabad, where the products of licence demanding applicants (manufacturers) are tested. Other small laboratories are in Bombay, Calcutta and Madras.

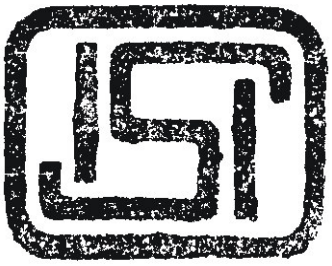

Fig. 3

Agencies of international level like I.S.O. Organization for standardization and I.E.C, (International Electrotechnical Commission) helps in certification and promote and encourage it.

Following are the eleven departments, under which the cerufication is passed.

1. Agricultural and Food Products
2. Chemicals
3. Civil Engineering
4. Consumer Products and Medical Instruments
5. Electronics and Telecommunication
6. Electro Technical
7. Marine, Cargo movement and Packaging
8. Petroleum, coal and related products
9. Mechanical Engineering
10. Structural and Metals
11. Textile

List of I.S.I. Certified Food Products

1. Edible common salt
2. Arrowroot
3. Custard Powder
4. Biscuits
5. Baking powder
6. Cocoa powder
7. Milk powder
8. Condensed Milk
9. Baker's yeast
10. Infant Milk Food
11. Vermicelli, Macroni and Sphagetti
12. Besan
13. Cheese
14. Coffee Powder
15. Ice Cream
16. Egg Powder
17. Saccharin
18. Drinking chocolate
19. Rum, Beer, Gin, Whisky, Brandy

List of some domestic Electrical equipments with I.S.I. Mark

1. Electric Iron
2. Electric Fan and Regulator

3. Electric Kettle and Jug
4. Electric Hot Plate
5. Domestic Food Mixers
6. Switches
7. Pressure Cookers (Non Electric)
8. Gas Stove

Export Inspection Council : This council has been constituted to check quantity of number of food materials meant for export.

Standard for weights and measures Act, 1976 : This act establishes standards for weights and measures to regulate inter-state trade or commerce of goods which are sold or distributed by weight, measure or number. This Act aims at the caliberation of the use of weights or measures as a standard and this is used in case of commodity in packaged forms. It defines the standard units of weights and measures which are based on metric system. It also quotes the derived unit which means a unit derived from the base or a supplementary unit or both.

Ecomark : The Government of India has instituted a scheme known as ecomark, for labelling environment friendly products. This scheme administered by BIS provides labelling of household and consumer products which meet certain environmental criteria along with the quality requirement prescribed in ISI.

In 1992, most of the European countries joined and formed *European Economic Committee* which at present comprises of 12 member countries namely Belgium, France, Italy, Germany, Luxemburg, Netherland, Denmark, Ireland, UK, Grace, Portugal and Spain. The 7 of these members are from *European free trade association* (EFTA). In this some common standards dealing with minimum menufacturing practices for quality management and proper inspection and testing are prescribed and only those products confirming to these standards are allowed to be marketed. Thus to boost the export of European market it is necessary to confirm to these standards.

ISO 9000 (International Organization for Standardization) is about corporate white quality systems. A quality system is designed to ensure the continued repeatability of a set of product and service characteristics that have been explicitedly & implicitedly agreed to by the customer and supplier.

ISO 9000 is a series of international standards for total quality systems. The major objective is :

To facilitate international trade by creating atmosphere of mutual trust with regard to maintainance of quality of the product. ISO 9000 looks at every aspect of the company's operations including :

* Tooling Procedure
* Assembly line Equipment
* Personnel Management
* Accounting Procedures.

The Indian Standards ISO 9000 series clearly describes the quality system models, one of which can be chosen by the suppliers which is most appropriate to the nature of operation and the quality requirement of their customer. This series is a set of 5 primary standards namely ISO 9000 I, ISO 9000 II, ISO 9000 III, ISO 9000 IV & ISO 9000.

ISO 9000 and ISO 9000 IV refers to the guidelines framed whereas 9000 I, II and III address specific quality systems.

ISO 9000 states guidelines for selection and use of quality management and quality assurance standards.

ISO 9000 I refers to the quality system pertaining to the models for quality, assurance in design, development and service. ISO 9000 I which refers to the quality assurance in design, development and service is for use when confirmance to specified needs is to be assured by the supplier throughout the whole cycle from design to servicing.

ISO 9000 II refers to the quality system pertaining to the models for quality assurance in production and installation. ISO 9000 II which takes into account production, installation and servicing is for use when the specified requirement for products are stated in terms of an established design or specification and the supplier's capability in only production, installation and servicing are to be demonstrated.

ISO 9000 III refers to the quality systems pertaining to the model for quality to the model for quality assurance in final inspection and test. ISO 9000 III appliers to a situation where the supplier's capability of only inspection & test conducted on finished products can be satisfactorily demonstrated.

ISO 9000 IV states quality management and quality systems, elements and their guidelines.

These ISO series are equivalent to the Indian, British & European standards which are ISI 4000, BS 5750, EN 29000.

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Lesson 18

SOCIAL HEALTH PROBLEMS - I

SMOKING

Smoking is a worldwide public health problem. In India, tobacco smoking and chewing is; one of the principal causes of preventable disease and early death. Though tobacco has been in use for several centuries, its deleterious effects were increasingly recognised in the 20th century only. Since then, several harmful effects of tobacco usage have been brought in to focus. The medical community all over the world is constantly campaigning to cut down tobacco consumption by public at large. As a result of mass education, tobacco smoking has decreased considerably among the educated people in the developed countries. No statistics is yet available from India. Systematic studies of the deleterious consequences of tobacco smoking and chewing are also lacking in this country. Large studies in western populations have demonstrated that male smokers above 35 years of age have 70% higher death rate than non smokers.-The maximum risk of death is in 45-55 age group. The risk of excess death is also evident in females, though not as much as in males. The morbidity and mortality due to cigarette smoking is directly proportional to the number of cigarettes smoked per day, age of smoking and the degree or smoke inhalation.

(i) *Nicotine:* Nicotine is a highly toxic substance which exerts deleterious effects on the heart, blood vessels and nervous system. It has also several harmful biochemical effects.

(ii) *Carbon monoxide:* Carbon monoxide is a toxic gas that interferes with oxygen transport and utilisation in the body and may produce a subtle abnormality of the functioning of the brain.

(iii) *Carcinogens:* Carcinogens i.e. cancer producing substances are also present in tobacco smoke.

(iv) *Lung irritants:* Cigarette smoke increases respiratory secretions and produces abnormalities of the lung function.

Harmful effects of cigarette smoking

(i) *Diseases of the heart and blood vessels:* Heart attack in early life is the most important consequence of cigarette smoking. Sudden death may be the first manifestation of heart disease due to cigarette smoking. The risk of sudden death in the middle aged male smokers is 2 to 3 times more than that of non-smokers. Heavy smokers are more prone to heart disease than light smokers. Women smokers are also at a high risk of developing heart attack and the use of both cigarettes and oral contraceptives result in a 10 fold increase in the risk of heart disease. The risk of heart disease due to smoking decreases considerably, if smoking is stopped for more than I year. The risk of complications following heart surgery is more in smokers as compared to non-smokers.

Cigarette smoking is also a strong predisposing factor for poor blood circulatioin in the legs. : Sometimes the circulation in the lower limbs becomes so poor that gargerine sets in the feet and a part of lower limb needs amputation. Cigarette smoking also increases the risk of stroke (paralysis). Smoking does not produce high blood pressure by itself but it accentuates the severity of hypertnsion which can lead to several complications.

(ii) *Cancer:* Cigarette smoking is a well documented cause of lung cancer. The risk of lung cancer is directly proportional to the amount of smoking. On giving up cigarettes, there

is a gradual decline in the risk of lung cancer. Cigarette smoking is also a causative factor for the cancer of mouth, wind pipe and the food pipe. Cancer of several other regions in the body may be related to cigarette smoking or chewing.

(iii) *Respiratory disease:* Cigarette smoking is a major cause of chronic bronchitis and prolonged respiratory disablity. Patients with chronic bronchitis suffer from frequent cough with yellow sputum and breathlessness. Ultimately they die of respiratory or heart failure. Patients with chronic bronchitis are also more prone to develop pneumonia and influenza.

(iv) *Disorders of the intestinal tract:* Smokers have a higher prevalence of peptic ulcer. Healing of peptic ulcer is retarded by continued smoking. The risk of recurrence of peptic ulcer is also increased.

(v) *Smoking and pregnancy:* Smoking may delay conception. Smoking also results in low birth weight due to impaired circulation through the placenta. Smoking during pregnancy increases the risk of miscarriage and foetal death. Pregnancy may also adversly affect the physical and mental growth of the child after birth.

(vi) *Smoking and drug effectiveness:* Smoking adversely influences the action of several drugs including drugs used to treat high blood pressure and bronchial asthma.

Involuntary smoke inhalation: Contamination of atmosphere by cigarette smoke results in involuntary i.e. inhalation by non-smokers. This is called involuntary or passive smoking. Environmental contamination by cigarette smoke is now recognised as an important cause of air pollution. Its effects on non-smokers may be comparable to those occuring in light smokers. Passive smoking may contribute to development of chronic bronchitis and lung cancer.

Prevention of smoking: Cessation of smoking is primarily achieved by self motivation based on awareness of ill effects of smoking. Drug therapy plays minimum or no role in stoppage of smoking.

Alcoholism

Alcoholism has been defined as both a chronic disease and a disorder of behaviour, characterised by drinking of alcohol to an extent that surpasses the social drinking customs of the community and that interferes with the drinker's health, inter-personal relations or means of earning a livelihood. In other words, it is addiction to alcohol.

The *causation of alcoholism* is complex. Recent observations point to a genetic influence on the development of alcoholism especially in males. The incidence is higher in children of alcoholic parents than in those of non-alcoholic parents. In addition there are psychological factors, Mental tensions and serious setbacks in personal life drive a person to adopt alcohol as a means of finding temporary relief from his problems. Besides, the company of alcohol addicts gradually lures a person into the evil of alcoholism. At times individuals drink excessively with full knowledge that such a action will result in physical injury to themselves and irrepairable harm to their families. This represents total psychological dependence on alcohol in which the individual needs alcohol for adquate functioning and continues drinking despite social or occupational problems.

The *syndrome, of alcoholism* consists of two phases: problem drinking means very frequent use of alcohol, often to alleviate tension or solve other emotional problems. Alcohol addiction consists of physiologic dependence on alcohol as manifested by evidence of withdrawal symptoms when intake is interrupted. Continued drinking results in tolerance to effects of alcohol. Prolonged, regular alcohol intake results in liver and brain damage frequently. Alcohol drinking also results

in impairment of social and occupational life. Alcoholics are often simultaneously depedent on sleeping pills and other drugs. Alcoholics are often depressed. Majority of the people who commit suicide are alcoholics in the western countries.

Acute-intoxication by alcohol leads to drowsiness, lack of inhibition, slurring of speech, errors of commission, unstable gait and vomiting. Severe intoxication is marked by unconsciousness, low blood pressure, respiratory failure and death.

Alcohol withdrawal leads to anxiety, tremors, irritability, hyperreactivity and defective cognition. Severe alcohol withdrawal (delirium tremens) consists of mental confusion, tremors, visual hallucinations, sweating, dehydration, fits and low blood pressure. Delirium tremens culminate in death not too infrequently. It usually manifests 24-72 hrs after the last drink.

Alcoholic hallucinosis either during heavy drinking or on withdrawal. It consists of a major psychiatric disorder (psychosis) characterised by auditory hallucinations which may provoke the patient to behave aggressively.

Chronic drinking can also cause brain damage, manifested by erratic behaviour, loss of memory, emotional instability and defective vision.

Every alcoholic should be carefully screened for an underlying major psychiatric disorder which may be the cause of alcoholism.

Alcoholism also leads to heart disease (arrhythmias and heart failure) diseases of the nerves (neuropathy) liver failure (cirrhosis and jaundice). Alcoholism also predisposes to peptic ulcer and may precipitate bleeding from stomach. Children born of mothers who take alcohol during pregnancy are likely to have low birth weight, mental retardation and a variety of birth defects including abnormalities of the heart. Physical and mental growth of these children may also be impaired.

'Treatment of the problem of drinking is difficult but by no means impossible. Total abstinence (not controlled drinking) should be the primary goal. Psychological therapy should include the help of social agencies and religious counselling. The patient should be seen frequently by the doctor who is aware of his social and medical problems.

Alcoholics should not be placed in jobs which require working alone e.g. sales executive. Highly competitive posts should be avoided to minimize the stress and strain of daily life. Positions that require quick decision making on important matters should also be prohibited.

Hospitalisation is not indicated unless required due to other medical grounds. Cardiac, liver and neurological problems of alcoholics should be treated adequately.

Unpleasant effects due to alcoholism can be accentuated by intake of a drug called *disulfiram.* Intake of this drug may motivate a patient to stop drinking. This drug may have dangerous side effects and therefore should be taken only under strict medical supervision.

Alcoholic hallucinosis requires hospitalisation and care by an expert psychiatrist. Delirium tremers also require hospitalisatin, and sedation by sleeping pills till withdrawal syndrome disappears.

Alcoholics should be advised to take a balanced diet, rich in vitamin B complex and vitamin C to correct vitamin deficiency.

Drug dependency and drug abuse

Drug dependency means drug under compulsion and includes both drug addiction and habituation. Drug dependence may be due to one or more of the following:

(i) *Psychological depdence* i.e. psychological craving for the drug.

(ii) *Physiological (physical) dependence* i.e. occurance of withdrawal symptoms on discontinuation of the drug.

(iii) *Tolerance i.e.* the need to increase the does with continued use to obtain the same desired effects.

Drug dependency occurs due to frequent use of large amounts of drugs though some drugs produce dependence after single time use. Dependency increases with the passage of time. Polydrug usage is very common.

Opiates: The term opiates includes a number of drugs with actions that mimick those of morphine. The commonly abused opiates are (i) *Opium,* a crude preparation of the milky juice of poppy fruit (ii) *Morphine,* a refined product of the milky juice of poppy fruit. Morphine is used in treatment of several disease states (iii) *heroin,* a derivative of morphine which has no known medical use, (iv) *Purify Synthetic Opiates* like propoxyphene, pethidind and pentazocine and (v) others including *Codeine.* All these drugs share the common effects of damping of pain perception, feeling of drowsiness and euphoria. Tolerance to any of these drugs is likely to cause tolerance to other drugs of the same variety (i.e. cross tolerance). Each of these substance is capable of producing a psychological as well as physical (physiological) dependence and withdrawal syndrome similar to others but the frequency, amount and duration for which each drug should be taken before addiction occurs varies from drug to drug.

All the opiate drugs can be taken by mouth or by intravenous or intramuscular injections. Opium is taken by mouth. Morphine is taken by injection most often. Heroin can be taken by intravenous injection but most of the crude preparations of herion (e.g. smack/brown sugar) are inhaled as these drugs can be readily absorbed by lungs too. Street heroin typically contains only 5-10% of pure heroin, the remainder consists of contaminants such as fruit sugars, quinine, powdered mil, caffeine etc. which are used to cut the drug and increase the margin of profit.

Opiates cause the following effects on body systems; lack of appetite, constipation, hepatitis, nausea and vomiting, decreased pain perception, euphoria, drownsiness, decreased sexual drive, respiratory depression, low blood pressure, infection of the lungs and heart valves. Contaminants may cause permanent brain damage, loss of vision and brain abscess. A large number of intravenous drug abusers have required immuno-deficiency syndrome (AIDS) antibodies.

Administration of high doses accidentally or for suicidal purposes may cause severe respiratory depression, slow pulse, low blood pressure, decrease in body temperature, frothing through mouth and absence of responsiveness. Death may occur from cardiorespiratory arrest. Effects of opiates on body can be reversed by a drug called naloxone.

There are 2 categories of opiate abusers (i) medical abuser, who suffers from a chronic painful condition and misuses the prescribed drug (ii) street abuser. A significant proportion of street abusers have antisocial personality. However, the majority of street abusers belong to a class with high level of premorbid functioning. They start taking opiates occasionally, often after experimenting with tobacco, then alcohol and charas. The outcome is usually serious once opiate addiction is established. At least 25% of opiate addicts die within 10-20 years due to suicide, homicide, accidents, infections or over-dosage. A large number of opiate addicts also become alcoholics. Another group at high risk is that of physicians, nurses and pharmacists who have easy access to drugs.

Symptoms of opiate with withdrawal include nausea, diarrhoea, cough, excessive salivation, running nose, excessive sweating, goose pimples, muscle twitching, tremors, hot and cold flushes,

mild fewer, rapid respiration, high blood pressure, fast pulse, diffuse body pain, inability to sleep, yawning spontaneous ejaculation, orgasm and craving for the drug. Withdrwal symptoms disappear within 5-8 days but mild symptoms may persist upto 6 months.

Opiate withdrawal can be treated by gradual withdrawal of the drug over 5-10 days. Atternatively, a long acting opiate like methadone may be administered in tapering doses. Some symptoms of opiate withdrawal can also be eliminated by clonidine, a drug which does not belong to the class of opiates.

Rehabilitation of the opiate addict requires help by a physician as well as the family of the addict. The victim should be motivated towards abstinence by counselling arid education. Addicts often to conceal their dependence on drugs. Intravenous drug abusers can be readily recognised by multiple puncture marks over the forearm or wrist. Patients should be educated about their responsibility towards improving their lives and the potential consequences of continued addiction. Group therapy goes a long way in motivating drug addicts to shed off their dependence. In this type of treatment, several addicts sit together and discuss and narrate their problems to one another. Besides that an ex-addict describes his experiences and the way to deaddiction. Nearly one-third addicts become drug free within one year of such therapies. Another one-third do not use opiates anymore though they may continue to abuse milder drugs. Well educated addicts and those in job have a better prospect of getting rid of drug addiction. Persistent and heavy addiction is usually a difficult problem and may require hospitalisation for several weeks to months. Chronic administration of methadone may be necessary.

Marijunana (Charas, Hashish, Bhaog and Ganja): Cannabis saltiva, a plant, is the source of marijuana. Different parts of this plant vary in potency. The derivative of the flowering tops of the female plant (hashish, charas) is the most potent, followed by the dried leaves and flowering shoots of the female plant (bhang) and the resinous mass from small leaves of non-flowering shoots (ganja). The least potent parts are the lower branches and leaves of the female plant and all parts of the male plant. Hashish, charas and ganja are inhaled by smoking while bhang is taken by mouth. Effects occur within 10-20 mins and last for 2-3 hours.

The effects of marihuana are mild euphoria followed by sleepiness. In the acue state the user develops an altered time perception, less inhibited emotions, impaired immediate memory, redness of eyes and rapid heart rate. High doses produce transient alteration of psyche. Marijhuana frequently aggravates pre-existing mental illness and slows the learning process in children. Long term usage adversely affects the nose, larynx (voice box) and the lungs. Electrocardiogram (ECG) may become abnormal during marihuana intoxication but no long term heart disease is known to occur. Sperm counts may decrease. Abnormal menstruation and lack of ovulation is also known to occur. Marihuana abuse during pregnancy may cause impaired growth of foetus. Sudden withdrawal of marihuana produces lack of sleep, nausea, tremor, bodyache and irritability. The withdrawal symptoms are milder in comparison to those observed with heavy opiate or alcohol addicts and rarely require medical therapy. Tolerance to the effects of marihuana develops rapidly.

Other drugs: A large number of drugs are abused all over the world. The pattern of drug abuse varies in different countries and the pattern in a single country also keeps on changing with time. Herion was the most commonly abused drug in the western countries till early 1970s, when marihuana took over. In India, opium, charas and ganja have been the traditionally abused drugs since the ancient times. At present, crude preparations of herion including brown sugar and smack top the list at least in the urban areas. Charas, ganja and bhang continue to be popular among Indians. Cocaine *is the* most popular drug in America at present, but it is not commonly abused in India due to its/high cost.

Other drugs which are occasionallyjased by habitual drug users are sleeping pittls, L.S.D. (lysergic acid diethylamide), pheftcyclidine, amphetamines and antihistafninic drugs Sleeping pills can produce dependency and resemble alcohol in their behavioural manifestations. Acute intoxication with sleeping pills produces drownsiness, errors of commission, slowed speech and thinking, impaired memory, disinhibition and unstable gait. Respiratory depression, low blood pressure, unconsciousness and death may occur. Withdrawal symptoms include'restlessness, tremor, anxiety, fits weakness, palpitations and delirium. Overdosage with sleeping pills may be complicated by pneumonia and other serious complications, which may culminate in death. Treatment requires hospitalisation.

LSD, when ingested, produces a feeling of tension followed by emotional release (laughing or crying), perceptional distortions such as visual illusions and hallucinations, changes in time sense and mood lability. Its use during first trimestes of pregnancy commonly leads to abortion or birth defects. Treatment of LSD addiction includes treatment with antipsychotic drugs. Phencyclidine has simillar effects. It can be inhaled, injected, swallowed or smoked. Overdosage may be fatal due to severe elevation of blood pressure, respiratory depression or fits.

Amphetamines are abused because of the ability of these drugs to produce excessive physical activity and a sense of enhanced mental capacity. Acute intoxication causes excessive sweating, rapid pulse, high blood pressure, confusion and disorientation. Tolerance develops rapidly. Continued use may cause mental illness. Sudden withdrawal is characterised by depression and excessive sleeping and eating.

General treatment of drug addiction: Nearly all drug addicts require psycho-social care. This is a very time consuming process and should be done with the help of family members as well as a trained psychologist. For this purpose, all the major psychiatric departments of different hospitals run a regular 'de-addiction clinic' which aims at treating cases of addiction by psycho-therapy and drug treatment, whichever is appropriate. In some cases who are not likely to give up drugs at home, a brief period of hospitalisation may also be necessary in order to control the withdrawal symptoms which may prompt the person fcf resort to drugs again.

AIDS

AIDS, the acquired immuno-deficiency syndrome (sometimes called "slim disease") is a fatal illness caused by a retrovirus known as the human immuno-deficiency virus (HIV) which breaks down the body's immune system, leaving the victim vulnerable to a host of life-threatening opportunistic infections, neurological disorders, or unusual malignancies. Among the special features of HIV infection are that once infected, it is probable that a person will be infected for life. Strictly speaking, the terms AIDS can be called our modern pandemic, affecting both industrialized and developing countries.

Public Health Concern

Recognized as an emerging disease only in the early 1980s, AIDS has rapidly established itself throughout the world and it likely to endure and persist well into the 21st century.

The number of people living with HIV continues to grow, as does the number of deaths due to AIDS. A total of 39.5 million (34.1 million — 47.1 million estimated) people are living with HIV in 2006, about 2.6 million more than in 2004. This figure includes the estimated 4.3 million (3.6 million — 6.6 million) adults and children who were newly infected with HIV in 2006.

The interaction of HIV/AIDS with other infectious diseases is an increasing public health concern. Tuberculosis, bacterial infection and malaria have been identified as the leading cause

of HIV-related morbidity in Sub-Sharan Africa. HIV infection increases the incidence and severity of clinical malaria in adults.

In India, the early cases of HIV/AIDS were concentrated primarily to Chennai and Mumbai. The disease soon spread to other parts of the country, often following the path of major highways and labour migrants. By mid-2003, Tamil Nadu had nearly half of the reported AIDS cases. Mumbai and rest of Maharashtra now share about 19.74 per cent of the AIDS reported cases. Fig. 1 shows the distribution of reported AIDS cases by states upto August 2006.

Epidemiological Features

1. **Agent factors**

(a) **Agent:** When the virus was first identified it was called "lymphadenophathy—associated virus (LAV)" by the French scientists. Researchers in USA called it "human T-cell lymphotropic virus III (HTLV-iii)". In May 1986, the International Committee on the Taxonomy gave it a new name: Human Immuno-deficiency Virus (HIV).

The virus is 1/10,000th of millimeter in diameter. It is a protein capsule containing two short stands of genetic material (RNA) and enzymes. The virus replicates in actively dividing T_4 lymphocytes and like other retroviruses can remain in lymphoid cells in a latent state that can be activated. The virus has the unique ability to destroy human T_4 helper cells, a subset of the human T-lymphocytes. The virus is able to spread throughout the body. It can pass through the blood-brain barrier and can then destroy some brain cells.

(b) **Reservoir of Infection:** These are cases and carriers. Once a person is infected, the virus remains in the body life-long. The risk of developing AIDS increase with time. Since HIV infection can take years to manifest itself, the symptomless carrier can infect other people for years.

(c) **Source of Infection:** The virus has been found in greatest concentration in blood, semen and CSF. Lower concentrations have been detected in tears, saliva, breast milk, urine, and cervical and vaginal secretions. HIV has also been isolated in brain tissue lymph nodes, bone marrow cells and skin. To date, only blood and semen have been conclusively shown to transmit the virus.

2. **Host factors**

(a) **Age:** Most cases have occurred among sexually active persons aged 20-49 years. This group represents the most productive members of the society and those responsible for less than 3 per cent of the cases.

(b) **Sex:** In North America, Europe and Australia, about 51 per cent of cases are homosexual or bisexual men.

(c) **High Risk Groups:** Male homosexuals and bisexuals, heterosexual partners (including prostitutes), intravenous drug abusers, transfusion recipients of blood and blood products, haemophiliacs and clients of STD.

Mode of transmission

The causative virus is transmitted from person—to—person, most frequently through sexual activity. The basic modes of transmission are :

(a) **Sexual Transmission—**AIDS is first and foremost a sexually transmitted disease. In the USA, over 51 per cent of the cases were in homosexual or bisexual men. In contrast, in equatorial Africa, AIDS is acquired mainly through heterosexual contact. (infected man to woman; infected woman to man) from male to female is twice as likely as from female to male.

(b) **Blood contact—**AIDS is also transmitted by contaminated blood — transfusion of whole blood cells, platelets and factors VIII and IX derived from human plasma.

(c) **Maternal**—foetal transmission: mother-to-child transmission HIV may pass from an infected mother to her foetus, through the placenta or to her infant during delivery or by breast-feeding. In the absence of any intervention, rates of this form of transmission can vary from 15-30 percent without breast feeding.

Incubation period

While the natural history of HIV infection is not yet fully known, current data suggest that the incubation period is uncertain, (from a few months to 10 years or even more) from HIV infection to the development of AIDS. The virus can lie silent in the body for many years. The percentage of people infected with HIV, who will develop clinical disease remains uncertain—possibly 10-30 per cent will develop AIDS, and another 25-30 per cent will develop AIDS-related complex. However, it is estimated that 75 per cent of those infected with HIV will develop AIDS by the end of ten years.

Control of AIDS

Until a vaccine or cure for AIDS is found, the only means at present available is health education to enable people to make life—saving choices:

(1) Avoiding indiscriminate sex, using condoms. There is, however, no guarantee that the use of condoms will give full protection.

(2) One should also avoid the use of shared razors and toothbrushes.

(3) Intravenous drug users should be informed that the sharing of needles and syringes involves special risk.

(4) Women suffering from AIDS or who are at high risk of infection should avoid becoming pregnant, since infection can be transmitted to the unborn or newborn.

(5) Educational material and guidelines for prevention should be made widely available.

(6) All mass media channels should be involved in educating the people on AIDS.

National AIDS Control Programme

With the spread of AIDS from one country to another it became necessary to initiate a national control programme. The Govt. of India in 1985 constituted a task force to look into this matter. It began by pilot screening programme of high-risk population. National AIDS control Programme was launched in 1987. In the years 1992, the Ministry of Health and Family Welfare set up a National AIDS Control Organization as a separate wing to implement and closely

monitor the various components of the programme. The Government of India launched a 5 year HIV/AIDS Control Project from September 1992 to September 1997 as 100 per cent centrally sponsored project for all states/UTs. The project was later on extended upto March 1992.

The Phase II (1999-2006) of the National AIDS Control Programme has become effective from 9th November 1999. It is also a 100% centrally sponsored scheme implemented in 32 States/ UTs and 3 Municipal Corporation namely Ahmedabad, Chennai and Mumbai through AIDS control Societies. The three new states of Chhattisgarh, Uttaranchal and Jharkhand are in the process of establishing their State AIDS Control Societies.

The National AIDS Control Programme Phase II has two key objective:

1. To reduce the spread of HIV infection in India; and

2. To strengthen India's capacity to respond to HIV/AIDS on a long term basis.

1st Proof - 1.10.08 Through Bhattacharya Ji

2nd Proof - 23.10.08 Self (Bhattacharyas)

3rd Proof - 24.11.08 Self