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Review Article

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Incidence, Epidemiology and Prevention of Cancer and Management of Cancer Patients-an Overview

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Cancer is characterized by the malignant cellular growth that tends to spread. It is the second leading cause of death after heart disease. Cancer incidence varies with regions and populations around the world. The incidence of various types of cancer ranges from rare of gallbladder to 12% of most common cancer of lung. In men, the minimum incidence of cancer is that of gallbladder (4.4%) an the maximum is the incidence of lung cancer (18.6%). In women, the minimum incidence of cancer is that of endometrium (3.44%) and the maximum is that of breast cancer (18.18%). Dietary prevention of cancer is also a long term process and it may take 10 to 60 years to have an impact, the time scale depending, to some extent on the type of cancer. The epidemiological data suggest that the major causative factor for the incidence of cancer is environment. The environmental factors causing cancer include diet, radiation, chemicals and tobacco. The diet plays a major role in the development and prevention of cancer. Consumption of more meat, fatty foods of animal origin and salted foods increase the incidence of cancer. Method of preparation like grilling and barbecuing of meat also increase the incidence of cancer. Intake of foods of plant origin reduce the chance of cancer incidence.

Key words: Cancer, incidence, epidemiology, prevention, management

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Introduction

Cancer is a human disease and is defined as malignant tumor or a disease which is characterized by excess of cells beyond the number needed for normal function of the body. Cancer may grow slowly or rapidly. Many tumors grow into nearby organs or tissue, or spread to other parts of the body. This process is called metastasis. Metastasis usually occurs through the blood stream or lymph vessels. Benign tumors are made up of cells that are not cancerous; they usually do not grow and spread in the same way as malignant tumors. The benign tumors are not dangerous.

A combination of genetic, environmental and lifestyle factors are involved in turning a normal cell into an abnormal cell and an abnormal cell into cancer. In most cases, the cancer process starts when the genetic material responsible for cell division inside a cell becomes faulty. This may occur by chance or because a cancer causing substance called carcinogen has been introduced into, or produced by, the body.

Potential carcinogens are present in the air, food and water and enter the human body through breathing, eating and drinking. Other carcinogens are found in tobacco, industrial compounds and in the form of viruses. The human bodies are designed to deal with carcinogens either by eliminating the carcinogens before they do any harm to the body or repairing any damage done by them. Sometimes, the body's defenses fail and a carcinogen becomes activated inside a body cell and damages the genetic material of the cell permanently. Once the damage occurs, the cell can no longer function properly. The cell then may grow and multiply abnormally and as it divides, the flawed genetic material may be passed on. In full fledge cancer, the abnormal cells must reproduce to such an extent that they start to occupy the space of normal cells or threaten the function of healthy cells or organs.

Cancer may develop in any part of the body and is named after the name of that body part where it is developed. The most common types of cancer are mouth and pharynx, nasopharynx, Larynx, esophagus, lungs, stomach, colon and rectum, breast, ovary, endometrium, cervix, prostrate, pancreas, gall bladder, liver, bone marrow, intestine, thyroid, kidney, bladder, skin and uterine cancer etc. The rates of cancer incidence vary with factors such as age, sex, economical condition, migration and urbanization etc.

Between 30 and 40% of cancer cases throughout the world are preventable by feasible dietary means. Of course death cannot be prevented, so the main task for dietary prevention of cancer is, first, to reduce the occurrence of premature cancers greatly and, second, to seek to avert many of the cancers that are deferred to, or destined for, old age. Information about the effect of various diet constituents including energy and related factors, carbohydrates, fat and cholesterol, protein, vitamins (especially Vitamins A, C, D and E), vegetables and fruits, meat, poultry, fish and eggs, milk and dairy products, coffee, tea and other drinks, cereals (grains), pulses (legumes) and minerals are beneficial both for the patients and diet care professionals. The specific mechanism by which individual constituents of diet affect the cancer process are not fully understood and no doubt many remain to be identified. However, existing knowledge gives key insights into the cancer process, which, in turn, can lead to make better dietary guide lines for prevention of cancer. The nutritional needs of cancer patients changes during the

course of the disease and with different treatments so the nutritional care for cancer patients must be individualized and readjusted to meet the specific changing nutritional needs and dietary/eating peculiarities.

This review paper is to provide recent information to the researchers of the field and medical professionals and guide lines to the diet planners in hospitals. The review will help in educating the cancer patients.

Incidence of cancer

There is marked variation in the incidence of and mortality from, many cancers in different regions and populations around the world, but certain general patterns can be identified. With few exceptions, the economically developing countries of Africa, Latin America and Asia have relatively high rates of cancers of the upper aero digestive tract (mouth and pharynx, larynx and esophagus) and of the stomach, liver and cervix. In contrast, the economically developed countries of Europe, North America and Australia have relatively high rates of cancers of the colon and rectum and of the hormone-related cancers of the female breast, the endometrium and the prostate. This pattern has now also emerged in urban areas of developing countries. Lung cancer, mainly caused by use of tobacco, is now the most common cancer throughout the world (WCRF, 1997).

Cancer rates may change dramatically. For example, in most populations in the developed world, stomach cancer has been declining rapidly in recent decades, whereas rates of cancers of the colon, breast and prostate have been rising. Further variations in cancer patterns are seen in studies of migrants. Also, patterns of cancer are now changing rapidly within the economically developing world as populations age and become increasingly industrialized and urbanized. Such variations with time, migration and urbanization indicate that cancer rates are strongly influenced by environmental factors, including diets (WCRF, 1997).

It has been estimated that 10 million new cases and over 7 million deaths from cancer occurred in 1996 (WHO, 1997). Cancer is second to heart disease as a cause of death in westernized countries. In Britain, it accounts for 23% of all deaths and the risk of it increases with age. Rate of stomach cancer in men and women are declining and rate of skin and bladder cancer are increasing. In women, the rates of incidence of breast and lung cancers are increasing. In men rates for cancer of the prostate, colon and esophagus are increasing (Thomas, 1994). Approximately 150 diseases caused by abnormal cells that develop and divide uncontrollably. The high risk countries for incidence of cancer are North America, Europe, Australia while low risk countries are Central and South America, Asia and Africa. The incidence of various types of cancer has been reported by the World Health Organization (WHO, 1997) as under.

Gastro-intestinal cancer

The incidence of mouth and pharyngeal cancer is relatively high in the developing world, notably in India and elsewhere in Asia and is more common in men. This cancer is the fifth most common incident cancer. Nasopharyngeal carcinoma is a rare cancer in most parts of the world.

The age standardized incidence rate for either sex is generally less than 1 per 100,000 population. Cancer of the larynx is the fourteenth most common cancer in the world. Approximately 60% of the global incidence of laryngeal cancer is found in developing countries. Men account for 85% of the worldwide incidence of laryngeal cancer. Cancer of the esophagus is the eighth most common cancer in the world. Esophageal cancer incidence is higher among males than females.

Cancer of the stomach is the second most common cancer .in the world and is decreasing throughout the world. Stomach cancer rates are 50% lower in women than in men (Aoki et al, 1992, Parkin et al, 1992, WHO, 1997). Incidence rates of colorectal cancer have generally increased. Cancer of colon and rectum is the forth most common cancer in the world. Central and South America, Asia and Africa are areas of low risk. In both men and women, incidence of colon cancer occurs at a similar rate. However, cancer of the rectum is more common in men than women, with a 20-50% higher incidence (WHO, 1997).

Lung cancer

Lung cancer have increased throughout the world with sharp increase for women almost everywhere. Cancer of the lung is the most common cancer in the world. In 1996, an estimated 1,320,000 new cases were diagnosed worldwide, accounting for 12.8% of all new cancers (WHO, 1997). Rates of lung cancer are highest in North America and Europe and lowest in Africa, Asia and South America (Parkin ***et al.***, 1992).

Liver cancer

Cancer of the liver is the sixth most common cancer in the world. In 1996, an estimated 540,000 new cases were diagnosed worldwide, accounting for 5.2% of all new cancers. In the developing world, liver cancer is very common, accounting for more than 80% of the global cases. China alone accounts for 55% of the worldwide incidence. Incidence among men is over twice that among women. Infection with hepatitis B virus is the predominant cause of liver cancer, with more than 80% of cases being the result of infection with this virus. (WHO, 1997).

Breast cancer

The incidence of breast cancer have also increased overall. It is low in developing countries and high in developed countries. Cancer of the breast is the third most common cancer in the world. In 1996, there were an estimated 910,00 new cases diagnosed worldwide, accounting for 9% of all new cancers. Over 50% of breast cancer incidence (494,000 cases) occurred in the developed world in 1996. The lowest rates are reported in Africa and Asia. However, breast cancer is increasing in many countries, particularly in areas which have previously had low rates, although at least part of the increase is due to early detection of cases by screening (WHO, 1997; Doll ***et al.***, 1994).

Glands cancer

Cancer of the pancreas is the thirteenth most common cancer in the world. Men have

approximately a one third greater age adjusted incidence rate than women (Parkin *et al.*, 1993). Most gallbladder cancers arise in the epithelium and are classified as carcinomas. The incidence of this cancer is low compared with other sites, even among those populations who are at highest risk and accounts for about 1% of all cancer deaths. In high risk populations, incidence in women is approximately double that of men but, in other populations, the rates are similar for men and women (Parkin *et al.*, 1992). Cancer of the prostate is the ninth most common cancer in the world. The rate of this male hormone related cancer is generally low in developing world and high in the developed world. In 1996, there were an estimated 400,000 new cases diagnosed worldwide, accounting for 3.9% of all new cancers (WHO, 1997). Thyroid cancers are relatively rare, representing fewer than 2% of all recorded neoplasm's worldwide (Goodman *et al.*, 1988; Omran and Ahmed, 1993 and WHO, 1997). In patients with a history of non melanoma skin cancer, a low fat diet reduces the actinic keratosis (Black *et al.*, 1994).

Ovarian, endometrial and cervical cancers

Cancer of the ovary is the fifteenth most common cancer in the world. Cancer of the endometrium is the sixteenth most common cancer in the world. Africa and Asia have the lowest incidence of endometrial cancer. Cancer of the cervix is the seventh most common cancer in the world. The highest incidence rates are seen in parts of Sub-Saharan Africa, South East Asia and Latin America. Developing countries account for 80% cases of the cervical cancer (WHO, 1997).

Kidney and bladder cancers

Cancer of the kidney is the seventeenth most common cancer in the world. Worldwide, there is at least a ten fold difference in the incidence of renal cancer. The rates are lowest in Asia and South America (Parkin *et al.*, 1992). Cancer of the bladder is the eleventh most common cancer in the world. Incident of bladder cancer is two to five times higher in men than in women (Parkin *et al.*, 1992; Silverman *et al.*, 1992). In 1996, approximately 75% of cases were male (WHO, 1997). In tropical and subtropical countries, where schistosoma haematobium is endemic, bladder cancer is the most common cancer in men and second only to breast cancer in women, accounting for about 30% of all cancers (Badawi *et al.*, 1995). The predominant histological type of bladder cancer is transitional epithelial cell carcinoma (Silverman et al, 1992). However, squamous cell carcinoma is the major type in areas where urinary schistosoma infestation is prevalent (Badawi *et al.*, 1995).

The rates of incidence of the various types of cancer are shown in the decreasing order in Table 1.

Salam *et al.* (1992) conducted two studies in Peshawar and one study in D.I. Khan on the incidence of various types of cancer. They showed strong correlation among the frequency of various malignant tumors in Peshawar and D. I .Khan. Their data is presented in Tables 5 and 6.

Epidemiology of cancer

Knowledge of the causes of human cancer comes primarily from epidemiological studies which compare population groups or categories of individuals who share some common

Table 1: Incidence of various types of cancer in decreasing order

Name of cancer	Percent prevalence
Lung Cancer	12.80
Stomach Cancer	10.00
Breast Cancer	9.00
Colorectal Cancer	8.50
Mouth and Pharynx	5.60
Liver Cancer	5.2
Cervical Cancer	5.00
Esophageal Cancer	4.60
Prostate Cancer	3.90
Bladder Cancer	3.00
Pancreatic Cancer	2.00
Laryngeal Cancer	1.80
Ovarian Cancer	1.80
Endometrial Cancer	1.60
Kidney Cancer	1.60
Thyroid Cancer	1.00 -2.00%
Nasopharyngeal Cancer	less than 1%
Gall Bladder Cancer	Rare

Source: WHO (1997).The incidence of cancer in various regions and population of the world are summarized in Table 2.

Table 2: Incidence of cancer in the different regions or population of the world

		Esophagus		Lung		Stomach		Colon/Rectum			
Country	Region or population	Men	Women	Men	Women	Men	Women	Men	Women	Breast women	Prostate men
Asia											
Japan	Osaka	8.4	1.8	41.5	11.7	73.6	32.7	26.5	16.4	21.9	6.6
China	Shanghai	14.9	6.4	53.0	18.1	51.7	21.9	17.8	15.6	21.2	1.7
Hong Kong	All	18.1	3.6	78.7	32.6	22.1	11.2	35.5	26.0	32.3	7.6
Singapore	Chinese	10.9	2.7	69.7	21.9	34.7	15.6	35.4	28.6	31.6	7.6
	Malay	1.2	0.9	34.0	12.1	6.4	5.4	15.1	12.1	23.2	9.0
	Indian	3.2	3.4	20.7	5.2	15.9	7.5	15.8	16.9	34.0	11.0
Thailand	Chiang Mai	4.1	2.7	40.5	29.5	11.6	6.0	9.9	7.7	13.7	4.0
Philippines	Manila	3.1	2.3	53.4	16.3	13.5	8.1	18.7	15.0	49.7	16.9
India	Bombay	11.4	8.4	14.0	3.0	7.3	4.3	6.4	5.1	24.6	6.9
	Madras	7.6	6.3	8.5	1.4	15.1	6.7	3.9	3.4	19.9	2.1
Israel	All Jews	1.4	1.1	28.3	9.6	14.6	7.5	35.9	30.5	64.7	17.5
Kuwait	Kuwaitis	3.7	1.7	14.5	4.8	4.1	2.0	4.3	4.5	17.2	4.4
	Non-Kuwaitis	1.9	1.9	44.9	12.7	14.4	5.9	7.3	9.3	35.6	10.5
Caribbean, Centeral and South America											
Martinique	All	13.7	2.5	11.0	3.0	24.9	10.6	9.2	8.8	28.2	48.2
Cuba	All	5.2	1.7	44.3	15.7	9.8	5.0	13.7	14.6	35.0	27.3
Costa Rica	All	3.8	1.2	12.7	4.7	46.9	21.3	9.1	9.6	26.7	23.7
Colombia	Cali	3.6	1.6	24.6	9.8	36.3	19.9	8.0	9.2	34.8	26.1
Brazil	Goiania	9.8	2.6	26.0	11.6	28.2	14.9	13.4	12.7	40.5	29.0
Ecuador	Quito	4.4	0.5	8.3	3.9	29.5	22.7	8.7	8.4	26.2	23.0
Paraguay	Asuncion	11.2	1.7	18.2	3.6	14.4	5.8	7.3	10.6	36.3	22.0

Table 2: Continue

Country	Region or population	Esophagus		Lung		Stomach		Colon/Rectum		Breast	Prostate
		Men	Women	Men	Women	Men	Women	Men	Women	women	men
Peru	Trujillo	1.0	0.6	9.5	4.2	28.9	26.4	6.0	9.0	28.3	19.9
Oceania											
Fiji	Melanesian	1.8	-	5.5	1.4	7.7	2.0	7.3	5.1	20.1	5.0
New Caledonia	Melanesian	12.5	-	34.3	14.2	15.6	6.8	7.9	2.3	16.6	3.7
CookIslands/Niue	Polynesian	10.6	-	71.1	4.7	24.0	14.6	15.9	8.7	45.3	15.9
New Zealand	Polynesian	6.1	0.7	110.4	80.7	33.7	20.3	23.1	18.9	61.4	36.5
Hawaii	Polynesian	11.2	1.3	94.6	28.6	40.2	17.8	42.1	19.3	95.0	40.8
Europe and the Newly-Independent States											
UK	Birmingham	5.5	3.6	75.0	18.8	22.3	8.7	38.0	25.4	63.4	25.0
France	Somme	19.3	1.0	64.6	3.9	14.5	6.5	35.6	21.9	55.4	28.6
Netherlands	Maastricht	3.7	0.9	83.4	9.1	16.6	6.3	36.0	27.4	68.1	29.6
Germany	Saarland	6.1	0.8	69.6	7.3	20.4	11.5	40.5	29.4	56.3	28.9
Switzerland	Vaud	8.0	2.4	58.2	9.8	11.5	4.4	32.2	23.1	70.8	34.2
Italy	Florence	3.1	0.8	64.2	8.9	40.2	19.1	38.7	27.8	65.4	22.0
Spain	Zaragoza	4.6	0.4	42.2	3.6	20.9	9.4	203	14.2	39.5	17.6
Norway	All	2.6	0.8	33.9	9.7	15.7	8.0	35.6	28.2	54.8	43.8
Sweden	All	3.2	0.9	25.2	9.5	12.7	6.5	29.4	24.2	62.5	50.2
Denmark	All	3.9	1.3	58.5	23.1	12.5	5.7	37.6	30.1	68.6	29.9
Iceland	All	4.0	2.2	35.4	25.9	28.8	9.9	27.7	20.9	69.7	52.4
Finland	All	3.3	2.2	65.8	7.6	20.3	11.2	22.1	17.3	52.5	36.1
Poland	Warsaw	5.2	0.9	65.2	16.4	21.5	8.6	21.2	16.1	38.7	11.9
Slovakia	All	5.6	0.5	79.1	7.8	27.1	12.2	34.2	20.5	34.5	19.9
Hungary	Szabolcs	5.7	0.5	69.6	9.1	26.4	9.3	20.8	16.6	29.6	14.3
Romania	Cty Cluj	1.9	0.4	36.9	6.1	26.1	10.7	15.0	10.4	31.1	9.9
Belarus	All	4.4	0.5	55.6	5.3	46.7	20.1	17.9	13.3	24.7	9.0
Latvia	All	4.3	0.6	63.2	7.3	34.1	15.5	19.4	14.7	32.1	15.3
Estonia	All	5.0	0.7	67.0	7.7	37.0	18.6	23.7	18.3	33.9	18.8
Russia	St Petersburg	11.1	3.7	77.6	9.1	52.8	25.3	34.2	25.9	40.6	13.9
North America											
Canada	All	4.2	1.3	68.5	23.9	12.4	5.4	44.4	33.7	71.1	51.4
USA	Seer										
	White	4.0	1.3	64.3	29.9	8.0	3.5	46.5	33.2	89.2	61.8
	Black	13.9	3.6	90.0	28.1	12.4	5.6	38.6	32.3	65.0	82.0
USA	Los Angeles										
	Japanese	5.7	0.1	34.9	17.5	29.7	13.8	54.5	39.5	72.7	32.9
	Chinese	2.9	0.8	42.6	18.2	13.0	7.9	36.0	23.5	48.7	19.8
	Filipino	0.6	0.7	30.8	10.8	4.0	3.7	22.9	15.6	52.2	28.6
	Korean	2.8	0.5	38.3	12.4	41.5	22.9	12.3	12.4	16.9	8.9

Source: WHO (1997). The world wide incidence and mortality rates from different types of cancers in men and women are shown in Tables 3 and 4.

characteristics. The principal causes of most cancers are environmental in origin and diet may be a major environmental factor (NAS, 1982). One estimate of the proportion of all cancers attributable to diet ranges from 10-70%, with an overall average of 35%. This compares with 25-40% from tobacco and 2-8% from occupational exposure. Food additives contribute less than 1%,

Table 3: estimated number of new cases and deaths from cancer worldwide in 1996 (men)

Cancer site	New cases (Thousands)	Percent of total	Deaths (Thousands)	Percent of total
Lung	988	18.60	878	22.40
Stomach	634	11.94	518	13.22
Colon, rectum	445	8.38	257	6.56
Prostrate	400	7.53	204	5.21
Mouth & Pharynx	384	7.23	237	6.05
Liver	374	7.04	370	9.44
Oesophagus	320	6.02	305	7.78
Bladder	236	4.44	107	2.73
Other	1531	28.82	1043	26.61
Total	5,312	100.00	3,919	100.00

Source: WHO (1997).

Table 4: Estimated number of new cases and deaths from cancer worldwide 1996 (women)

Cancer Site	New Cases (Thousands)	Percent of Total	Deaths (Thousands)	Percent of Total
Breast	910	18.18	390	12.22
Cervix	524	10.47	241	7.55
Colon, rectum	431	8.61	253	7.93
Stomach	379	7.57	317	9.93
Lung	333	6.65	282	8.83
Mouth & Pharynx	192	3.84	129	4.04
Ovary	191	3.82	125	3.92
Endometrium	172	3.44	68	2.13
Other	1874	37.44	1387	43.45
TOTAL	5,006	100.00	3,192	100.00

Source: WHO (1997).

Table 5: Five commonest tumors in males in Peshawar and D.I.Khan

Site of cancer	Peshawar study-I	Peshawar Study-II	D.I. Khan Study
Lymph node	14.5	15.6	6.4
Skin	16.8	14.9	12.
Oral Cavity	6.8	7.4	-
Esophagus	6.1	6.3	-
Larynx	5.7	4.8	-
Prostate	-	-	6.4
Liver	-	-	4.3
Connective tissue	-	-	2.8

Table 6: Five commonest tumors in females

Site of Cancer.	Peshawar Study-I	Peshawar Study-II	D.I. Khan Study
Breast	18.8	17.8	5.7
Skin	12.8	17.5	4.9
Esophagus	5.4	5.7	3.5
Liver	-	-	4.3
Lymph nodes	7.4	7.1	-
Oral cavity	6.7	3.8	-
Connective tissue	-	-	3.5

Table 7: Dietary factors implicated in the promotion of/or protection against cancer

Possible initiators and promoters of cancer	Fat, cholesterol, obesity, protein and meat, sodium (salt), nitrate and nitrite, N-nitroso compounds, mycotoxins, other naturally occurring carcinogens & contaminants and alcohol.
Possible protective constituents of food against cancer	Non-starch polysaccharides (dietary fiber) and starch, vitamin A and carotene, vitamins C, E, D, selenium, calcium and other minerals and vitamins fruit and vegetables

but virtually this is no risk. Some food additives may protect from cancer (Doll and Peto, 1981). The international correlation between the meat intake and the incidence of colon cancer has been measured as 0.85 for men and 0.89 for women (Armstrong and Doll, 1975). Major changes in the rates of a disease within a population over time provide evidence that non genetic factors play an important role in that disease. In Japan, for example, the rates of colon cancer has risen dramatically since 1950 (Aoki ***et al.***, 1992).

Role of diet in cancer

Cancer development is thought to be a several stage process of initiation, followed by promotion. The initiation step is brought about by carcinogens which are widespread in the environment and which cause changes in the DNA of cells. Before malignancy develops, another stage, promotion is necessary. In general, diet is thought to be particularly important in the promotion of cancer and in protection against the effects of carcinogens, rather than as a carrier of carcinogens themselves. It is to be expected that the causes of cancers in different organs of the body would be different, but the way in which diet is thought to be involved is often uncertain. In addition, several sites have a dietary constituent in common. Those that have been implicated in either promotion of or protection against cancer are shown in Table 7 (NRC. 1982).

Changes in diet could prevent upto half of all cases of breast cancer, three out of four cases of stomach cancer and three out of four cases of colon and rectal cancer (WCRF, 1994). Epidemiological evidence suggests that a diet rich in fruits and vegetables may reduce the risk of various forms of cancer, most likely contributable to their vitamins and minerals content. Recent intervention studies from population of Linxian, China have indicated that vitamins and minerals supplementation, specifically beta carotene, vitamin E and selenium, may play a role in the reduction of cancer risk (Mobarhan, 1994).

Effect of milk and milk products on cancer

Diets high in milk and dairy products possibly increase the risk of prostate and renal cancer (Armstrong and Doll, 1975; Role ***et al.***, 1986; Maclure and Willett, 1990).

Effect of egg on cancer

Diets high in eggs may increase the risk of pancreatic cancer, but is, as yet, insufficient (Zheng ***et al.***, 1993). Consumption of eggs possibly increases risk of colorectal cancer (Hiramatsue

et al., 1983). Diets high in eggs may increase the risk of ovarian cancer but is not proven yet (Snowdon, 1985).

Effect of meat and meat products on cancer

Eating more than 80 g (3 ounces) of red meat, that is beef, lamb, or the products made from these meats, probably increase the risk of cancers of the colon and rectum and may also increase the risk of developing cancers of the pancreas and kidney, as well as prostate and breast cancer. Red and processed meat is a major source of animal fat which possibly increases the risk of cancers of the lung, colon and rectum, breast, edometrium and prostate. Red meat should contribute less than one tenth or 10% of the total calorie intake. Three ounces or less is roughly equivalent to 2-3 slices of beef or lamb. White meat is the better choice if meat has to be eaten. Salted fish increases the risk of nasopharyngeal cancer, especially when eaten frequently in early childhood (HO, 1971; Huang *et al.*, 1978). Diets high in cured meats may increase the risk of stomach cancer, but yet to be investigated more (Boeing *et al.*, 1991). Diets high in meat and fish that has been grilled (broiled) and barbecued possibly increase the risk of stomach cancer (Hansson *et al.*, 1993). Diets high in meat possibly increase the risk of pancreatic cancer, perhaps particularly at the higher levels and with certain preparation methods commonly used in Western societies (Zheng *et al.*, 1993). Diets high in cured or smoked meat or fish may increase the risk of pancreatic cancer, but is, as yet, insufficient (Anderson *et al.*, 1996). Red meat probably increases the risk and processed meat possibly increases risk of colorectal cancer (Steinmetz and Potter, 1993; Gerhardsson de Verdier *et al.*, 1991). Diets high in meat possibly increase the risk of breast cancer (Toniolo *et al.*, 1994); Diets high in fish may decrease the risk of breast cancer; (Rose *et al.*, 1994) and ovarian cancer (Mori *et al.*, 1988). High meat diets possibly increases the risk of prostate cancer and renal cancer. Diets high in animal protein may increase the risk of breast cancer, but is, as yet insufficient (Armstrong and Doll, 1975).

Effect of cereals on cancer

Carbohydrates provide about half the calories in our diets. In nature, foods rich in starches and non-starch polysaccharides such as wholegrain bread and cereals, pulses and tubers (potatoes), contain valuable amounts of vitamins, minerals and dietary fibre - all of which are important for good health and for preventing cancer. The starch in foods is thought to protect against cancers of the colon and rectum. Processing can destroy nutrients and other food components that are important in fighting cancer. That is why wholegrain bread and brown rice are generally better than their white counterparts. A diet containing a large amount of refined sugar may increase the risk of cancers of the colon and rectum. There is no evidence that the naturally occurring sugars found in whole fruits affect cancer risk so, grapes, bananas, peaches and strawberries should be preferred than foods made with refined sugar like cakes, pastries, biscuits and chocolate. Diets high in cereals possibly increase the risk of esophageal cancer. However, any relationship between cereal consumption and esophageal cancer is probably not with cereals as such, but with diets that are deficient in a number of protective micro nutrients or contaminated with mycotoxins or both (Thurnham *et al.*, 1985). Deits high in starch possibly

increase the risk of stomach caner. It is likely that this applies to starch in refined form (Ramon *et al.*, 1993b; Hansson *et al.*, 1993). Diets high in extrinsic (refined) sugars possibly increase risk of colorectal cancer. The evidence is strongest for sucrose (Luceri *et al.*, 1996a).

Effect of fiber on cancer

Diets high in fiber may decrease the risk of stomach cancer, but not yet proven ((Hansson *et al.*, 1993). Diets high in NSP/dietary fiber possibly decrease the risk of pancreatic cancer (Howe and Bruch, 1996). Diets high in fiber possibly decrease the risk of colorectal cancer (Ma *et al.*, 1996). NSP/dietary fiber possibly decreases the risk of breast cancer (Cohen *et al.*, 1991). The importance of dietary habits for colorectal cancers and adenomas was studied in Denmark. It was concluded that the intake of crude dietary fibers is associated with reduce risk of cancer as well as adenomas (Olsen *et al.*, 1994).

Effect of fruits and vegetables on cancer

Eating 400-800 g (15-30 ounces), of vegetables and fruits daily could decrease the cancer risk upto 20%. Eating plenty of vegetables and fruits protects against many cancers, particularly those of the mouth, pharynx, esophagus, lung, stomach, colon and rectum. Vegetable and Fruits also reduce the risk of those cancers that affect the larynx, pancreas, breast and bladder. It possibly lower the risk of cancers of the liver, ovary, endometrium, cervix, prostate, thyroid and kidney too. The protective effects of vegetables and fruits is due to complex interactions between vitamins, minerals, fibre and plant chemicals. Diets high in vegetables and fruits decrease the risk of cancer of the mouth and pharynx, esophageal and lung cancer (Gridley *et al.*, 1992; Zheng *et al.*, 1992; De Stefani *et al.*, 1990; Steinmetz and Potter, 1991 and McPhillips *et al.*, 1994). Also diets high in vegetables and fruits decrease the risk of lung cancer is convincing (McPhillips *et al.*, 1994). Diets high in vegetables and fruits, collectively and separately decrease the risk of stomach cancer. Raw vegetables, alliums vegetables and citrus fruits are particularly abundant and consistent for a protective effect. Any evidence not consistent with a protective effect relates almost entirely to salted and pickled vegetables. (Fontham *et al.*, 1986; Kneller *et al.*, 1992 and Kato *et al.*, 1990).

There is substantial evidence from epidemiological studies that diets high in vegetables and fruits probably decrease the risk of pancreatic cancer (Stemmetz and Potter, 1991). Diets high in vegetables and fruits probably decrease the risk of breast and bladder cancer (Ingram *et al.*, 1992 and Bruemmer *et al.*, 1996); and possibly decrease the risk of ovarian, Endometrial and thyroid cancer (Role *et al.*, 1986; Levi *et al.*, 1993 and Franceschi *et al.*, 1989). A population based case control study was conducted in Maxico City during 1989-1990 to evaluate the relation between chili pepper consumption and gastric cancer risk. Results indicated that chilli pepper consumers were at high risk for gastric cancer compared with non consumers (Lopez- Carrilo *et al.*, 1994).

Effect of carotenoid and vitamins on cancer

High dietary carotenoids intake possibly decreases the risk of esophageal, stomach,

colorectal, breast, cervical and lung cancers (Hu *et al.*, 1994; Singh and Gaby, 1992; Zaridze *et al.*, 1993; Jan *et al.*, 1994; Harris *et al.*, 1986 and Steinmetz *et al.*, 1993). The evidence suggests that high dietary carotenoids may decrease the risk of ovarian, endometrial and balder cancer but is yet to be proved (Engle *et al.*, 1991; Vecchia *et al.*, 1986; Moon and Mehta, 1986 and Vena *et al.*, 1992). High dietary retinol intake possibly has no relationship with the risk of stomach cancer (Haenszel *et al.*, 1985). High dietary vitamin E intake possibly decreases the risk of lung and cervical cancer (Mayne *et al.*, 1994; Knekt, 1988). Evidence on diets high in vitamin E and the risk of colorectal cancer is relatively null but higher pre diagnostic blood levels are associated with a lower risk of subsequent cancer. The evidence suggests that higher dietary vitamin E may reduce the risk of colorectal cancer, but is, as yet, insufficient (Longnecker *et al.*, 1992). Vitamin E and A were negatively associated with adenoma occurrence (Olsen *et al.*, 1994). Vitamin D and higher intakes of both folate and methionine may reduce the risk of colorectal cancer but is at present insufficient (Lointier *et al.*, 1987; Giovannucci *et al.*, 1993). High dietary vitamin C intake possibly decrease the risk of cancer of the mouth and pharynx (Rossing *et al.*, 1989; Gridley *et al.*, 1990; Ramaswamy *et al.*, 1996; Zheng *et al.*, 1992). Also, high dietary vitamin C intake

possibly decreases the risk of esophageal, lung, stomach, pancreatic and cervical cancer (Chen *et al.*, 1992; Steminmetz *et al.*, 1993; Singh and Gaby, 1992; Zhang *et al.*, 1994; Appel *et al.*, 1991 and Van Enwyk *et al.*, 1992). High dietary vitamin C intake may reduce the risk of colorectal cancer, but the evidence is, at present, insufficient (Dion *et al.*, 1982). High dietary vitamin C may decrease the risk of breast cancer (Jain *et al.*, 1994) and bladder cancer but yet to be proven (Steinmetz and Potter, 1991).

Effect of minerals on cancer

Epidemiological and experimental data and plausible biological pathways, amount to evidence that diets high in salted foods probably increase the risk of stomach cancer (Kneller *et al.*, 1992). Daily consumption of salt should be limited to less than 6 grams (or 0.2 g of sodium)- that is roughly equivalent to only a teaspoon of salt a day - from all sources. Evidence suggests that iron intake may increase the risk of colorectal caner but is, as yet, insufficient (Nelson *et al.*, 1994) and also iron overload with diverse aetiologies may increase the risk of primary liver cancer. However this is to be proven yet (Hann *et al.*, 1992). High dietary selenium intake possibly decrease the risk of lung and stomach cancer (El-Bayoumy et al, 1993; Sivam *et al.*, 1997) and may decrease the risk of stomach, liver and thyroid cancer, but is, as yet, insufficient (Kobayashi *et al.*, 1986; Han, 1993 and Knizhnikov *et al.*, 1993). Diets deficient in iodine or with an excessive intake of iodine probably increase the risk of thyroid cancer (Wigren *et al.*, 1992; Kanno *et al.*, 1992). The chlorinated drinking water may increase the risk of bladder cancer though not proven yet (Zierler *et al.*, 1986).

Effect of fat on cancer

High fat diets increase the number of tumors initiated by a variety of chemical carcinogens in virtually all animal models of breast, bowel, pancreas and prostate cancer provided that the diets fed supply sufficient of the essential n-6 poly-unsaturated, linoleic acid, for which there is a tumor requirement of approximately 10% total energy. Lowering the total amount and kind

of fat is important for reducing cancer risk. Saturated fat possibly increase the risk of developing cancers of the lung, colon, rectum, breast, prostate and endometrium. Healthy eating involves reducing all fat consumption to less than a third or 30% of one's total calorie intake. It is best to avoid fats of animal origin, hydrogenated oils, palm and coconut oils. Diets high in total fat possibly increase the risk of lung, colorectal, breast and prostate cancers. Effect of fat is minor compared to that of cigarette smoking (Knekt ***et al.***, 1991b; Alavanja ***et al.***, 1993; Guillem and Weinstein, 1990; WCRF, Expert Pannel, 1994; Armstrong and Doll, 1975; Rose ***et al.***, 1986). As a whole diets high in total fat may increase the risk of ovarian and endometrial cancer but is, as yet, insufficient (Armstrong and Doll, 1975; Rose ***et al.***, 1986). Diet high in total fat or consumption of fried foods may increase the risk of bladder cancer, but is, as yet, insufficient (Bruemmer ***et al.***, 1996; Wakabayashi ***et al.***, 1992).

Effect of cholesterol on cancer

The evidence relating cholesterol to cancer risk is conflicting. In a report of 1500 individuals in Scotland for example, there was a strong negative association in men between serum cholesterol and lung cancer but there was not such association in the few patients who developed colon cancer, nor in women who developed breast cancer (Isles ***et al.***, 1990). A number of studies have reported that a low serum cholesterol is associated with increased risk of cancer. But some of these studies were small and the increased risk was usually shown for lung cancer which is primarily related to cigarette smoking. The evidence regarding dietary cholesterol and lung cancer is somewhat mixed. One of four cohort studies and three of five case control studies have shown moderate to strong increases in risk with higher intakes and no studies have shown a statistically significant decrease in risk. Diets high in cholesterol possibly increase the risk of lung and pancreatic cancer (McMichael ***et al.***, 1984; Howe ***et al.***, 1992). Diets high in cholesterol may increase the risk of endometrial caner but is, as yet, insufficient (Goodman ***et al.***, 1994).

Effect of tea on cancer

High consumption of coffee and black tea probably has no relationship with the risk of stomach cancer (Hansson ***et al.***, 1993; Heilbrum ***et al.***, 1986 and IARC, 1991). However, high consumption of green tea possibly decreases the risk of stomach cancer (Yamane ***et al.***, 1995). Coffee may decrease the risk of colorectal cancer, but not yet proven (Jacobsen and Thelle, 1987). Also, high consumption of coffee possibly increase the risk of bladder cancer at high levels of intake, but is probably not associated with risk at consumption below 5 cups per day (Avanzo ***et al.***, 1992).

Effect of alcohol on cancer

Drinking alcohol regularly increases the risk of cancers of the mouth, pharynx, larynx, esophagus and primary cancer of the liver. Furthermore, the risk is even greater for people who drink and smoke. Drinking alcohol probably also increases the risk of developing cancers of the colon, rectum and breast. High alcohol consumption, irrespective of the type of beverage,

increases the risk of laryngeal and mouth & pharynx cancer (Hedberg *et al.*, 1994; Keller and Terris, 1965). High alcohol consumption also increases the risk of oesophageal cancer (Blot, 1992; Yamada *et al.*, 1992). High alcohol intake possibly increases the risk of cancer of the gastric cardia (Vaughan *et al.*, 1995) and also increases the risk of lung cancer but any impact of alcohol consumption is minor compared with that of cigarette smoking (Potter *et al.*, 1982).

Heavy and persistent drinking of alcohol, leading to cirrhosis and alcoholic hepatitis which increase the risk of primary liver cancer (Austin, 1991) but has no relationship with the risk of pancreatic cancer (Lowenfels *et al.*, 1993). High alcohol consumption probably increases the risk of breast cancer (Longnecker, 1994) and cancers of colon and rectum. The effect generally seems to be related to total ethanol intake, irrespective of the type of drink (Garro and Lieber, 1990). The association between alcohol and colon and rectal neoplasms were examined in the Iowa Women's Health Study (USA). Results showed that alcohol intake was inversely associated with distal colon neoplasm, however, no association was observed with proximate colon neoplasm (Gapsture *et al.*, 1994).

Effect of obesity on cancer

Animal studies which suggest an effect of fat in the promotion of cancer can largely be explained by the fact that over feeding had a definite and marked effect on the progression of cancers of the large bowel, breast and probably pancreas. However, a marked energy restriction of approximately 20% is needed to overcome the effect of fat in animal studies. There is no evidence that this level of energy restriction alters cancer risk in humans. Risks of mortality from, or development of, post menopausal breast cancer and cancer of the endometrium are probably increased with increasing obesity, but may be decreased in pre-menopausal breast cancer (Willett *et al.*, 1987). Diets high in energy possibly increase the risk of pancreatic, gallbladder and colon cancer (Pariza, 1987; Zatonski *et al.*, 1992; Giovannucci *et al.*, 1995). High energy intake may increase the risk of prostate cancer, but is, as yet, insufficient (Rohan *et al.*, 1995). Higher body mass and adult weight gain probably increases the risk of breast cancer after menopause (Pike, 1990; Radimer *et al.*, 1993).

Effect of additives, contaminants and residues on cancer

Contamination of foods with chemicals and other substances is the major problem in Pakistan. Chemical residues may be found on vegetables and fruits. Dietary residues of DDT or its metabolites may increase the risk of breast cancer, but more information is needed (Wolff *et al.*, 1995). Aflatoxin contamination of food probably increases the risk of primary liver cancer (Scorsone *et al.*, 1992).

Effect of nitrate and n-nitroso compounds on cancer

Nitrate and nitrite are present naturally in food, the level developing on fertilizer use (both organic and inorganic). The major sources are vegetables, which supply 70-90% of the total and water which supplies 5-20%. Less than 3% of the total is supplied by added nitrate and nitrite used to preserve foods such as ham, bacon and corned beef. Added nitrate is metabolized in the

body to nitrite which is also produced in the body by white blood cells in response to infection and inflammation. Under certain conditions, nitrite can combine with nitrogen-containing substances to form N-nitroso compounds, most of which are known to be potent carcinogens. There is however, no consistent epidemiological evidence to suggest that individuals consuming more nitrate or nitrite are at greater risk than those consuming less, rather the reverse. In Britain, mortality from stomach cancer is lowest in areas where exposure to nitrate is highest, for example in East Anglia (Forman ***et al.***, 1985).

A case control study on diet and gastric cancer was carried out in Spain, in 1988 and 1989. Cases and controls were selected from 15 hospitals. An increased risk of gastric cancer was observed for high consumption of exogenous nitrosamines. An inverse association with the risk of gastric cancer was seen for high intake of fibre, vitamin C, folate, carotene and nitrates. High consumption of vitamin C seemed to neutralize the increased risk related to simultaneous consumption of nitrosamines. For histologic type, no meaningful differences in the effect of most of the nutrients were found between intestinal and diffuse cancers. The findings are consistent with previously reported results about the protective effect of fruit and vegetables and the increased risk associated with foods that are important sources of nitrites and preformed nitrosamines (Gonzalaz ***et al.***, 1994). Ecological evidence supported by experimental data suggests that exogenous dietary N nitrosamine exposure and endogenous N nitrosamine formation possibly increases the risk of oesophageal cancer (Lu ***et al.***, 1991).

Effect of food preparation and pattern of eating on cancer

The risk of cancer is also associated with the method of food preparation. Overcooked, barbecued, grilled or fried meat and fish may increase the risk of stomach cancer due to carcinogens produced on the surface of food by direct flame. Nitrites and nitrous compounds formed or added to food during smoking and curing process can be transformed into carcinogens, therefore, such food should be eaten occasionally. Frequent eating possibly increases the risk of colorectal cancer (Verdier and Longnecker, 1992).

Effect of temperature on cancer

Cooking meat at high temperatures possibly increases the risk of colorectal cancer (Robert Thomson ***et al.***, 1996) and esophageal cancer (De Stefani ***et al.***, 1990). Refrigeration decreases the risk of stomach cancer is convincing (Joossens and Kesteloot, 1996).

Effect of exercise on cancer

Three cohort studies of lung cancer have shown moderately decreased risk with higher levels of physical activity, after taking into account potential confounding by smoking habits, where as one case control study reported a weekly increased risk. Physical activity possibly decreases the risk of lung, colon and postmenopausal breast caner (Thompson, 1992; Brownson ***et al.***, 1991 and Simon, 1984).

Effect of smoking on cancer

Smokers are at a higher risk of lung, as well as other cancers and other diseases, against which antioxidants may have a protective effect. Moreover, smokers tend to consume diets relatively low in antioxidants. Smoking is associated with the consumption of diets including relatively fewer vegetables and fruits (McPhillips ***et al.***, 1994). For these reasons, it could be argued that smokers should consume relatively high quantities of antioxidant nutrients, in the form of supplements and that the more cigarettes smoked and the longer the duration of the habit, the higher the dose required. There are a number of arguments against any such policy. First, smoking is the overwhelming cause of lung cancer and any protective effect of antioxidants or other dietary factors is certain to be modest by comparison. Second, it would be unfortunate if smokers were encouraged to believe that they might continue their smoking habit safely if they were to take supplements.

Prevention of cancer

Cancer can be prevented if proper eating habits and life style are adopted. Proper storage and preparation of food help in prevention of cancer. Control of additives and insecticidal residues in foods help in reduction of cancer incidence. Methods of preparation of foods also reduce the occurrence of cancer. The World Cancer Research Fund (WCRF) has developed 14 diet and health recommendations for prevention of cancer (WCRF, 1997). These recommendations must be adopted to reduce the chances of incidence of cancer. These recommendations are as under.

Food supply and eating

Choose predominantly plant-based diets rich in a variety of vegetables and fruits, pulses (legumes) and minimally processed starchy staple foods.

Maintaining body weight

Avoid being underweight or overweight and limit weight gain during adulthood to less than 5 kg (11 pounds).

Maintaining physical activity

If occupational activity is low or moderate, take an hour's brisk walk or similar exercise daily and also exercise vigorously for a total of at least one hour in a week.

Vegetables and fruits

Eat 400-800 g (15-30 ounces) or five or more portions a day of a variety of vegetables and fruits, all year round.

Other plant foods

Eat 600-800 g (20-30 ounces) or more than seven portions a day of a variety of cereals (grains), pulses (legumes), roots, tubers. Proper minimally processed foods. Limit consumption of refined sugar.

Alcoholic drinks

Alcohol consumption is not recommended. If consumed at all, limit alcoholic drinks to less than two drinks a day for men and one for women.

Meat

If eaten at all, limit intake of red meat to no more than 80 grams (3 ounces) daily. It is preferable to choose fish, poultry or meat from non-domesticated animals in place of red meat.

Total fats and oils

Limit consumption of fatty foods, particularly those of animal origin. Choose modest amounts of appropriate vegetable oils.

Salt and salting

Limit consumption of salted foods and use of cooking and table salt. Use herbs and spices to season foods.

Storage

Do not eat food which, as a result of prolonged storage at ambient temperatures, is liable to fungal contamination.

Preservation

Use refrigeration and other appropriate methods to preserve perishable food as purchased and at home.

Additives and residues

When levels of additives, contaminants and other residues are properly regulated, their presence in food and drink is not known to be harmful. However, unregulated or improper use can be a health hazard and this applies particularly in economically developing countries.

Preparation

Do not eat charred food. Consume the following only occasionally: meat and fish grilled (broiled) in direct flame; cured and smoked meat.

Dietary supplements

For those who follow the recommendations presented here, dietary supplements are probably unnecessary and possibly unhelpful, for reducing cancer risk.

Nutritional management of cancer patients

Theologides (1987) has beautifully summarized the nutrition management of cancer patients. It is reproduced here with little modification

General principles

Because there usually are some nutritional problems from the time of the original diagnosis of a visceral cancer, the nutritional management should be coordinated with the overall therapeutic plan from the beginning. The specific nutritional recommendation will depend on the nutritional state of the patient, the type of cancer, the anticipated therapy and the prognosis. After the development of cancer, the nutritional needs may be higher than they were when the person was healthy. If a minimum of 30 to 35 kcal/kg with 1.0 g of protein/kg body weight was adequate for a person to maintain weight when afebrile with normal metabolic rate and limited activity, then after the development of cancer he or she may need more calories, protein, vitamins and minerals to maintain a nutrition and energy balance. With progression of the disease and with various therapies, the need for specific nutrients, minerals and vitamins may change.

When curative surgery is anticipated, a short period of nutritional rehabilitation, replenishment of deficiencies and correction of water, electrolyte and acid base imbalances may be advisable. Following curative surgery, the management should be directed toward restoring the patient's nutritional health to the pre-illness level with increased protein and calorie intake, adequate carbohydrates, fat, vitamins and minerals, along with exercise and other physical therapy when needed. Adverse nutritional implications from complications or after effects of surgery should be prevented when possible or managed.

In an effort to maintain their weight, patients receiving radiotherapy should be prevented from resorting to a deficient and imbalanced diet because of anorexia and nausea. After curative radiotherapy, a nutritional rehabilitation, if needed, can be achieved with a gradual increase of food intake of a balanced, easily digestible and absorbable diet.

The major nutritional problems appear in patients with recurrent, un-resectable, radioresistant, or disseminated cancer who are treated with aggressive chemotherapy. If the gastrointestinal tract is functioning properly in the patient with advanced cancer who is receiving chemotherapy, gastrointestinal feeding is preferable to parenteral nutrition. This enteral feeding can be accomplished through oral alimentation or through a nasogastric, esophagogastric, gastric, or jejunal feeding tube.

For oral feeding, the value of common food is modified in texture and consistency, blenderized and liquefied. The importance of frequent small feedings of food high in caloric and nutritional value is emphasized. Because it is digested faster, a diet of low fat content should be implemented. A regimen of nibbling rather than of widely spaced large meals is very important, especially in the presence of early satiety.

For tube feeding, either the common family food, blenderized and liquefied, or commercial preparations can be used. A very thin, pliable nasogastric tube should be inserted; this can be tolerated well, sometimes for weeks. The risk of a properly performed feeding gastrostomy is minimal, while a jejunostomy, although technically more difficult, has the advantage that it obviates the problem of gastric reflux. The percutaneous insertion of a gastric feeding tube is gaining popularity. Because a bolus might cause diarrhea and, occasionally manifestations of a dumping syndrome, the feeding should be given slowly through the tube. For the passage of

thicker preparations through a thin tube, a mechanical pump is more dependable than gravity flow to drip feed. In patients receiving hyperosmolar and high protein feedings, the water balance should be evaluated and corrected daily. Without sufficient water, the kidneys may be unable to clear metabolic products, resulting in hyperosmolar loading. If there is no glucose in the urine, a specific gravity higher than 1.02 should alert one to give the patient more water.

After chemotherapeutic control of the cancer, the restoration of wasted tissues is a slow process, but correction of vitamin and mineral deficiencies can often be accomplished rapidly. With reactivation of the disease, a new constellation of nutritional challenges appears and a revised nutritional approach may be needed.

Elemental diets

The commercially available elemental or defined diets contain mixtures of pure amino acids or protein hydrolysate, simple carbohydrates, small quantities of essential fatty acids and other triglyceride, minerals and vitamins. The powder or concentrated forms can be diluted in water, soft drinks, or milk. For patients with lactase deficiency, the formulas containing milk products and milk as a diluents should be avoided. The elemental diets have properties that are potentially useful in the nutritional support of the cancer patient. They can be nutritionally balanced and complete dietary formulations. They have a high nutritional value, relatively flexible composition and complete water solubility. They are easily digestible and readily absorbable. Elemental diets also may have several undesirable effects and potential complications. In most cases these are described on the package by the manufacturer. Fortunately major complications are uncommon.

In the nutritional management of the patient with advanced cancer, the elemental diet can be used either as a sole source of nutritional support or as a nutritional supplement between meals. Because it is bulk-free and is absorbed directly across a relatively short segment of the small intestine, the main indications for its use are malfunction of the gastrointestinal tract, short bowel and intestinal fistulas. Such a diet may protect the patient from losing weight due to mal absorption resulting from radiotherapy or chemotherapy induced enteritis. As a supplement between meals, elemental diets can provide additional calories and nutrients to the anorectic, cachectic cancer patient. Three packages or cans of elemental diet taken between meals may increase caloric intake by at least 900 Kcal/day.

Slow sipping is important to prevent bloating, nausea and hyperosmolar diarrhea. Because of the un-palatability of elemental diets and the flavor fatigue, it is very difficult to convince patients to continue using them for more than a few days. Only the appreciation of their importance in contributing calories and nutrients can assure patients' acceptance and cooperation for longer periods.

When the elemental diet is given as the sole source of total nutritional support, it is very difficult to voluntarily consume the required volume orally. In such cases a small, pliable nasogastric tube should be inserted to secure total control of the caloric and fluid intake. This hypertonic elemental diet should be given through a tube at a constant rate for a 24 hour period rather than intermittent boluses. A pump with a controllable speed for continuous administration is preferable to the unpredictable gravity dripping from a plastic bag reservoir. Because most

elemental diets are hyperosmolar, tube feeding should begin with diluted solutions in small volumes per hour, then increase in concentration and volume gradually so as to reach the required caloric and fluid intake in 5 to 6 days. Because there may be some fluid retention during the administration of the elemental diet, occasional monitoring of the patients' fluid and electrolyte balance and urine specific gravity is imperative. Patients on nasogastric tube feeding have a potential risk of aspiration, which should be guarded against. When the reasons and indications for elemental diet alimentation are no longer present, the patient should return gradually to oral feeding with common food.

Total parenteral nutrition

Total parental nutrition (TPN) plays an important role in the nutritional management of selected patients with advanced cancer. It can provide all the nutritional requirements for prolonged periods during and after the administration of effective chemotherapy, which is very toxic to the gastrointestinal tract. The technique involves insertion of a catheter into a central vein (usually subclavian) and infusion of concentrated nutrient preparations in quantities and composition individualized for the patient. With this approach nutrition may be improved and maintained in a malnourished, debilitated cancer patient. This assures a better tolerance of the effective anti neoplastic therapy in certain malignant neoplastic diseases. TPN should not be used merely to prolong survival of patients who are at the end stage of their disease.

The patients selected for TPN should have a cancer that is potentially controllable with surgery or radiotherapy and responsive to available chemotherapy. They should also be unable to meet their nutritional requirements through enteral feeding because they are unable to ingest adequate food or because their gastrointestinal tracts are incapable of adequately digesting and absorbing nutrients. Patients who meet the above criteria and weight 5 kg or more below the ideal body weight and who have a serum albumin concentration of less than 3 g/100 ml are candidates for TPN.

The goal should be to provide adequate nutrition before and during treatment and to maintain and increase the body weight aiming toward a better tolerance of the effective treatment and a faster recovery. It is hoped that with tumor regression and recovery from the adverse effect of chemotherapy and radiotherapy, appetite will return, the gastrointestinal tract will function properly and the weight that was gained will be maintained with oral intake.

TPN usually requires a team of a physician, nurse, pharmacist and dietitian to prepare the nutritional fluids, insert the central catheter, guarantee sterility and supervise safe administration. Close clinical observation and biochemical monitoring are imperative to prevent or correct various metabolic complications that may occur with TPN and meticulous care is needed to keep the incidence of catheter-related infections very low.

A 10% fat emulsion (intra-lipid) given intravenously in a peripheral vein concomitantly with glucose and free amino acid solutions represents another approach to short-term parenteral alimentation. It can be used also in cancer patients who have an inadequate or imbalanced oral food intake to provide essential fatty acids, triglyceride, amino acids, lipids and glucose. This combination of free amino acids, lipids, glucose, electrolytes, other minerals and vitamins is a

simple approach to parenteral nutrition through a peripheral vein and it is useful for short term nutritional maintenance. There are potential local and systemic complications: the intra-lipid cannot be mixed with other intravenous (IV) fluids and no electrolyte solutions or drugs can be added to the preparation. Administration of Intralipid and amino acids with sugar should be through a separate IV needle or through a shared needle or catheter with a Y-connector at the needle bulb or catheter end.

It is hoped that with the improved surgical techniques, more aggressive radiotherapy, more effective chemotherapy and prevention and control of complications of the disease and of the treatment, a greater number of patients with cancer may live longer. Maintenance of a good nutritional state may improve the quality of life. In selected patients who are markedly malnourished, cannot maintain adequate protein and calorie intake and have a malignancy that is potentially responsive to aggressive chemotherapy, the parenteral nutrition might make the inference between life and death. For the cancers in which chemotherapy is not very effective, concomitant enteral or parenteral hyper alimentation is of questionable benefit.

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