Pain & Palliative Care in Homeopathy A study on the Effectiveness of Homeopathic Medicines in the Management of Pain in Cancer Dr Rithesh B

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- Dr. Rithesh.B

Introduction

Cancer is a common and widely publicized disease, and in spite of ever increasing effort to understand it as a process, its incidence in the population is rising. The main reason for this is the close correlation of the number of cancer cases with the increasing age of the patients and the number of more aged people in the western society (and also in Kerala) at least is rising .It used to be suggested that same aspect of the ageing process increased the susceptibility to cancer, perhaps by impairing immune surveillance .However it is now generally accepted that the relationship of many cancer cases to increasing age is rather a reflection of time required to accumulate a critical number of genetic abnormalities for cancer to arise .Cancer may affect any organ or tissue ,but while some cancer are common, Eg; lung ,breast, gut, prostate, others are very rare ,those affecting the young people often being amongst the rarest .In particular ,cancer affect epithelial tissue and over a 99% of tumor are derived from this tissue .This is not surprising since many of the known cancer causing agents (carcinogens) are from natural radiation. In the air we breathe and from the food stuff we ingest, and epithelial cells are the first line of defense to the outside world, in the skin, lung, and gastrointestinal tract¹

The prevalence of acute and chronic cancer pain and the profound psychological and physical burdens engendered by this symptom oblige all treating physician to be skilled in pain management.²

Since homoeopathic treatment has found to be effective in managing cancer pain, and since a scientific study on this subject is not known to be conducted, an attempt is made to evaluate the effectiveness of homoeopathic medicines with appropriate statistical analysis. A prospective study is conducted by studying the cancer patient with pain attending the out patient and in patient department of Govt. Homoeopathic medical college, Calicut. The patients are assessed at the time of consultation using, Pain rating scale, Degree of distress score and performance status score to rate the pain and quality of life.

Even though the aim of study is palliation of pain, cases are taken according to homoeopathic philosophy giving importance to the general symptoms .The selection of medicine are also based on the homoeopathic philosophy giving more importance to the general symptoms and if case demands taking sectoral totality of pain giving importance to its modifying factors and the characteristics of pain. Miasmatic aspect of the cases are studied and given due importance in the selection of medicine.

AIM AND OBJECTIVE OF THE STUDY

To assess the efficacy of homoeopathic treatment in the management of pain in cancer.

Review of Literature

Neoplasia literally means 'the process of new growth' and a new growth is called "neoplasm". However all new growth are not neoplasms, since examples of new growth of tissues and cells also exist in the process of embryogenesis, regeneration and repair, hyperplasia and hormonal stimulation. There fore a satisfactory definition of neoplasm or tumor is "a mass of tissue formed as a result of abnormal, excessive, uncoordinated, autonomous and purposeless proliferation of cells". ³

The branch of science dealing with the study of neoplasm is called "Oncology" (oncos -tumor, logos -study). Neoplasm may be "benign" when they are slow growing and localized with out causing much difficulty to the host .or "Malignant" when they proliferate rapidly, spread through out the body and may eventually cause death of the host .The common term used for all malignant tumor is cancer. Hippocrates (460-377 BC) coined the term "Karkinos" for the cancer of the breast .The word cancer means "Crab" thus reflecting the prime character of cancer since it sticks to the part stubbornly like a crab⁴.

International union against cancer (IUAC) has defined cancer as a "disturbance of growth characterized primarily by excessive proliferation of cells with out apparent relation to the physiological demand of the organ involved"⁵.

All tumors benign and malignant have the basic component,

- 1) Parenchyma composed of proliferating tumor cell –parenchyma determines the nature and evolution of the tumor.
- 2) Supportive stroma comprised of fibrous connective tissue and blood vessels, it provides the framework on which the parenchyma tumor cell grow .³
- 3) The tumor derives their nomenclature on the basis of the parenchymal component comprising them. The suffix "oma" is added to denote benign tumors, malignant tumors of epithelial origin are called "carcinomas" while malignant mesenchymal tumors are named "sarcomas" (sarcos-fleshy) however some cancers are composed of highly undifferentiated cells and are referred to as undifferentiated malignant tumors. Although the broad generalization regarding nomenclature of tumors usually holds true in majority of instances, some examples contrary to the concept are –melanoma for carcinoma of melanocytes, hepatoma for carcinoma of hepatocytes, lymphoma for malignant tumor of lymphoid tissue and seminoma for malignant tumor of testis.³

Tumors composed of a single type of parenchyma cells that differentiate towards more than one cell line are called mixed tumor. Teratomas on the other hand are made up of a number of parenchymal cell types arising from totipotent cells derived from more than one germ cell layer. Choristoma refers to ectopic rests of normal tissues. Hamartoma is a mass of disorganized but mature cells of tissue indigenous to the particular site .³

Classification	of tumor ³

Tumor of origin	Benign	Malignant
	Beiligh	Manghant
I. Tumors of one		
parenchymal cell		
type		Squamous cell
A) Epithelial tumors		carcinoma
:	Squamous cell	
1.Squamous	papilloma	
epithelium	papillollia	Transitional cell
epitielium	Turnelting	
	Transitional cell	carcinoma
	papilloma	
2. Transitional		Adenocarcinoma
epithelium		Basal cell carcinoma
	Adenoma	Melanoma
3. Glandular	-	Hepatoma
epithelium	Nevus	Choriocarcinoma
4.Basal cell layer	Liver cell adenoma	
skin	Hyaditidiform mole	
5. Neuroectoderm		
		Lineeruse
6. Hepatocytes		Liposarcoma

7. Placenta		Fibrosarcoma
B . Non epithelial	Lipoma Fibroma	Myxosarcoma
B . Non epithelial (mesenchymal) tumors	Myxoma	Chondrosarcoma
1.Adipose tissue 2.Adult fibrous tissue	Chondroma	Osteosarcoma
3.Embryonic fibrous tissue	Osteoma	Synovial sarcoma Leiomyosarcoma
	Benign synovioma	Rhabdomyosarcoma
4. Cartilage	Leiomyoma	Mesothelioma
5. Bone 6. Synovium	Rhabdomyoma	Angiosarcoma Lymphangiosarcoma
7. Smooth muscle	Hemangioma	-
8.Skeletal muscle	Lymphangioma	Invasive meningoma
9. Mesothelium	Glomus tumor	Leukemias
10. Blood vessels 11. Lymph vessels	Meningiomas -	Malignant lymphomas Neurogenic sarcoma
12. Glomus	Pseudolymphomas	Neurogenic sarconia
13. Meninges	Neurilemmoma,	Neuroblastoma
14. Hematopoetic cells	neurofibroma	Malignant mixed salivary tumor
15. Lymphoid tissue		
16. Nerve sheath	Ganglioneuroma Pleomorphic	
17. Nerve cells	adenoma	Immature teratoma
II) Mixed tumors Salivary glands		
III) Tumors of more		
than one germ cell layer	Mature teratoma	
Totipotent cells in		
gonads or in		
embryonal nests		

Table: 1

2.1. Cancer incidence:

The incidence of cancer has increased considerably over the last 50 years because of tobacco smoking and greater life expectancy. $^{\rm 6}$

Cancer rate could further increase by 50% (10 million new cases globally in 2000) to 15million new cases in the year 2020, according to the world cancer report, the most comprehensive global examination of the disease to date⁶.

In the year 2000 malignant tumor were responsible for 12% of the nearly 56 million death world wide from all the causes .In 2000, 5.3 million men and 4.7 million women developed a malignant tumor and all together 6.2 million women died from the disease. The report also reveals that cancer has emerged as a major public health problem in developing countries matching its effect in industrialized nation ⁷.

In USA in 2003, 556000 deaths was from cancer alone, representing 25% of all mortality, a frequency surpassed only by death caused by cardiovascular diseases.⁴

The estimated cancer incidence by site and sex are as follows. In male thirty percentages is prostate cancer. Fourteen percentage lung and bronchus related cancer. Colon and rectum accounts for eleven percentage of total cancer; urinary bladder seven percentages, melanoma of the skin five percentage. Four percentages is Non Hodgkin's lymphoma, three percentages each cancer of oral cavity, kidney and leukemia. Pancreatic cancers account for two percentages and all other sites nineteen percentages. In female thirty one percentages is breast cancer. The lung and bronchus related cancers and colon and rectal cancers twelve percentages each. Six percentages is uterine cancer. Non –Hodgkin's lymphoma, melanoma of the skin and ovarian cancer four percentages each. Pancreatic cancers, thyroid cancers and cancer of the urinary bladder

two percentages each. All other sites accounts for the rest twenty percentages.⁴The estimated frequency of cancer death by site and sex are as follows, in male thirty one percentages death are due to cancer related to lung and bronchus. Prostate cancer accounts for eleven percentages, ten percentage death due to colon and rectal cancer. Pancreatic cancer and Non-Hodgkin's lymphoma accounts for five percentages of death each. Four percentages due to leukemia, three percentages each due to carcinoma of the esophagus, liver, urinary bladder and kidney. Two percentages due to all other sites. In females twenty-five percentage deaths is due to cancer related to lung and bronchus. Breast cancer accounts for fifteen percentages of death. Eleven percentages due to cancer related to colon and rectum, six-percentages death due to cancer of pancreas and five percentages due to cancer of the ovary. Four percentages of death each due to leukemia and Non-Hodgkin's lymphoma. Two percentages each due to cancer of the uterus, brain and multiple myeloma. Twenty-three percentages of the remaining death are due to cancer of all other sites. Over the past 50 years, the overall age adjusted cancer death rate has significantly increased in men, where as it has fallen significantly in women .The increase in men can be largely attributed to lung cancer .The improvement in women is mainly attributed to a significant decline in death rate from the cancer uterus, stomach, liver, and most notably from cancer of the cervix, one of the most common malignant neoplasia in women. Striking is the alarming increase in the death rate form the carcinoma of the lung in both sexes .In women carcinoma of the breast occur 2.5 times more frequently than those of the lung Because of the large difference in the cure rates of these two cancers, however lung cancer has become the leading cause of cancer death in woman.⁴

2.2. Cancer incidence in India and Kerala

The rate of cancer occurrence in Kerala and in India is much lower compared to western countries .It is now estimated that 25000 new cancer cases occur in Kerala in one year. Among the males 50% of cancers in the mouth, throat and lung are caused by tobacco and alcohol habits. Among women the incidence of tobacco related cancer is 15%.⁸

st prevalent cancer in India				
Rank	In men	In women	Overall	
1	Lung	Breast	Lung	
2	Stomach	Cervix	Stomach	
3	Colon /rectum	Liver	Liver	
4	Prostate	Stomach	Colon/rectom	
5	Oral	Lung	Esophagus	
6	Liver	Oral	Breast	
7	Esophagus	Ovary	Oral	
8	Bladder	Body of the uterus	Cervix	

Most prevalent cancer in India ⁹

Table: 2

Cancer incidence in Kerala-Trivandrum

Male, age [0-85+]¹⁰

Cancer	Cases	Crude rate
Lip	11	0.4
Tongue	124	4.5
Salivary glands	13	0.5
Mouth	207	7.6
Pharynx	128	4.7
Esophagus	70	2.6
Stomach	81	3.0
Small intestine	5	0.2
Colon	42	1.5
Rectum and anus	52	1.9

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Liver	57	2.1
Gallbladder etc.	5	0.2
Pancreas	40	1.5
Nose sinuses etc.	9	0.3
Larynx	93	3.4
Lung (incl. trachea and bronchus)	197	7.2
Bone	17	0.6
Melanoma of skin	16	0.6
Other skin	37	1.4
Connective tissue	22	0.8
Breast	3	0.1
Penis	19	0.7
Prostate	88	3.2
Testis	13	0.5
Kidney etc.	28	1.0
Bladder	44	1.6
Eye	2	0.1
Brain, central nervous system	60	2.2
Thyroid	46	1.7
Other endocrine	5	0.2
Hodgkin lymphoma	20	0.7
Non-Hodgkin lymphoma	64	2.3
Multiple myeloma	25	0.9
Leukemia	94	3.4
All sites	2017	73.9
All sites but skin	1980	72.6
	Table: 3	

Table: 3

2.3. Etiology and pathogenesis of cancer

A lot of clinical and experimental research and epidemiological studies have been carried out in the field of oncology, so as to know the possible cause of cancer and mechanism involved in the transformation of a normal cell into a neoplastic cell. It is widely known that no single factor is responsible for the development of tumours.³

Based on the current state of knowledge these factors are broadly described under 2 main headings.

- 1. Predisposing epidemiological factors, which include a number of endogenous host factors and exogenous environmental factors.
- 2. Carcinogenesis that encompasses exogenous agents like chemical, physical, hormonal and biological substances.

Epidemiological factors

1. Familial and genetic factors:

In general the risk of developing cancer in relatives of a known cancer patient is about three times higher as compared to control subjects. The overall estimate suggests that genetic cancer comprises not greater than 5% of all cancer. Some of the common examples are,

- a. Retinoblastoma: about 40% of retinoblastoma are familial and show an autosomal dominant inheritance. Retinoblastoma susceptibility gene- Rb gene located on chromosome 13 was the first cancer predisposing gene identified.
- b. Familial polyposis coli autosomal dominant inheritance.

- c. Multiple endocrine Neoplasia.
- d. Von Recklinghausen's disease.
- e. Cancer of the breast- female relatives of breast cancer patients have 2 to 3 times higher risk of developing breast cancer

2. Racial and geographical factors

Differences in racial incidence of some cancer are largely attributed to the influence of environmental and geographic differences affecting the whole population such as climate, water, soil, diet habits, customs, etc. some of the examples of racial and geographical variations in various cancer are as under,

a. White Europeans and Americans develop most commonly malignancies of lung, breast and colon. Liver cancer is uncommon in these races.

b. Black Africans on the other hand have more commonly cancer of the glans penis, cervix and liver.

c. Japanese have five times higher incidence of carcinoma of the stomach than the Africans.

3. Environmental and cultural factors:

Surprising, as it may seem, we are surrounded by an environment of carcinogens we eat, drink, inhale and touch. Some examples are,

a. Cigarette smoking is the single most important environmental factor implicated in the etiology of cancer of the oral cavity, pharynx, larynx, esophagus, lung, pancreas and urinary bladder.

b. Alcohol abuse predisposes to the development of cancer of the oropharynx, larynx, esophagus and liver.

c. Cancer of the cervix is linked to a number of factors such as age of the first coitus, frequency of coitus, multiplicity of partners, parity etc,.

d. Penile cancer is rare in Jews and Muslims as they are customarily circumcised .

e .Betel nut cancer of the cheek and tongue is quite common in some parts of India due to the habitual practice of keeping the bolus of pan in a particular place in the mouth for a long time.

f. A large number of industrial and environmental substances are carcinogens and are occupational hazards for some population-arsenic, benzene, vinyl chloride.

g. Certain constituent of diet have been implicated in the causation of cancer. Over weight individuals, deficiency of Vit.A and people consuming diet rich in animal fat and low in fiber content are more at risk of developing certain cancer of the colon.

4. Age:

Generally cancer occurs in older individuals past 5th decade of their life. Some tumors have two peaks of incidence, example acute leukemia occurs in children and older age group.

f one plots on a semi log scale, the age distribution of some of the most important cancers, then most carcinoma are rare under the age of 30, but then the incidence rate increases dramatically $(10^3 - 10^4 \text{ times})^{-10^4}$ with the age .The most attractive explanation for this exponential relationship is that, three to seven hits (mutations) are required for a cancer to form. Of course not all cancers show a sharp rise in incidence rate with age, testicular cancer show a peak incidence between the second and fifth decades and then declines, while the peak incidence of leukemia and nervous system cancers occur not only at greater age but also in early childhood, suggesting the influence of prenatal factors¹.

5. Sex: -

Most tumors are generally more common in men than in women except cancer of the breast, gall bladder, thyroid and hypopharynx. Cancer of the breast is the commonest cancer in women through out the world, while lung cancer is the commonest cancer in men.

6. Pre-malignant lesions:

Pre-malignant lesions are a group of conditions, which predisposes to the subsequent development of cancer.

Some examples of pre-malignant lesions are,

1) Carcinoma in situ (intraepithelial neoplasia) –when the malignant cells are confined to the epithelium without invasion across the basement membrane it is called as carcinoma in situ or intra epitheleal neoplasia.

e.g.: uterine cervix at the junction of the ecto and endo cervix.

Bowen's disease of the skin.

- Some benign tumors
 Eg: multiple adenoma of the large intestine have high incidence of developing adenocarcinoma. Neurofibromatosis may develop in to sarcoma.
- 3) Miscellaneous conditions certain inflammatory and hyperplasic conditions are prone to the development of cancer.

Eg: patients of long standing ulcerative colitis

Cirrhosis of the liver Chronic bronchitis

2.4. Carcinogenesis

Carcinogenesis means induction of tumors, agents which can induce tumors are called carcinogens. Carcinogens are a variety of extrinsic agents which are broadly divided in to 4 groups.

- A- Chemical carcinogens
- B- Physical carcinogens
- C- Hormonal carcinogens

D- Biologic carcinogens (chiefly viruses)

Carcinogenesis by these are discussed below

a) Chemical carcinogenesis

The first ever evidence of any cause for neoplasia came from the observation of Sir. Percival Pott in 1775 that there was higher incidence of cancer of the scrotum in chimney-sweepers in London than in the general population .The first successful experimental induction of cancer was produced by two Japanese workers in 1914 in the rabbits skin by repeatedly painting with coal tar.

The induction of cancer by chemical carcinogens occurs after a delay of weeks to months in the case of experimental animals, and often several years in man.

Depending up on the mode of action of carcinogenic chemicals they are divided in to 2 broad groups,

- 1) Initiators of carcinogenesis
- 2) Promoters of carcinogenesis

Important chemical carcinogens are

[Carcinogen		Tumour
	1	Initiators of carcinogenesis i)Direct acting carcinogens a) Alkylating agents wAnticancer drugs Eg : cyclophosphamide clorambucil,busulfan w B propiolactone w Epoxide b) Acylating agents –acetyl imidazole	Lymphoma Leukemia
	 ii) Indirect acting carcinogens (pro carcinogens) a) Polycyclic aromatic hydrocarbons (in tobacco, smoke ,fossil fuel tar ,mineral oil smoked animal food ,industrial and atmospheric pollutants) w Benzanthracene w Benzapyrene w Methylcholantrene 		-lung cancer -skin cancer -Cancer of the oral cavity -Sarcoma
		 b) Aromatic amines and azo dyes wb naphthylamine Benzidine -Azo dyes (scarlet red ,butter yellow) -Acetyl aminoflurene 	Bladder cancer Hepatocellular carcinoma
	c) Naturally occurring products – Aflatoxin B1 – ActinomycinD – Mitomycin D – Safrole – Betel nuts		Hepatocellular carcinoma
		 d) Miscellaneous Nitro so compounds Vinyl chloride monomer Asbestos 	-Gastric carcinoma -Hemangiosarcoma -Bronhogenic carcinoma,

	 Arsenical compounds Metals (nickel, lead, cobalt) Insecticides, fungicides (eldrin, dieldrin, chlordane) 	mesothelioma Epithelial hyperplasia, basal cell carcinoma Lung cancer Experimental cancer
2	Promoters of carcinogenesis a) Phorbol esters b) Phenols c) Hormones (estrogen) d) Drugs (eg-phenobarbitol) e) Artificial sweeteners (Eg- saccharin. cyclamates)	

b) Physical carcinogenesis

Table: 4

Physical agents in carcinogenesis are divided in to 2 groups

1) Radiation - both ultraviolet and ionizing radiations , is the most important physical agent

2) Non -radiation physical agents are the various forms of injury and are less important

1) Radiation carcinogenesis: - ultraviolet light and ionizing radiations are the two main forms of radiation carcinogens. Property common between the two forms of radiation carcinogens is the appearance of carcinogenic effect long after exposure often 10 -20 years or even after.

UV light penetrates the skin for a few millimeters only so that its effect is limited to the epidermis .In humans, excessive exposure to UV rays can cause various forms of skin cancers – squamous cell carcinoma, basal cell carcinoma and malignant melanoma.

Ionizing radiation of all kinds like X-rays, alpha, beta and gamma radiations, radioactive isotopes, protons and neutrons can cause cancer in animal and in man. Most frequently radiation induced cancers are all forms of leukemias (except CLL); others are cancer of the thyroid skin, breast, lung and salivary glands.

2) Non radiation physical carcinogenesis:

Asbestosis and asbestos associated tumors of the lung are malignant mesothelioma of the pleura .Other example of physical agents in carcinogenesis are the implants of inert materials such as plastic ,glass etc in the prosthesis or otherwise ,and foreign bodies observed to cause tumour development in experimental animals .

C) Hormonal carcinogenesis:

Cancer is more likely to develop in organs and tissues, which undergo proliferation under the influence of excessive hormonal stimulation. Hormone sensitive tissue developing tumors are the breast, endometrium, myometrium, vagina, thyroid, liver, prostate and testis.

The main hormones inducing cancer are

- a) Estrogen examples of estrogen induced tumors are, women receiving estrogen therapy and women with estrogen secreting granulosa cell tumor of the ovary have increased risk of developing endometrial carcinoma.
- b) Contraceptive hormones the sequential types of oral contraceptives increases the risk of developing breast cancer.
- c) Anabolic steroids Consumption of anabolic steroids by athletes increases the risk of developing benign and malignant tumors of the liver.
- d) Hormone dependant tumors induction of hyper function of adenohypophysis is associated with increased risk of developing neoplasia of the target organ following preceding functional hyperplasia. There is tumour regression on removal of the stimulus for the excessive hormonal secretion, Eg: prostate cancer usually responds to the administration of estrogens.

Breast cancer may regress with oopherectomy, hypophysectomy or on administration of male hormones.

Thyroid cancer may slow down in growth with the administration of thyroxin that suppresses the secretion of TSH by the pituitary.

D) Viral carcinogenesis

Oncogenic viruses can be transmitted by one of the three routes –vertical transmission, horizontal transmission, by inoculation. Based on the nucleic acid content oncogenic viruses fall in to two broad groups,

i) DNA oncogenic viruses

ii) RNA oncogenic viruses

DNA oncogenic viruses:

	Virus	Host	Associated tumor
1)	Papova virus Human papilloma virus	Human	Papillomas on the skin, larynx ,genitals: skin cancer in epidermodyspalsia
	Papilloma viruses	Cotton tail rabbits Bovine	Papilloma Alimentary tract cancer
	Polyoma virus	Mice	Various carcinomas, sarcomas
	Sv -40 virus	Monkeys Hamsters	Harmless Sarcoma
2	Herpes viruses Epstein bar virus	Human	Burkitt lymphoma Nasopharyngeal carcinoma
	Human herpes virus B	Human	Kaposi's sarcoma B cell lymphoma
	Luckes frog virus	Frog	Renal cell carcinoma
	Mareks disease virus	Chickens	T cell leukemia lymphoma
3	Adeno viruses	Hamsters	Sarcoma
4	Pox viruses	Rabbits Human	Myxomatosis Molluscum contagiosum papilloma
5	Hepadino viruses Hepatitis B virus	Human	Hepatocellular carcinoma
		Table: 5	

RNA oncogenic viruses:

RNA oncogenic viruses are retroviruses, i.e. they contain the enzyme reverse transcriptase .The enzyme, reverse transcriptase is required for reverse transcription of viral RNA to synthesize viral DNA strands .They are divided in to three subgroups- acute transforming viruses, slow transforming viruses and human T cell lymphotrophic viruses (HTLV).The former two are implicated in inducing a variety of tumors in animals while HTLV is causative for human T cell leukemia and lymphoma.

RNA oncogenic viruses

	Viruses	Host	Associated tumour
1	Acute transforming viruses Rous sarcoma virus Leukemia –sarcoma virus	Chickens Avian, feline bovine, primate	Sarcoma Leukemia, sarcoma
2	Slow transforming viruses Mouse mammary tumor virus	Mice, cats bovine Daughter mice	Leukemia, lymphoma Breast cancer
ω	Human T cell lymphotrophic virus HTLV- 1 HtLV-11	Human	Adult T cell leukemia lymphoma
		Human	T cell variant of hairy cell leukemia
4	Hepatitis C virus HCV	Human	Hepatocellular carcinoma
		Tables 6	

Table: 6

Molecular genetics of cancer

Broadly speaking genes and molecular factors involved in pathogenesis of cancer can be grouped in to 4 categories

- 1) Oncogenes (i.e. cancer causing genes)
- 2) Anti- oncogenes (i.e., cancer suppressing genes)
- 3) Mutator genes (i.e., genes that regulate DNA repair)
- 4) Telomerase in cancer (i.e., telomere shortening as cancer suppressor mechanism)

Oncogenes: -

Oncogenes are cancer causing genes. They are derived from proto-oncogenes or cellular oncogenes (c-oncs) which are detected on normal animal and human cell and promote normal growth and differentiation of cells.

Human oncogenes are identified by DNA transfection and by non-random chromosomal abnormalities.(e.g.: translocations)

Mechanism of activation of cellular oncogenes: ³

There is similarity between normal genes coding for proteins for growth and differentiation on one hand and oncogenes of viral and tumor origin on the other .How these normal genes are activated to become oncogenes is explained on the basis of two type of mechanisms

i) Change in the structure of the gene &

ii) Change in the regulation of gene expression

Based on this, examples of activation of human oncogenes in human tumors are under,

- a) Point mutations and deletion, Eg: ras oncogene
- b) Chromosomal translocations, Eg: Philadelphia chromosome seen in 95 % of the case of chronic myeloid leukemia .In 75% case of Burkit's lymphoma, translocations of c- myc -proto-oncogene from its site on chromosome 8 to a portion on chromosome 14.
- c) Gene amplification chromosomal alterations that results in increase in the number of a gene, eg: Neuroblastoma having n-myc -HSR region Erb-B in breast and ovarian cancer.

Mechanism of action of oncogenes:

Oncogenes have oncoproteins, which are altered form of their normal counter parts, proto-oncogenes, regulating growth and differentiation. Thus proliferation of cells by oncogenes induces the steps shown below³,

The important oncogenes and their mode of activation and associated human tumor :

Туре	Oncogenes	Associated tumor
1) Extra cellular growth factor		
β chain of PDGF FGF	sis	Sarcoma, glioblastoma
	hst,k53	Stomach cancer, kaposi's sarcoma

2) Trans membrane growth factor receptors .EGF –	erb B1	Carcinoma lung
receptors	her -2 /neu	Ca breast, ovary
CSF receptor		
	Fms	Leukemia
3) Intracellular signal transduction proteins GTP binding	Ras	Ca lung ,colon ,pancreas
Non receptor tyrosine kinase	Abl	CML,ALL
4) Transcription proteins (nuclear regulatory proteins)		
Transcription		
activators		
	Мус	Burkit's lymphoma
	N -myc	Neuroblastoma
5) Cell cycle regulatory proteins		
Cyclins	Cyclin D	Mantle cell lymphoma
Cyclin dependent	CDK 4	Glioblastoma,
kinase		melanoma
6) Apoptotic inhibitors		
Anti apoptotic genes		
	bcl -2	Follicular B cell lymphoma

Ta	ble	::	7

Anti –oncogenes (tumor suppressor genes):

Tumor suppresser gene or anti –oncogenes, just as proto- oncogenes are also a pair of normal genes which perform the physiologic function of regulation of cell growth. Mechanism of stimulation of carcinogenesis by tumor suppresser gene is mutation in both alleles producing deficiency of normal gene product, which normally suppresses tumor formation. Homozygous deletion or mutation at specific genetic loci are seen in such tumor cells Eg: in retinoblastoma, Wilm's tumor, familial adenomatous polyposis coli, breast cancer etc. Important tumor suppresser gene and associated human tumors:

Gene	Location	Associated human tumor
1) Rb	Nucleus	Retinoblastoma
		Osteosarcoma
2) p53	Nucleus	Colorectal cancer
		50% of all human cancers
3) APC	Cytosol	Familial APC
4) WT1	Nucleus	Wilm's tumor
5) NF 1	Plasma membrane	Neurofibromatosis type 1
6)BRCA 1,BRCA 2	Nucleus	Ca breast ,Ca Ovary

Table: 8

Mutator oncogenes :

Normal cells have caretaker genes to take care of the integrity of genetic information in response to DNA damage. The mutated version of mutator gene is characterized by loss of normal surveillance function and render the DNA susceptible to accumulation of mutations and therefore progression to cancer.

Telomerase in cancer:

Telomeres are the terminal tips of chromosome which progressively shorten due to repetitive cell division .Telomerase is the enzyme required for continued recognition of telomere in successive cell divisions. Cancer cells express telomerase with consequent telomere lengthening and further immortalization of cancer cells.

High risk factors for cancer

- a) Excess alcohol drinking
- b) Excess tobacco chewing, smoking
- c) Lack of green and yellow vegetables
- d) Excess consumption of high fat diet
- e) Early age at marriage, multiple sexual partners, poor genital hygiene.

2.5. Clinical effects of Neoplasia:

Two major aspects of clinical; significance in assessing the course and management of neoplasia are: tumor host inter-relationship and laboratory diagnosis of cancer

Effects of tumor on host:

Malignant tumors produce more ill effects than the benign tumors

1) Local effects:

Both benign and malignant tumors cause local effects on the host due to their size or location. Some of the local effects of tumors are

- i) Compression
- ii) Mechanical obstruction
- iii) Tissue destruction
- iv) Infarction, ulceration, hemorrhage
- 2) Cancer cachexia:

Patients with advanced and disseminated cancers terminally have asthenia, emaciation and anorexia, together referred to as cancer cachexia .Cachectin or tumor necrosis factor alpha derived from macrophages play a contributory role in cachexia.

3) Fever:

Fever of unexplained origin may be presenting feature in some malignancies such as in Hodgkin's disease, adenocarcinoma of the kidney, osteogenic sarcoma and many other tumors. The exact mechanism of tumor-associated fever is not known but probably the tumor cells themselves elaborate pyrogens.

4) Para-neoplastic syndromes:

Para-neoplastic syndromes (PNS) are a group of conditions developing in patients with advanced cancer which are not explained by direct and distant spread of tumor. About 10- 15% of the patients with advanced cancer develop one or more of the syndromes included in the P.N.S listed below, ³

Clinical syndrome	Underlying cancer	Mechanism
1.Endocrine syndrome i. Hypercalcemia	Lung(sq.cell.ca),kidney, breast Adult T cell leukemia-lymphoma	Parathormone like protein Vitamin D
ii.Cushings syndrome	Lung(smallcell.ca),pancreas, neural tumors	
	Lung (small cell ca),prostate	ACTH or ACTH like

	intracranial tumor	substances
iii. Inappropriate		
anti diuresis	Pancreas(islet cell tumor) mesothelioma, Fibrosarcoma	ADH or atrial natriuretic factor
iv. Hypoglycemia	Bronchial carcinoid tumor , Ca pancreas, stomach	Insulin or insulin like substance
v. Carcinoid		
syndrome	Kidney,liver,cerebellar hemangioma	Serotonin Bradykinin
vi. Polycythemia		
	Thymoma	Erythropoietin
2.Neuromuscular syndrome	Lung (small cell ca), breast	
i. Myasthenia gravis		
ii. Neuromuscular		
disorders		
3.Osseous,joint&soft tissues	Lung	Immunologic
i. Hypertrophic	Lung	
osteoarthropathy		Immunologic
ii.Clubbing of		
fingers	Pancreas ,lung ,GIT	
	Advanced cancers	
4.Hematologic syndromes		
i.Thrombophlebitis	AML, adenocarcinoma	
ii. Non bacterial	Thymoma	Not known

thrombotic		
endocarditis	Lymphoma of small bowel	
		Not known
ii. DIC	Advanced cancers	
iii. Anemia		
5.Gastrointestinal		
syndromes	Stomach, large bowel	Hypercoagulabilty
i. Malabsorption		
	Bowel	Hypercoagulability
6.Renal syndromes		
I. Nephritic	Lymphoma	
syndrome		
		Chronic thrombotic phenomina
7.Cutaneus syndromes	Multiple myeloma	Unknown
Syndromes		UTIKIOWI
i. Acanthosis	Kidney, lymphoma, solid tumors.	
nigricans		Hypoalbuminemia
ingrioune		in poulouin interna
ii.Seborrheic		
keratosis		
		Renal vein
iii.Exfoliative		thrombosis,Systemic amyloidosis
dermatitis		
		Immunologic
8.Amyloidosis		Immunologic
		Immunologic
		5

i. Primary	Immunologic
ii.Secondary	AA protein

Table: 9

2.6. Warning signals of cancer:

? A change in character of pain

- ? Unexplained and persistent or increasing pain in the body
- ? Convulsion originating in adult life
- ? Unremitting continued fever not responding to treatment

? Haemoptysis with out any apparent cause

? A persistent cough

Warning signals of oral cancer and precancerous lesions:

Cancer of the oral cavity is predominantly seen among those who chew tobacco. The red and white coloured patches, non healing ulcers of long duration, ulcers caused by sharp tooth. Inability to tolerate spices along with glistening appearance of the tongue and lining of cheeks especially in tobacco habituates.

Warning signals of breast cancer:

Lump in the breast which is increasing in size and or causing pain and changes in the overlying skin .Red or brown coloured discharge from the nipple or any swelling in the axilla.

Warning signals of uterine cervical cancer:

Bleeding per vagina after intercourse, excess discharge from the vagina, bleeding after attaining menopause and bleeding between menstrual periods.

Head and Neck cancer:

Painless enlargement of lymph nodes in the neck, appearance of tumors or nodules in the neck especially in tobacco habituates and alcoholics.

Gastro intestinal cancer or colorectal cancer:

Bleeding per rectum with altered bowel habits like constipation alternating with diarrhea and passing mucous with abdominal discomfort.

Skin cancer:

Changes in moles or pigmented skin lesions like appearance of fissuring, ridges, furrows and ulceration in the moles, appearance of satellite lesions, itching with bleeding and sudden enlargement of pigmented moles.

Cancer of larynx (Vocal cord):

Progressive hoarseness of voice ultimately resulting in absence of sound especially in a smoker or alcoholic. *Cancer of the Esophagus:*

Progressive dysphagia i.e. difficulty in swallowing solid food and later on leading to difficulty in drinking.

Tumors:

Swelling or enlargement of lymph nodes in the neck, bony swellings or tumors anywhere in the body. *Leukemia:*

Combined symptoms of lymph node enlargement, pallor, loss of weight, fatigue, bleeding gums, weakness, recurrent fever and bone pain. *Non healing ulcers:*

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Non-healing ulcers of long duration anywhere in the body which is not responding to antibiotic treatment may transform to malignancy.

Others:

Bleeding from any orifice like nose, ear, anus, vagina, mouth or from the conjunctiva may be warning signal of cancer in rare cases.

Any individual having any of the above-mentioned warning signals need not panic, as it does not mean that they have cancer. But they should consult a doctor so that they can have the necessary investigations done to rule out cancer.

2.7. Host response against tumor: ³

It has long been thought that host defense mechanism in the form of immunological response exist so as to counter the growth and spread of cancer, albeit more often partially.

- 1) Certain cancers evoke significant lymphocytic infiltrates. E.g.: medullary carcinoma breast, seminoma testis.
- 2) Rarely a cancer may spontaneously regress partially or completely, E.g. malignant melanoma.
- 3) It is highly unusual to have primary and secondary tumor in the spleen due to its ability to destroy the growth and proliferation of tumor cells .
- 4) There is an increased frequency of cancer in immuno deficient hosts, Eg: AIDS.

A) Tumor antigens:

There are two types of antigens,

- i) Tumor specific antigens (TSA) these are located on tumor cells but are not present on normal cells Examples of TSA are ,
 - * Mutant form of ras and p53 proteins and
 - * bcr –abl gene
- ii) Tumor associated antigens (TAA)

These are present on the tumor cells as well as on some normal cells, Examples of TAA are,

- CD -10 antigen on the early B lymphocytes expressed in B –cell leukemia, lymphomas.
- Prostate specific antigen (PSA) expressed by normal as well as malignant prostatic epithelium.

B) Immune responses

The nature of host immune response to tumors can be categorized as under,

i)Cell mediated mechanism:

- a) Specifically sensitized cytotoxic T lymphocytes (CTL) which are directly cytotoxic,
- b) Natural killer cells (NK) destroy tumor cells with out sensitization, either directly or by antibody dependent cellular cytotoxicity (ADCC),

- c) Macrophage mediated cytotoxicity by ADCC or by cytotoxic products,
- ii) Humoral mechanism:

Humoral antibodies are capable of killing free tumor cells in the blood and in the serosal cavities.

- iii) Inhibitory (regulatory) mechanisms:
 - a) CD8+T suppresser cells may play a regulatory role in humoral and cell mediated tumor immunity.
 - b) Humoral blocking factors, possibly antigen –antibody complexes, may either block the antigen sites on the tumor cells or block the receptors on the immune competent cell.

2.8. Diagnosis of cancer ³

The most certain and reliable method which has stood the test of time is the histological examination of biopsy.

Histological methods:

These methods are based on the microscopic examination of properly fixed tissue (excised tumor mass or open needle biopsy from the mass), supported with complete clinical and investigative data. These methods are most valuable in arriving at the accurate diagnosis.

Cytological methods:

Cytological methods for diagnosis consists of study of cells shed in to the body cavities (exfoliative cytology) and study of the cells by putting a fine needle introduced under vacuum in to the lesion (fine needle aspiration cytology -FNAC).

Histochemistry and cytochemistry:

Histochemistry and cytochemistry are additional diagnostic tools, which help in identifying the chemical composition of cells and their products by special staining methods.

Immnohistochemistry:

This is an immunological method of recognizing a cell by one or more of its specific component in the cytoplasm, cell membrane or nucleus .These cell component (called antigens) combine with specific antibodies on the formalin fixed paraffin sections or cytological smears .The complex of antigen –antibody on the slide is made visible for light microscope identification by either fluorescent dyes or by enzyme system. The specific antibody against a particular cellular antigen is now a days obtained by hybridoma technique for monoclonal antibody production. These monoclonal antibodies impart objectivity to the subjective tumor diagnosis made by the surgical pathologist. ³

One important group of such antibody stains is directed against various classes of intermediate filaments which is useful in classification of poorly differentiated tumors of epithelial or mesenchymal origin.

Tumor markers (Biochemical assays):

Tumor markers are biochemical assays of products elaborated by tumor cells in blood or other body fluids, it is there for, a pertinent to keep in mind that many of these products are produced by normal cells too, and thus the biochemical estimation of the product in the blood reflects the total substance and not by the tumor cells alone. Tumor markers include -cell surface antigens (or oncofetal antigens) cytoplasmic proteins, enzymes, hormones and cancer antigens. However two of the best known examples of oncofetal antigens are secreted by fetal tissues as well as by tumors are alpha fetoproteins (AFP) and carcino embryonic antigens (CEA).³.

Modern aids in tumor diagnosis:

In addition to the methods described above some more modern techniques have emerged for pathologic diagnosis but their availability as well as applicability is limited, Eg:,

- i) Flow cytometry
- ii) In situ hybridization
- iii) Molecular diagnostic techniques.³

2.9. Pain

Pain, it has been said is one of natures earliest signs of morbidity, and it stands pre eminent among all the sensory experiences by which humans judge the existence of disease within themselves.¹¹

Pain is mainly a protective mechanism for the body, it occurs whenever any tissue is being damaged, and it causes the individual to react to remove the pain stimulus. 12

The painful experiences of the sick pose manifold problems for the physicians. They must be prepared to diagnose the disease in patients who have felt only the first rumbling of discomfort before other symptoms and signs have appeared. To deal intelligently with pain problems, the physician requires familiarity with the anatomy of sensory pathways and the sensory supply of the body segments, insight into the psychological factors that influence behavior and a knowledge of medical and psychiatric diseases.¹¹

The dual nature of pain is responsible for some of our difficulty in understanding it. Easier to comprehend is its evocation by particular stimuli and the transmission of pain impulse along certain pathways i.e, the sensation of pain. Far more abstruse is its quality as a mental state, i.e. the quality of anguish or suffering – "a passion of the soul" –in the words of Aristotle- which defies definition and quantification.¹¹

Unlike most sensory modalities, which are aroused by a specific (adequate) symptom, such as pressure, heat or cold, pain may be evoked by each of these stimuli, if it is intense enough.

The theory of specificity of sensations proposed at the end of last century maintains that each type of sensation is conveyed by a separate anatomical pathway. This theory has repeatedly been questioned. Sir.Henry Head postulated the existence of two sets of sensory inputs to the central nervous system, the epicritic and protopathic inputs. Epicritic sensation allowed discrimination of touch, temperature and pain, where as protopathic sensation was a poorly localized, unpleasant and long-lasting sensation. The protopathic sensation is physiologically inhibited in the central nervous system by the epicritic system. Later Trotter and Davies dismissed this theory after performing careful examinations on themselves.¹³

Pain has been classified into two major types. **Fast pain and slow pain**.¹²

Fast pain is felt within 0.1 second after a pain stimuli is applied. Where as slow pain begins only after 1 second or more and then increase slowly over many seconds and sometimes even minutes.

When pain is the result of physiologic activity in the normal pain receptors and there is no primary dysfunction of the nervous system, it is called nociceptive pain. Nociceptive pain may indicate a disorder in any other system or organ, whereas pain resulting from the dysfunction of the central or peripheral nervous system is called neuropathic pain.¹³

Pain and temperature are sensations mediated at a primary afferent level by fibers of smaller diameter than the fibers mediating touch, vibration and position sense. Cold sensation is mediated by small myelinated fibers (A delta fibers); warm sensation mediated by unmyelinated warm specific C-fibers and pain is mediated by small myelinated A-delta nociceptor and unmyelinated C- nociceptors.¹³

The anatomical and functional aspect of cutaneous pain afferents have been studied in detail. The skin, subcutaneous tissue, muscles and joints are sensitive to a variety of potentially harmful mechanical, thermal and chemical stimuli. Numerous free nerve endings are responsible for conveying sensory information that is decoded as pain in the central nervous system. Although the ultra structural characteristics of pain receptors (nociceptor) are not well known, classically two types of nociceptors have been characterized in the human skin according to their receptor-response features,

They are 1. Those associated with unmyelinated C-fibers and

2. Those associated with small myelinated A-delta $\ensuremath{\mathsf{fibres}}^2$

The glabrous and hairy skin is richly innervated by nociceptors with unmyelinated C-fiber. These are known as C-polymodal nociceptors (CPN) because they respond to a variety of noxious stimuli i.e, mechanical, thermal and chemical stimuli. A simple C-polymodal nociceptor innervates an area of skin approximately 1cm^2 usually in a single continuous receptive field. These nociceptors have threshold that are well below the level at which tissue damage occurs. These nociceptors also respond to temperature below 15°c.^{13}

Another receptor is the A-delta nociceptor. They mainly respond to mechanical stimulation. These nociceptor display a smaller, usually punctiform receptive field and have higher mechanical and heat threshold than C-polymodal nociceptors.¹³

A third type of cutaneous nociceptor composed of unmyelinated C-fibers has been recently described. These nociceptors are activated only during inflammation. In the absence of inflammation they do not respond even to high noxious stimulation.¹³

The sensations evoked by activation of A-delta and C- nociceptors are different. Excitations of cutaneous C-polymodal nociceptors evoke a burning sensation. Where as A-delta nociceptors evoke a sharp pain that is projected to a punctiform area. 13

In addition to their role in pain sensation, C- polymodal nociceptors are involved in neurogenic inflammation. Excitation of C-polymodal nociceptors determines the release of algogenic substances from nociceptive terminals in the skin, causing local vasodilatation and thus reddening of skin. This flare reaction spreads some centimeters around the site of stimulation through an axonal reflux that depends on a network of fine dermal afferent fibers, originally described as a nocifensor system. Denervation of skin impairs the flare reaction.¹³

The peripheral afferent fibers have their cell bodies in the dorsal root ganglia. Central extension of these nerve cells project via the dorsal root to the dorsal horn of the spinal cord or in the case of cranial pain afferents to the nucleus of the trigeminal nerve, i.e. the medullary dorsal horn. The fine myelinated and unmyelinated fibers occupy mainly the lateral part of the root entry zone and with in the spinal cord many of the thinnest fibers form a discrete bundle the tract of Lissauer.¹¹

The afferent pain fibers after traversing Lissauer's tract terminate in the posterior grey matter of dorsal horn, predominantly in the marginal zone. Most of the fibers terminate with in the segment of their entry in to the cord, but some extend rostrally and caudally to one or two adjacent segment ipsilaterally and some via the anterior commissure to the contra lateral dorsal horn. The neurons in the dorsal horn are arranged in a series of six layers or laminae.

Transverse section through the sixth cervical segment of the spinal cord of the cat, illustrating the subdivision of the gray matter into laminae according to Rexed. LM and VM, later-omedial and ventromedial groups of motor neurons.

Fine myelinated A-delta fibers terminates principally in lamina 1 of Ryxed and also in outermost part of lamina 2, some A-delta pain fibers penetrate the dorsal gray matter and terminate in the lateral part of lamina 5.Unmyelinated C-fibers terminate in lamina 2(substantia gelatinosa).From these cells of termination, secondary neurons connect with central and lateral horn of cells in the same and adjacent spinal segments and sub serve both somatic and autonomic reflexes .Other secondary neurons sub serving pain stem and thalamic structures. The axons from each dermatome decussate one to three segments higher than the level of root of entry. In this way the dorsal horn and anterior spinal commissure form a continuous pain pathway, the full length of the spinal cord. Crossing fibers are added to the inner side of the spinothalamic tract, so that the longest fibres from the sacral segments come to the most superficial and fibers from successively more rostra level occupy a progressively deeper position.¹¹

In addition to the lateral spinothalamic tract the anterolateral fasciculus of the spinal cord contain a more slowly conducting medially placed system of fibers, which project via short interneuron chain to the reticular core of the medulla and midbrain and then to the medial and intra laminar nuclei of the thalamus. This group of fibers is known as the spinoreticulo thalamic or paleo spinothalamic pathway. This tract conducts diffuse poorly localized pain arising from deep structures like gut peritoneum etc.

The direct spinothalamic fibers as they approach the thalamus segregate in to two bundles. The lateral division terminates in the ventro basal and posterior group of nuclei. The medial division terminates mainly in the intralaminar complex of nuclei and in the nucleus submedius. Spinoreticulo thalamic fibers project in to the medial intralaminar thalami nuclei. Projections from the dorsal column nuclei, which have a modulating influence on pain transmission, are mainly to the ventrobasal and posterior group of nuclei. Each of the four thalamic nuclear groups that receive nociceptive projections from the spinal cord has a distinct cortical projection and is thought to play a different role in pain sensation.¹¹

The ventrobasal complex and probably the posterior group of nuclei send their axons to two mass cortical areas, the post central cortex (a small number terminate in the perceptual cortex) and the upper bank of sylvian fissure. The areas are mainly concerned with the reception of tactile and proprioceptive stimuli and with discriminative sensory function including pain.¹¹

Stimuli that activate pain receptors vary from one tissue to another. An adequate stimulus for skin is one that injures tissue, i.e. pricking, cutting, crushing, burning and freezing. These stimuli are ineffective when applied to the stomach and intestine where pain is produced by local effects of an engorged or inflamed mucosa, distension or spasm of the smooth muscle, and traction on the mesenteric attachment. In skeletal muscle pain is caused by ischemia as well as by injuries of connective tissue sheath, necrosis and heamorrhage.¹¹

In the painful lesions due to tissue damage, proteolytic enzymes are released which act on gamma globulins to liberate substances that excite peripheral nociceptors. Bradykinins, histamines, prostaglandins, serotonins and similar polypeptides as well as potassium ion elicit pain. Vascular permeability may also be increased by these substances. In addition, direct stimulation of nociceptor release substance that enhances pain perception. The best studied of these is substance- P, which is released from C-fiber terminals in the skin during peripheral nerve stimulation. It causes erythema by dilating cutaneous vessels and edema by releasing histamine from mast cells. This reaction is called neurogenic inflammation and this is mediated by antichromic action potential from the small nerve cells in the spinal ganglia. ¹¹

In 1965, Melzac and Wall propounded a new theory to explain the mechanism of pain. They observed in decerebrate and spinal cats, that peripheral stimulation of 'large myelinated fibers' produced a negative dorsal root potential and that stimulation of small C fibers caused a positive root potential. They postulated that these potentials which were a reflection of pre synaptic inhibition or excitation modulated the activity of secondary transmitting neuron (T cells) in the dorsal horn, and that this modulation mediated through an inhibitory interneuron (I cell). The essence of this theory is that the larger diameter fiber excites the inhibitory cells; conversely the small pain afferents inhibit the inhibitor cells, leaving the transmitter cells in an excitatory state. Melzac and Wall emphasized that the transmission of pain impulses from the dorsal horn must also be under the control of a descending system of fibers from the brainstem, thalamus and limbic lobes.¹¹

The gate control hypothesis of Melzak &Wall: A stimulus presented to the skin activates both large and small diameter fibers. If the stimulus is light, large fiber input predominates, the inhibitory inter neuron (I) is excited and the transmission cell does not fire. If the stimulus is intense, small fiber input predominates, the inhibitory interneuron is shut off and the transmission cell (T) is activated resulting in pain

The most important contribution in recent years to our understanding of pain has been the discovery of an endogenous neural system for analgesia, which can be activated by the administration of opiates or by naturally occurring brain substances with the pharmacological properties of opiates.¹¹

The endogenous analgesic system was first demonstrated by Reynolds in 1969. This analgesic system consists of three major components, $^{\rm 11}$

1. The peri acquiductal gray and peri-ventricular areas of the mesencephalous and upper pons surrounding the aqueduct of sylvius and portions of the third and fourth ventricles. Neurons from these areas send their signals to **2.** The raphe magnus nucleus, a thin midline nucleus located in the lower pons and upper medulla, and the nucleus reticularis para giganto cellularis located laterally in the medulla. From these nuclei, the signals are transmitted down the dorsolateral column in the spinal cord to **3.** Pain inhibitory complex located in the dorsal horn of the spinal cord. At this point the analgesia can block the pain before it is relayed to the brain. ¹²

Further investigation disclosed that stimulation produced analgesia (SPA) produces its effects by inhibiting the neurons of laminae 1 and V of the dorsal horn, i.e. the neurons that are stimulated by nervous stimuli.¹¹

The opiates also act on the neurons of laminae 1 and V of the dorsal horn, suppressing the inputs from Adelta and C- fibers. There are several opiate receptors in the CNS, they have been found in the spinal cord in the terminals of the A- delta and C fibers in the dorsal horn neurons, as well as in medullary reticular nuclei, medial thalamus and amygdaloid nuclei. The analgesic effects of the opiates are both presynaptic and post synaptic after the discovery of specific opiate receptor in the CNS. Several naturally occurring peptides, which proved to have a potent analgesic effect and bind specifically to opiate receptors, were identified. These endogenous morphine like compounds are known as 'endorphins' meaning morphine within. The most important endorphins in the body are beta endorphin (a fragment of pituitary hormone – beta lipoprotein) and enkephalin, they are found in greatest concentration in relation to opiate receptors in the mid brain.¹¹

Theoretical mechanism of action of enkephalin (endorphin and morphine) on the transmission of pain impulses from the periphery to the CNS is as follows. Spinal inter neurons containing enkephalin synapse with the terminals of pain fibers and inhibit the release of the presumptive transmitter, substance P. As a result, the receptor neurons in the dorsal horn receive less excitatory (pain) impulses and transmit fewer pain impulses to the brain. Morphine binds to the unoccupied enkephalin receptors, mimicking the pain suppressing effects of the endogenous opiate enkephalin.

There are two varieties of enkephalin met-enkephalin and leucoenkephalin. These are found most importantly in the brain stem and spinal cord. Beta endorphins is present in both the hypothalamus and pituitary gland.¹²

Although all the finer details of brain opiate system are not understood, activation of analgesia system by nervous signals entering the peri acquiductal gray and periventricular areas or inactivation of pain pathways by morphine like drugs can totally or almost totally suppress many pain signals entering through the peripheral nerves.

2.10. Cancer pain:

Relief of pain in cancer patients is an ethical imperative and it is incumbent up on clinician to maximize the knowledge, skill and diligence needed to attend this task. Unfortunately under treatment continues to be common. Under treatment have many causes, among the most important of which is inadequate assessment. Assessment is an ongoing and dynamic process that includes evaluation of presenting problems, elucidation of pain syndromes and pathophysiology and formulation of a comprehensive plan for continuing care. In this process pain treatment must be incorporated with in a broader therapeutic agenda, so that needs for tumour control and symptom palliation are concurrently addressed.²

Pain assessment in cancer population begins with an appreciation of the relationship among pain, nociception and suffering.

Nociceptors as we already discussed is the activity produced in the nervous system by potentially tissue damaging stimuli. Nociceptor is not equivalent to pain. There may be no report of pain despite overt tissue injury and should pain occur it may or may not be perceived by the clinician to be commensurate with the degree of injury.²

Pain can be conceptualized as the perception of nociceptors. Like other perceptions pain is determined by an interaction between activity and sensori-neural pathway and other factors- the neuropathic process and the psychological disturbances. Although psychological process can strongly influence the expression and impact of pain, nociceptive and neuropathic factors predominate in the cancer population. Elucidation of the lesion that induce these process is an essential element in the assessment and may alter prognosis, provide an opportunity for selecting the method of treatment.²

Suffering can be defined on the global perception of distress engendered by adverse factors that together undermine quality of life. Pain may contribute to suffering, but numerous other factors such as the experience of other physical symptoms, progressive physical impairment and psychological disturbances may be equally important.²

Factors contributing to pain and suffering and relationship between pain and suffering:²

Nociception		
Neuropathic	psychological	Other physiological process
mechanism	process	physical impairment
		Social isolation
		Family distress
		Sense of financial loss

Analgesia alone may not lessen the suffering and consequently pain therapy is not the sole objective in the supportive care of the cancer patients. Rather pain therapy must be the critical component of a more comprehensive therapeutic plan designed to address the diverse factors that impair quality of life. Frequency with which pain occurs varies with the stage of the disease and with the primary site of the tumour.⁶

; Moderate or severe pain occurs in

 $1/3^{rd}$ (30% - 40%) of the patients at the time of diagnosis More than $2/3^{rd}$ (60% - 100%) 0f patients with advanced cancer ¹⁵

Incidence of pain at primary site of cancer: ¹⁵

Percent of patient with pain	Site of cancer	
More than 80%	Bone, pancreas, esophagus	
71-80%	Lung. Stomach, hepato biliary, breast,	
61-70 %	cervix,ovary ,prostate Oropharynx, colon, brain. kidney/bladder Lymphoma, leukemia, soft tissue	
51-60 %	, ,	

Table: 10

The mechanism of pain in cancer patients can be described in three broad categories – nociceptors, neuropathic process and psychological influences. These pains can be experienced either as an acute attack or as a chronic pain.

This term nociceptive pain is applied when pain is produced by stimulation of specific sensory receptors located in tissue as a result of tissue damage and is associated with an identifiable somatic or visceral lesion. Nociceptive pain that originate from somatic structure is known as somatic pain. They are typically well localized and are described as sharp, throbbing or pressure like.

Pain originating from viscera is known as visceral pain. They are often more diffused and are usually described as gnawing or cramping when due to obstruction of hollow viscus and are sharp, aching or throbbing when due to involvement of organ capsule or other mesentry.

The term neuropathic pain is applied to pain that is caused by peripheral or central nervous system injury.¹⁵

Neuropathic pain is most strongly suggested when a dysaesthesia i.e. abnormal, abnormal unfamiliar pain occurs in a region of motor sensory or autonomic dysfunction that is attributable to a discrete neurological lesion. The diagnosis inferred from the distribution of the pain and identification of a lesion in neural structures that innervate these region.²

Neuropathic pain can be classified according to the presumed site of aberrant neural activity or generator that sustains the pain. They are pain that is caused by 1.Peripheral pain generator and 2. Central pain generator.²

Peripheral neuropathic pain is caused by injury to a peripheral nerve or nerve root and is presumably sustained by aberrant process originating in the nerve root plexus or nerve.²

Neuropathic pain due to a central pain generator include sympathetically maintained pain and a group of syndromes called de-afferentation pain.²

The de-afferentation pain include two sub group, one caused by injury to central nervous system called central pain or thalamic pain and another group precipitated by peripheral nerve injury called phantom pain. Sympathetic pain is caused by injury to sympathetic nerve. It is characterized by burning pain and allodynia and signs and symptoms of sympathetic dysfunction in the affected area like E vasomotor changes – erythema pallor edema

E Pseudo motor changes – sweating

E Trophic changes – thinning of skin and atrophy of subcutaneous tissue ¹⁵

The diagnosis of sympathetic pain has important clinical implication.²

The term psychogenic pain is applied to pain for which there is no physical basis in patients who have other evidence of psychopathology. ¹⁵

When there is no evidence of any psychopathology the term idiopathic pain is used.²

Pain in cancer can be of two types, acute pain and chronic pain.¹⁵

When pain is due to a definable acute injury or illness it is called acute pain. It has definite outlet and its duration is limited and predicted.

This pain is accompanied by anxiety and clinical signs of sympathetic over activity like tachycardia, tachypenea, hypertension, sweating, papillary dilatation and pallor. These are characteristics of a patient obviously in pain.¹⁵

When pain is the result of a chronic pathological process it is called chronic pain.

It has gradual or ill-defined onset, continuos unabated and may become progressively more severe. Patients with chronic pain appear depressed and withdrawn and as there are usually no signs and symptoms of sympathetic over activity. They are frequently labeled as not looking like somebody in pain.

These patients have symptoms of depression with lethargy, apathy, anorexia and insomnia. Personality changes may occur due to progressive alternation in lifestyle and functional ability.¹⁵

For patients with chronic pain of non-malignant origin, the pain is to said to lack positive meaning, where as for patients with chronic pain related to cancer the pain not only lacks positive meaning but it may have definite negative implication with regard to progress and life expectancy.¹⁵

Pain lasting more than two weeks should be considered as chronic and should be treated accordingliy.¹⁵

Evaluation of pain in cancer is based on the following factors,

1, Intensity of pain:

In cancer population evaluation of pain intensity is pivoted to therapeutic decision making. The selection of the medicine, potency and dosage may all be influenced by reported pain intensity. Furthermore intensity may also help characterize the pain mechanism and underlying syndrome. For example clinical observation strongly suggests that the pain associated with radiation induced nerve injury is rarely severe; the occurrence of severe pain in previously irradiated region therefore suggests the existence of occult neoplasm.²

There are various factors that will affect the pain intensity, ⁶

Pain will be increased if there is discomfort insomnia, fatigue, anxiety, fear, anger, sadness, depression and isolation .

Pain will be decreased if there is relief of other symptoms

good sleep, understanding , companionship, creative activity relaxation ,reduction in anxiety and elevation of mood.

2. Quality of pain:

The quality of pain often suggests its pathophysiology. As said earlier, somatic nociceptive pain is usually well localized and described as sharp, aching, throbbing or pressure like. Visceral nociceptive pains are generally diffuse and may be crampy or gnawing when due to obstruction of a hollow viscus or aching sharp or throbbing when due to involvement of organ capsule or mesentry. Neuropathic pains are often described as burning tingling or shock like. ²

1. Distribution of pain:

Patients with cancer pain commonly experience pain at more than one site. The topographic distribution of a specific pain may also have implication for diagnosis and treatment. The distribution of pain often clarifies its relationship to the underlying organic lesion. The term focal pain is used to denote one site of pain or pain that is experienced in the region of the underlying lesion.

The cause of pain in cancer can be grouped into 4 categories⁶.

- 1. Cancer itself
 - Eg: soft tissue, visceral, bone neuropathic.
- 2. Anti cancer treatment
 - Eg: chemotherapy causes mucositis, neuropathy and abdominal cramp
- 3. Debility
 - Eg: constipation muscle tension and sprain.
- 4. Concurrent disorders.

Eg: spondylosis, OA

Cancer related pain could present as an acute pain syndrome.

Cancer related acute pain syndrome is most commonly due to diagnostic or therapeutic intervention, where as chronic pains are mainly tumor related.²

Acute pain associated with diagnostic interventions are mainly due to, lumbar puncture headache, arterial or venous blood sampling, bone marrow biopsy, lumbar puncture, colonoscopy, myelography, per cutaneous biopsy and thoracocentesis.

Acute pain associated with therapeutic interventions is mainly, acute post-operative pain. Acute pain caused by other therapeutic interventions mainly pleurodesis, tumor embolisation, suprapubic catheterization, intercostal catheter and nephrostomy insertion.

Acute pains associated with analgesic technique are injection pain, opioid headache, spinal opioid hyperalgesia syndrome and epidural injection pain.

Acute pain associated with anti cancer therapies like, acute pain associated with chemotherapy infusion techniques that include intravenous infusion pain due to venous spasm, chemical phlebitis, vesicant extravasations, anthracycline associated flair reaction. Hepatic artery infusion pain and intraperitonial chemotherapy causing abdominal pain.

Acute pain associated with chemotherapies toxicity like mucositosis, corticosteroid induced perineal discomfort, steroid pseudo rheumatism, painful peripheral neuropathy, headache -due to intrathecal methotrexate meningitis syndrome, L-asparaginase associated dural sinus thrombosis and trans retinoic acid headache-, diffuse bone pain-due to trans retinoic acid and colony stimulating factors-, 5 –fluorouracil induced anginal chest pain, post chemotherapy gyenecomastia.

Acute pain associated with hormonal therapy like leutinising hormone releasing factor tumor flares in prostate cancer and hormone induced pain flare in breast cancer.

Acute pain associated with immuno therapy like interferon induced acute pain.

Acute pain associated with radiotherapy like incident pain associated with positioning, oropharyngeal mucositis, acute radiation enteritis and proctocolitis, early onset brachial plexopathy and sub acute radiation myelopathy.

Acute pain associated with infection like acute herpetic neuralgia.

2.11. Psychological aspects of Cancer Pain

The cancer patient faces a wide range of psychological and physical stresses throughout the course of illness. These stresses include fear of a painful death, physical disability, disfigurement and growing dependency on others. Although such fears exist in most, if not all cancer patients, the degree of psychological distress experienced varies greatly between individuals and depends on part on the patient's personality style, coping abilities, available social supports and medical factors. One of the most feared consequences of cancer is the pain. Pain has a profound impact on the patient's level of emotional distress and psychological factors such as mood, anxiety and the meaning attributed to pain can intensify a patients experience on cancer pains.²

Patient diagnosed with cancer often demonstrates a consistent pattern of emotional response. These responses usually consists of an initial period of shock, denial and disbelief, followed by a period of anxiety and depression. Disturbed sleep, diminished appetite and concentration, irritability, pervasive thoughts about cancer and fears about the future often interfere with normal daily activities. These stress responses usually occur at specific points in the course of cancer and its treatment - after diagnosis, with relapse , prior to diagnostic tests, surgery, radiation and chemotherapy, as well as after treatment has concluded and patient enters the phase of survival ship. Distress usually resolves slowly over a period of several weeks and patient gradually returns to their prior level of homoeostasis once a treatment plan has been agreed upon and emotional supports arrive.

Not only does pain have a profound impact on psychological distress in cancer patients, but also psychological factors appear to influence the experience and intensity of cancer pain. Psychological variables such as perceived control, meaning attributed to the pain experience, fear of death, hopelessness and anxious or depressed mood all appear to contribute to the experience of cancer pain and suffering.²

Pain is no longer considered simply as a nociceptive event, but is widely recognized and accepted as a psychological process involving nociceptors, perception and expression. Because of the important role played by psychological variables in the experience and intensity of cancer pains, appropriate and effective management of cancer pain requires detailed care taking that covers the mental, emotional and physical characteristics of the patient.²

Uncontrolled pain is a major factor in cancer suicides. Cancer is perceived by the public as an extremely painful disease compared to other medical conditions. The majority of suicides observed among patients with cancer had severe pain, which was often inadequately controlled or tolerated poorly.

Factors associated with increased risk of suicide in cancer patients are ,

- ? Pain -suffering aspects
- ? Multiple physical symptoms
- ? Advanced illness, poor prognosis
- ? Depression, hopelessness

- ? Delirium, dis inhibition
- ? Helplessness
- ? Pre existing psychopathology
- ? Suicide history, family history
- ? Inadequate social support

Recently published reports however suggest that suicidal ideation is relatively infrequent in cancer and is limited to those who are significantly depressed.

2.12. Pain measurement in persons in pain

The new emphasis on the varieties of clinical pain and their variability led to new concept of pain measurement. Instead of using stimuli such as radiant heat to obtain psychophysical standards to measure clinical pain, it became necessary to measure to measure the subjective experience of pain as such without reference to external causes.

The measurement of pain is essential,

- 1. To determine pain intensity, quality and duration
- 2. To aid diagnosis
- 3. To help decide on the choice of therapy
- 4. To evaluate the relative effectiveness of different therapies

Rating scales:

If the study of pain in man is to have a scientific foundation, it is essential to measure it. If we want to know how effective a drug is, we need numbers to say that the pain decreased by some amount.²

In cancer patients, the pain is treated as though it were a single unique quality that varies only in intensity. Therefore the methods of measurement included the use of verbal rating scales (VRS- mild, moderate, severe, excruciating) and numerical rating scale (VAS) (1-10)

VAS provide simple, efficient and minimally intrusive measures of pain intensity which have been used widely in clinical and research setting, where a quick index of pain is required and to which a numerical value can be assigned. The most common VAS consists of a 10 cm horizontal scale with the two end points labeled 'no pain' and 'worst pain imaginable' on one side and I cm interval marking on the opposite side with a sliding block attached, so that the patient can move the block to the area which best describes the pain he is feeling. In the scale, 0 means no pain and 10 is the worst imaginable pain. This distance in centimeters from the low end of the VAS to the patients mark is used as a numerical index of the severity of pain².





Assessment and evaluation of pain

Because pain is multidimensional, it is helpful to think in terms of total pain, encompassing physical, psychological social and spiritual aspects of suffering. The four dimensions of pain:⁶

Physical

;Other symptoms Undesirable effects of treatment Insomnia and chronic fatigue

Psychological Social

Anger at delays in diagnosis Worry about family & Anger at therapeutic failure finances Disfigurement Loss of social position Fears of pain &/or death Loss of role in family Feelings of helplessness Feelings of abandonment and isolation Loss of job, prestige & income

Spiritual

Why has this happened to me? Why does god allow me to suffer like this? What is the point of it all? Is there any meaning or purpose in life? Can I be forgiven for past wrong doing?

Evaluation of pain begins by asking the patient to identify the location of pain, and its duration. Then while the patient goes on describing the pain, the doctor reflects on the,

- 1. Cause of pain
- 2. The underlying mechanism
- 3. The contribution of non physical factors⁵

Description Location Duration PQRST characteristics



Non –physical factors Psycholoical Social

Spiritual

The patient's description of her pain may need to be prompted by a series of questions about the PQRST characteristic of pain,⁶

Palliative factor – what makes it better? Provocative factor – what makes it worse? Quality - what exactly it is like Radiation – does it spread anywhere? Severity – how severe it is

Temporal factor – is it there all time or does it come and go, or is it worse at any particular time of day or night.

In practice it may be better to begin with T and end with P -"TSRQP".⁶

2.13. Homoeopathic aspect of cancer and cancer pain

Homoeopathy is a specialized system of therapeutics developed by Dr.Samuel Hahnemann based on the natural law of healing, 'similia similibus curantur'.

The concept of health and disease in homoeopathy is unique. According to this, health is that balanced condition of the living organism in which the integral harmonious performance of the vital function tends to

the preservation of the organism and the normal development of the system. Disease is an abnormal vital process, a changed condition of life, which is inimical to the true development of the individual and tends to organic dissolution. Vital phenomena in health and disease are caused by the reaction of the vital substantial power or principle of the organism to various external stimuli. So long as healthy man lives normally in a favorable environment, he moves, feels, thinks, acts and reacts in an orderly manner. If he violates this law of life or become the victim of an unfavorable environment, disorder takes the place of order, disease destroy ease, he suffers and his body deteriorates.¹⁶

Dr.Hahnemann in §3 explains the things physician needs to know in order to become a true practitioner of the healing art, and the first thing he says is the physician should clearly perceive what is to be cured in every individual case of disease. In §72 he further says to perceive what is to be cured in disease, the classification of disease will serve as a general preliminary view, and he defines acute and chronic disease as follows, "they are diseases of such a character that, with small, often imperceptible beginnings, dynamically derange the living organism, each in its own peculiar manner, and cause it gradually to deviate from the healthy condition, in such a way that the automatic life energy, called vital force, whose office is to preserve the health, only opposes to them at the commencement and during their progress imperfect, unsuitable, useless resistance, but is unable of itself to extinguish them, but must helplessly suffer (them to spread and) itself to be ever more and more abnormally deranged, until at length the organism is destroyed; these are termed chronic diseases. They are caused by infection with a chronic miasm".¹⁷

In homoeopathy disease are classified in to indispositions, surgical disease and dynamic diseases. Hahnemann in §150 explains indispositions are nothing but those, manifested by one or more trivial symptoms, that have been only observed a short time previously .A slight alteration in diet and regimen will usually suffice to dispel. The dynamic diseases are divided in to acute and chronic as already described. The acute diseases are further classified in to individual, sporadic, endemic and epidemic according to the nature of onset, causation and number of person affected. The chronic disease are also further classified in to artificial chronic disease, inappropriately named chronic disease and true natural chronic disease(miasmatic disease).Artificial chronic disease are those caused by allopathic or antipathic drug overuse and these cases are the most difficult type of cases to cure. Inappropriately named chronic disease are those caused by improper way of living, occupation or habits of long standing duration. These diseases disappear of themselves without any treatment if the maintaining cause is removed. The true natural chronic disease are those arise from dynamic infection of chronic miasm. This true natural chronic disease can present with fully developed symptoms or with few symptoms only. Those with few symptom can be those with only mental symptoms or with physical symptoms, either presenting with few internal symptoms or with external symptoms ,the later variety is called local disease due to causa interna. The chronic disease with fully developed symptoms can be either due to a single miasm (psora, sycosis or syphilis) or due to a combination of miasms (psora+syphilis, psora+sycosis, syphilis+sycosis, psora +syphilis+sycosis)¹⁷. On the basis of this classification we can say that cancer fall under the category of true natural chronic disease.

Hahnemann in §80, while describing the nature of psoric miasm says, the monstrous internal chronic miasm - the psora, the only real fundamental cause and producer of all the other numerous, I may say innumerable, forms of disease, which, under the names of nervous debility, hysteria, hypochondriasis, mania, melancholia, imbecility, madness, epilepsy and convulsions of all sorts, softening of the bones (rachitis), scoliosis and hypnosis, caries, cancer, fungus haematodes, neoplasms, gout, hemorrhoids, jaundice, cyanosis, dropsy, amenorrhea, hemorrhage from the stomach, nose, lungs, bladder and womb, of asthma and ulceration of the lungs, of impotence and barrenness, of megrim, deafness, cataract, amaurosis, urinary calculus, paralysis, defects of the senses and pains of thousands of kinds, &c., figure in systematic works on pathology as peculiar, independent diseases¹⁷. From this it is clear that Hahnemann considered neoplasm to be of psoric origin. This may be because, during Hahnemann's period, not much was known about the etiology, pathogenesis and different types of tumors. At that time this type of long standing cases which rapidly proliferates and which were incurable, eventually causing the death of the patient was considered as something stubbornly sticks to the system like a crab and they named it as 'cancer' means crab. So the concept of cancer as something deep rooted in the system might have made Hahnemann to include cancer under psoric miasm. But considering the clinical presentation, the dominant miasm can be any of the three miasm depending up on the pathological process, their pace and their various expressions.

As already discussed in the aetiopathogenesis of cancer, the cause of cancer can be endogenous factors or environmental factors. The initiating cause of malignant change is a mutation in the gene. So that a normal gene become an abnormal gene. The genetic change may be a deletion or translocation of genes. So there is a deviation from normal functioning of the gene. The cells that undergone these changes deny the rules of normal growth regulation of the body and starts multiplying on its own deriving nutrition from the body. So the abnormal cell with the tendency for excess growth corresponds with the nature of sycotic miasm. As Dr.H.A.Roberts said, "the sycotic patient is too susceptible to the available constructive elements, he seizes up on and assimilate to the point of overgrowth of tissues. It explain the reason of pathology in all parts of the body that manifest overgrowth of natural tissues and where we find malignancies with overgrowth of tissues and infiltration we are almost always able to trace the sycotic trait"¹⁸. If there is a syphilitic trait the manifestations of the tumor will be different. Dr.H.A.Roberts says "one of the cardinal manifestation of syphilitic trait is destruction of tissues"¹⁸. If there is a syphilitic trait, it will present as rapidly progressing destructive change involving the bones with sever night pain, ulcer with punched out edges and glandular involvement. If the psoric miasm is the predominant one, the patient will present with more functional symptom like weakness, anorexia, cachexia etc., than due to structural change.

Even though we have good idea about the aetiopathogenesis of cancer and its miasmatic nature, most of the cancer cases remain incurable.

Dr.Kent in his lesser writings discuss why is cancer incurable. He tells that in most cases there is paucity of symptoms ,and there is nothing discoverable but the malignant growth and its associated features of hardness ,stinging pain ulceration ,enlarged glands and the tendency to involve the surrounding parts in its own development .If the child's mental symptoms could be fully ascertained and the symptom from child hood to the adult age ,some thing might be done .Cancer generally comes on in later life ,when childhood action have forgotten .If the symptoms that have appeared from birth to the present date are undiscovered ,it is no wonder that cancer is incurable.¹⁹

Hahnemann says all curable disease make themselves known to the intelligent physician in signs and symptoms Pathological conditions as also the patient are incurable when there are no signs and symptoms .In proportion as the pathology progress signs and symptoms decreases. This is true in cancer cases. In terminal cancer cases there are paucity of symptoms and even if there is any that are mainly the common symptoms or pathological symptoms.¹⁹

As **Dr. Stuart close** says, the gross tissue changes, organic lesions, morphological disproportion and the physical effect of mechanical causes are not primarily with in the domain of similia and there for are not the object of homoeopathic treatment, the morbid process from which they arise, or to which they lead are amenable to homoeopathic medications. Homoeopathic remedies by virtue of their power to control vital function and increase resistance often exercise a favorable influence upon the physical development as well as up on the tangible products of disease or accident .Thus the growth of the tumor may be retarded or arrested .So the conditions like cancer with gross tissue changes we can't expect a cure and the only thing a homoeopath can do is to palliate the suffering.¹⁶

2.14. Miasmatic aspect of pain:

Hahnemann included most of the pain in to psoric miasm. In § 80 under Psora he says "the monstrous internal chronic miasm - the psora, the only real fundamental cause and producer of all the other numerous, I may say innumerable, forms of disease, which, under the names of nervous debility, hysteria, hypochondriasis, mania, melancholia, imbecility, madness, epilepsy and convulsions of all sorts, softening of the bones (rachitis), scoliosis and kyphosis, caries, cancer, fungus haematodes, neoplasms, gout, haemorrhoids, jaundice, cyanosis, dropsy, amenorrhea, hemorrhage from the stomach, nose, lungs, bladder and womb, of asthma and ulceration of the lungs, of impotence and barrenness, of megrim, deafness, cataract, amaurosis, urinary calculus, paralysis, defects of the senses and **pains of thousands of Kinds, &c.**, figure in systematic works on pathology as peculiar, independent diseases"¹⁷. The symptom pain cannot be included in to one miasm alone. The miasm of pain can only be identified on the basis of its causation, its modifying factors, and the nature of pain.

So the pain of Psora is usually of a neuralgic type, and the pain is better by rest and warmth and worse by motion.

The pain of Sycosis are usually joint pains .The rheumatic pains are worse during cold, damp, better moving or stretching. Stitching, pulsating, wandering pains are sycotic.

The Syphilitic miasm has usually bone pains. Burning, bursting and tearing pain are syphilitic .The pain is aggravated at night²⁰.

2.15. Palliative Care:

Palliative care is the active total care of patients with life limiting diseases, and their families, by a multi professional team, when the disease is no longer responsive to curative or life prolonging treatments.

The word 'Palliative' is derived from the Latin word "pallium"- means a 'cloak'. In palliative care symptoms are 'cloaked' with treatments whose primary action is to promote comfort. However palliative care extends far beyond physical symptom relief. It seeks to integrate physical, psychological, social and spiritual aspects of care, so that the patient may come to terms with their impending death as fully and constructively as they can⁶.

The three essential components of palliative care bound together by the cement of skilled communication. \wedge

Hope

Honesty

Symptom relief Psychological support

Teamwork and partnership

Palliative care is,

- E Patient centered than disease focused
- E Death accepting but also life enhancing
- E A partnership between the patient and the carers
- E Concerned with healing rather than curing

Palliative care neither intentionally hastens nor intentionally postpones death. Palliative care is often said to be "low tech and high touch". It seeks to ensure that compassion and sciences are both governing forces in patient care. Accordingly high tech investigations and treatments are used only when their benefits clearly outweigh any potential burdens. Palliative care is about quality of life. It seeks to help patients achieve and maintain their maximum potential physically, psychologically, socially and spiritually; however limited they have become as a result of disease progression. Quality of life is what a person says it is. Quality of life relates to an individual's subjective satisfaction with life and is influenced by all aspects of personhood, physical, psychological, social and spiritual⁶.

Quality of life issues:²¹

To people living with cancer life is precious, when pain become part of each day of ones life these days are diminished and quality of life is eroded .

The list of damage that pain does to quality of life include,

- i) Sleep is disturbed.
- ii) Ability to work is impaired.
- iii) Exhaustion can become a constant companion.
- iv) Sadness, depression and worry are commonly felt emotions.
- v) Appetite diminished.
- vi) Simple pleasures such as enjoying once family are impaired or given up.
- vii) Trips and vacations are uncomfortable or impossible.
- viii) Reluctance to move or exercise is experienced.
- ix) Feeling of isolation from the worlds increase.
- x) Family and friends who are care givers become exhausted.

It is important to understand too, that the cancer pain undermine patients ability to fight to the cancer .If pain has its grip on him ,his appetite diminishes ,this means he may not be receiving sufficient nutrition to retain energy which in turn leads to exhaustion and feelings of sadness and depression. As the cycle continues a person is worn down gradually may become more vulnerable to infection and the ability to with stand necessary cancer treatment diminishes.

There is good quality of life when the aspiration of the individual are matched and fulfilled by present experience. To improve quality of life, it is necessary to narrow the gap between aspiration and what is possible. Palliative care aims to do this. 6

2.16. Palliation and Homoeopathy:

When faced with incurable disease, the thought occurs to great many physician to administer palliative medicines (antipathy) in an effort to alleviate the suffering and to attempt to hide from the patient and from the family the real seriousness of the situation .Although they may mean well, it is an effort explained in the wrong direction, and does more harm than can well be estimated.¹⁸

The physician who applies the single remedy in potentized form under the law of cure any length of time will easily be convinced that there is no other way of palliation that holds out any permanent hope for the patient.²²

Dr.H.A.Roberts holds the same opinion as Kent, he says, the basis of cure is the fundamental law of similars .The law of similars is the fundamental law also to the palliation of incurable states.¹⁸

When we are facing the incurable condition the administration of the similar remedy almost always ameliorates the situation, at least for three or four days and usually for a longer period. Then we may have a return of the symptom, when the indicated remedy will be called in to use again. Some times one symptom or a set of symptoms predominates and become the annoying, troublesome, disagreeable symptom complex. In these conditions we must retake the case and re-examine the remedy that have been using, to see if it corresponds with the disease condition. If the similarity exists in these especially troublesome manifestations, these patients can be made much more comfortable.¹⁸

In incurable cases or seemingly incurable cases we must not put a limitation on the possibilities of the similar remedy, for in many seemingly incurable condition, the similimum will so completely meet the situation as to obliterate the symptomatology of disease and pathology and will restore the patient to health.

Pain is one of the experience from which human life has ever strives to free itself. Pain in itself is a part of symptom, but for the physician ,he must take in to consideration the location, the kind of pain-whether steady or intermittent and if intermittent ,whether at regular interval or up on motion, or is it dull ,cutting ,blunt ,sharp, pressing or cramping, the time and circumstances of aggravation and amelioration, the reaction to thermic condition, and all the concomitant symptom that can be found. When the symptom of the pain itself is complete with the location, type, aggravation and amelioration and concomitant the picture is almost complete and we have a sound basis for the selection of a remedy which will relieve the pain promptly and the patient will be much more comfortable and happy in general than with any narcotics.¹⁸

2.17. General Management of the Cancer Patient

'Patient care' involves not only the medicines but also various other factors if we consider the holistic approach towards health.²³

These factors are -

- 1. Diet
- 2. Psychological care
- 3. Surgery
- 4. Infusions and transfusions

1. Diet:

In the fight against cancer, diet plays a very important role. The dietary advice to a patient suffering from cancer would be as follows,

a. Vegetables: All vegetables are good, preferably raw or as lightly cooked as possible. To avoid loss of water-soluble vitamins, wash the vegetables before cutting them into pieces, steam them and do not overcook.

b. Fruits: All fresh fruits are good. Avoid canned fruits because they contain large quantities of sugar.

c. Animal foods: Avoid all red meat. Eat only fish and chicken with the skin removed.

d. Peas, beans, lentils, seeds, nuts, legumes: They are good source of protein and should be used. Fresh vegetable oils should be used for cooking. Spices and natural flavorings can be used. Seed sprouts should be taken liberally.

e. Cereals: Use whole grains. Millet is especially good as it is rich in nitrilasides.²³

2. Psychological care:

The word cancer strikes terror in the heart of both - the patient and his relatives, and the "cancer phobia" proves a greater psychological stress than the disease itself.

It is often that the physician and the relatives, therefore, do not wish to let the patient know of the disease that he is suffering from. But patients who are aware of the disease, its prognosis and the chances available by way of therapeutic management respond better to treatment. Those who are left in the dark remain insecure and thus lack a positive attitude, which hinders their management. This does not mean that the physician must indiscriminately tell all the patients about the diagnosis of cancer. He has to use his knowledge of human nature and decide accordingly.

The patient also has a right to choose the mode of treatment. It is the duty of the physician to explain the various modes available - their effects and their side-effects to the patient and his relatives and guide them

in making a choice. This decision-making also becomes important in a patient who is in the terminal stages when the introduction of supportive measures would only prolong the agony.

The Hospice movement seeks to help the terminally ill patients by providing them with the opportunity to discuss their feelings and fears about death. Patients are able to cope better in the face of death through prayers and spiritual discourses.

In patients who undergo surgery, chemotherapy or radiotherapy, the disability and disfiguring that occurs subsequently may become stressful. Patients can cope better if they are prepared beforehand about these occurrences. The feelings of dependence, of being a burden, of not being able to function-all have to be tackled sensitive y by the physician and the patient's relatives.

Lastly, the physician must tackle his own feelings. He should seek to palliate to the best of his abilities when cure is not possible. An attitude of hopelessness and despair in the physician would have undesirable consequences in his management of the patient²³.

3. Role of surgery:

Surgery is advisable under the following circumstances,

i. The tumor mass presses upon vital structures to grossly impair the body functions.

Example:

* Space-occupying lesion in the brain.

* Tumor pressing upon ureters leading to obstructive uropathy.

* Tumor in pharynx/larynx which causes dysphagia and threatens to block the respiratory passages.

ii. Gastrostomy: - done to maintain nutrition when the gastro-

Intestinal tract functions well.

iii. Central venous access: - done to maintain parentral nutrition when the gastro-intestinal function is inadequate. It is usually done through the subclavian or external jugular vein.

iv. Tracheostomy: - done to maintain the patency of the respiratory passage when the tumour blocks the upper respiratory passage.

v. Ascitic and pleural tapping done to instantly relieve the distress caused by accumulation of large quantities of fluid.

Whenever surgery is used as a therapeutic mode for a cancer patient, we must remember that it only takes care of the expression of the disease and never of the disease itself. Hence homoeopathic treatment must also be given simultaneously²³.

4. Infusions and transfusions:

Intravenous fluids to maintain the fluid and electrolyte balance; packed cell transfusions to treat the anemia usually when the hemoglobin is below 6 gm%; platelet transfusion to treat the chronic thrombocytopenia following chemotherapy - may be used as and when required²³.

2.18. Rubrics for cancer and Cancer pain in Murphy's Medical repertory ²⁴

CANCER, general

acet-ac, alum, alumn, anan, anil, ant-chl, ant-m, apis, ambr, apoc, arg-m, arg-n, ARS, ars-br, ars-i, aster, aur, aur-ar, aur-i, aur-m, aur-m-n, aur-s, bapt, bar-c, bar-i, bell, bell-p, bism, BROM, bufo, CADM-S, cadm-i, cadm-m, calc, calc-i, calc-s, calen, carb-ac, CARB-AN, carbn-s, carb-CARC, caust, chel, chol, cic, cinnam, cist, cit-ac, clem, croc, crot-V. h, **CON**, *cund*, *cupr*, *dulc*, *elaps*, *eucal*, *euph*, *ferr-p*, *form*, *gali*, *graph*, *ham*, *hep*, *hippoz*, **HYDR**, hydr-ac, *iod*, *kali-ar*, *kali-bi*, kali-chl, *kali-cy*, *kali-i*, *kali-p*, *kali-s*, *kreos*, *lach*, *lap-a*, **LYC**, maland, med, merc, merc-i-f, mill, morg-g, morph, nat-m, NIT-AC, ol-an, op, ozone, petr, ph-PHOS, PHYT, psor, plb, rad-br, sedpic-ac, sang, scirr, sec, ac, r, semp, sep, SIL, squil, sol, sulph, sul-ac, symph, syph, strych-g, tarax, tax, ter, thuj, TRIF-P, viol-o, visc, x-ray, zinc

ANUS, cancer cund, ruta

AXILLA, cancer ASTER, carb-an, con, phyt

Axilla, cancer burning, towards axilla carb-an

Axilla, cancer indurated glands, with aster, carb-an, con

Axilla, cancer lancinating, pains, with Aster

www.similima.com

BLADDER, cancer

con, crot-h

BONE, cancer

aur-i, aur-m, cadm-m, calc-f, con, hecla, phos, symph

BRAIN, cancer

acet-ac, arn, *ars, ars-i*, art-v, *bar-c*, bell, *calc*, carb-ac, *carb-an*, caust, **CON**, *croc*, gels, glon, graph, hydr, hyper, *kali-i*, *kreos*, *lach*, merc, nit-ac, nux-v, **PHOS**, *plb*, sep, *sil*, stram, sulph, *thuj*, *tub*

BREAST, cancer

alum, alumn, anag, apis, arg-n, arn, ars, ars-i, ASTER, aur-ar, aur-m-n, bad, bapt, bari, bell, bell-p, brom, bry, BUFO, calc, calc-i, calen, carb-ac, CARB-AN, carbn-s, carb-v, carc, caust, cham, chim, cic, cist, clem, coloc, CON, CUND, dulc, ferr-i,form-ac, gali, GRAPH, hep, hydr, kali-c, kali-i, kreos, lach, lap-a, lyc, MERC, merc-i-f, nat-cac, nitac, ol-an, ox-ac, phos, PHYT, plb-i, psor, puls, sang, scirr, semp, sep, SIL, sulph, strychg, tarent-c, thuj, tub

Breast, cancer bleeding carb-an, kreos, lach, phos, sang, strych-g, thuj

Breast, cancer bloodly discharge from livid red spot on tumor, gradually invading *aster*

Breast, cancer burning, pains, better from external warmth ARS

Breast, cancer burning, pains, better from external warmth open tumor APIS, ars, *hydr*

Breast, cancer burning of edges, with bad odor hep

Breast, cancer contusion, from *bell-p*, **CON**, phyt

Breast, cancer cracked tissue, with cund

Breast, cancer discharge of blood and fetid ichor from livid red spot on tumor *aster*

Breast, cancer drawing pain toward axilla carb-an

Breast, cancer emaciated and cachectic ars, cund, hydr

Breast, cancer epithelioma

arg-n, ars, ars-i, brom, **BUFO,** calc, calc-p, *clem,* **CON,** *hydr, kreos, lach,* merc, *merc-i-f, phos, phyt, sep, sil,* sulph, thuj

Breast, cancer face gray, earthy, oldish, with BROM

Breast, cancer hen's egg in, size of hydr, phyt

Breast, cancer heaviness of breast, with con

Breast, cancer hidden, occult BUFO

Breast, cancer hot, nipples, with phos

Breast, cancer immovable mass con

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Breast, cancer indurated brom, cist, con, phos Breast, cancer indurated stony hard CON Breast, cancer inflamed, hard, very painful, worse by exposure to air phos Breast, cancer injury, caused by bell-p, con, phyt, symph Breast, cancer itching, with sil Breast, cancer lancinating pains ASTER, lach Breast, cancer lancinating pains bleeding easily phos Breast, cancer large as a small egg, as hydr Breast, cancer left breast aster, carb-ac, carb-an, carc, con, cund, hydr Breast, cancer left breast feels drawn in aster, carb-an, hydr Breast, cancer left breast ulcerated and open hydr Breast, cancer mastectomy, after calen, graph, sil Breast, cancer mastectomy, after cancer appears in the other breast, after lach, lac-c, lyc Breast, cancer nightly, pains aster Breast, cancer open tumors APIS, ars, bufo, carb-an, cund, hydr, phyt, sil Breast, cancer pains, with ars, aster, hydr Breast, cancer raw feeling MERC Breast, cancer right apis, carc, cund, hydr, lyc, phyt Breast, cancer right open, with burning pain hydr Breast, cancer scars, cancer in old GRAPH, sil Breast, cancer scars, cancer in old abscesses, after repeated GRAPH Breast, cancer sharp pains aster, clem, con Breast, cancer sharp pains shoulders and uterus, pains with clem Breast, cancer shooting pains con Breast, cancer skin, purple spots and wrinkled cund Breast, cancer skin, purple spots and wrinkled red spots over tumor aster, carb-an Breast, cancer sleep from pain, cannot aster, carc Breast, cancer smells like old cheese hep Breast, cancer sore pain MERC Breast, cancer stinging pain apis, hep Breast, cancer stinging of edges, smells like old cheese hep

Breast, cancer stony hard, large as tea cup CON

Breast, cancer ulceration calc, cund, hep, hydr, phos, PHYT, SIL, sulph

Breast, cancer, scirrhus

arg-n, ars, aster, brom, **BUFO**, carb-an, **CON**, cund, hydr, kreos, lap-a, phyt, sars, scirr, sil, sulph

Breast, cancer, scirrhus burning pain, with sep

Breast, cancer, scirrhus discharge, ulceration with fetid, bloody and sloughing cund

Breast, cancer, scirrhus hard as cartilage and uneven, which has grown to size of con Breast, cancer, scirrhus heaviness con

Breast, cancer, scirrhus injury, caused by bell-p, con, hyper

Breast, cancer, scirrhus left, of carb-ac

Breast, cancer, scirrhus left, of occasionally twitching in affected part, them con

Breast, cancer, scirrhus left, of very painful, worse in cold weather and during clem

Breast, cancer, scirrhus painful of right, about an inch in diameter, hard but mo chim

Breast, cancer, scirrhus purple, skin, in spots and wrinkled cund Breast, cancer, scirrhus right, in cund

Breast, cancer, scirrhus right, in adhering by entire base to thoracic walls *aster*

Breast, cancer, scirrhus sharp shooting pain con

Breast, cancer, scirrhus skin and axillary glands involved cund

CERVIX, cancer of uterine cervix aur, calth, carb-an, carc, **CON**, hydr, iod, kreos, thuj, ust

Cervix, cancer of uterine cervix os of cervix *aur, carb-an,* hydr, nux-v, plat, podo, ust

Cervix, cancer of uterine cervix pessary, after use of hyper

CHEMOTHERAPY, drugs, for side effects ars, **CADM-S**, chin, *ip*, nux-v, phos

CLAVICLES, cancer, fungus haematodes sep

Contusions, cancer after bell-p,*con*, phyt, symph

Deposits, removal of, cancer after kali-p, maland

www.similima.com
Emaciation, with cancer,

acon, ARS, cadm-s, carc, graph, HYDR, pic-ac, thuj

EPITHELIOMA,

acet-ac, arg-m, *ars*, **ARS-I**, ars-s-f, aur, aur-ar, *bell*, brom, calc-sil, carb-ac, carb-an, *carc*, clem, **CON**, *cund*, euph, fuli, *hydr*, *hydrc*, kali-ar, *kreos*, *lach*, *lap-a*, **LYC**, merc, merc-c, nectrin, nit-ac, phos, *phyt*, rad-br, raja-s, *ran-b*, ran-s, scroph-n, sep, *sil*, *sol*, sulph, *thuj*, uran-n

ESOPHAGUS, cancer

con, cund, hydr

EYES, cancer

aur-m-n, CALC, carb-an, carc, con, cund, hep, lach, lyc, PHOS, sep, sil, thuj

Eyes, cancer fungus

bell, CALC, lyc, PHOS, sep, sil, thuj

Eyes, cancer fungus medullaris

bell, CALC, lyc, sil

Eyes, cancer lachrymal glands *carb-an*

Eyes, epithelioma cund, *lach*

Eyes, epithelioma eyelids

cund, hydr, lach, phyt, ran-b, thuj

FACE, cancer

ARS, *aur, carb-an,* cinnb, *con, kali-ar,* kali-c, kali-i, lach, nit-ac, *phos,* sil, sulph, symph, thuj, zinc

Face, cancer antrum aur, symph

Face, cancer epithelioma ARS, cic, con, hydr, kali-ar, KALI-S, lach, lap-a, phos, sep, sil

Face, cancer lupoid *hep*, syph

Face, cancer lupus, from

alumn, *arg-n,* **ARS,** aur-m, carb-ac, *carb-v,* cist, **HYDRC,** kali-ar, *kali-bi,* kalichl, kreos, lach, psor,*sep, sil*

Face, cancer lupus, from near wing of nose aur

Face, cancer open, bleeding cist

Face, cancer scirrhus *carb-an*, sil

Fear, of cancer agar, ARS, CALC, calc-f, calc-p, CARC, chin-ar, ign, *kali-ar, med, nit-ac*, PHOS, PSOR, ruta

Fungus, haematodes

ant-t, **ARS**, bell, *calc*, **CARB-AN**, *carb-v*, clem, *kreos*, **LACH**, *lyc*, *merc*, *nat-m*, *nit-ac*, **PHOS**, *puls*, sep, **SIL**, staph, *sulph*, **THUJ**

GENITALIA, cancer, female

arg-m, ars, ars-i, aur-m-n, bell, bov, calc-ar, calc-o-t, calc-s, *calth, carb-an,* carc, cham, chin, *con, graph, hydr,* iod, irid, *kali-bi,* kali-p, kali-s, *kreos,* lach, *lap-a,* mag-p, med, murx, *phos,* phyt, rhus-t, *sec,* sep, *sil,* staph, sulph, tarent, thlaspi, *thuj,* tril, zinc

Genitalia, cancer, female bleeding, with

bell, crot-h, kreos, lach, sabin, thlaspi, ust

Genitalia, cancer, male

ars, bell, carb-an, CON, phos, phyt, sil, spong, thuj

Genitalia, cancer, male

ars, bell, carb-an, CON, phos, phyt, sil, spong, thuj

GLANDS, cancer

ars-i, ASTER, aur-m, brom, bufo, calc-f, CARB-AN, CARC, cist, CON, iod, PHYT, SCROPH-N, sul-i, syph

Glands, cancer adenocarcinoma

ars, aur-m, bufo, carb-an, carc, con, phyt

HODGKIN'S disease

acon, acon-I, ars, ars-i, bar-i, buni-o, calc-f, carc, cist, con, cund, ferr-pic, iod, kali-m, natm, ph-ac, phos, **PHYT**, saroth, **SCROPH-N**, syph, thuj, tub

INTESTINES, cancer, colon *alum, ars,* carb-v, graph, **HYDR,** *kali-c,* lyc, mur-ac, *nit-ac,* ruta, sep, spig

Intestines, cancer, colon sigmoid colon

hydr, ruta, scroph-n, semp

JAW, cancer

ant-c, arg-n, ars, aur, calc, fl-ac, graph, HECLA, merc, phos, rhus-t, sil, symph

Jaw, cancer bones

hecla, symph

LARYNX, cancer

ars, ars-i, bell, carb-an, clem, *con,* hydr, iod, kreos, lach, morph, nit-ac, phos, *phyt,* sang, thuj

LEUKEMIA, blood

acet-ac, acon, aran, **ARS**, ars-i, bar-i, bar-m, bry, calc, calc-p, carbn-s, carb-v, **CARC**, cean, chin, chin-s, con, cortiso, crot-h, ferr-pic, ip, kali-p, merc, **NAT-AR**, nat-m, nat-p, **NAT-S**, nux-v, op, phos, pic-ac, sulfa, sulph, syph, thuj, tub, x-ray

Leukemia, blood acute

ars, lach, merc, merc-c, nat-m, nit-ac, phos

Leukemia, blood acute children, in ARS

Leukemia, blood constitutions broken down by gonorrhea, syphilis, alcohol, etc. *crot-h*

Leukemia, blood gonorrheal *thuj*

Leukemia, blood lienalis kali-p

Leukemia, blood leucocythemia bell, cean, con, iod, lyc, merc, nit-ac, phos, rhus-t, sulph

Leukemia, blood lymphoid ARS, ars-i, carbn-s, carb-v, CEAN, kali-s, mur-ac, nat-ar, nat-m, PHYT, pic-ac, thuj

Leukemia, blood spleen, involvement ars, *cean*, nat-m, nat-s, querc, succ

LIPS, cancer

acet-ac, ars, ars-i, aur, aur-m, camph, carbcist, clem, CON, CUND, hydr, kali-chl, kalian, caust, cic, com, s, kreos, lach, lyc, phos, phyt, sep, sil, sulph, tab, thuj

Lips, cancer epithelioma

cic, con, hydr, lap-a, sep

Lips, cancer lower lip ant-chl, ars, cist, clem, con, CUND, dulc, lyc, phos, SEP, sil

Lips, cancer pressure of pipe con, cund, sep

Lips, cancer ulcers ars, aur-m, carb-an, clem, CON, cund, kali-bi, lyc, phos, phyt

LIVER, cancer

ars, carc, chel, chlol, chlor, chol, con, echi, HYDR, lach, myric, nat-s, nit-ac, phos, ther

Liver, cancer burning pain, with ars, bell, carb-an, con, hydr, lyc, sep, sil, tarent

Liver, cancer early carc, hydr, senec

Liver, cancer jaundice, with myric

Liver, cancer right lobe smooth, painless, can be felt reaching over arch of ribs cund

Liver, cancer scirrhus bor

LUNGS, cancer ars, calad, cob, con, phos

Lungs, cancer smoking, from ars, calad, con, phos

MELANOMA

arg-n, ars, card-m, cund, lach, ph-ac, sol

melanoma sunlight, from

carc, sol

Miasm, cancer ars, ars-i, CARC, cadm-s, con, hydr

MOUTH, cancer, palate aur, hydr

Mouth, cancer, palate hardness, with hydr NECK, cancer cist, con, hydr, merc, scroph-n

Neck, cancer glands, of cist, con, scroph-n

Noma, cancer alum, alumn, *ars,* calc, carb-v, *con,* elat, kali-chl, *kali-p,* merc, sil, sulph, tarent-c

NOSE, cancer

alumn, ars, AUR, aur-m, calc, carb-ac, carb-an, cund, kreos, merc, phyt, sep, sulph

Nose, cancer epithelioma

ars, ars-i, carb-ac, cund, hydr, KALI-S, kreos

Nose, cancer epithelioma nose wing med

Nose, cancer flat, on right side euphr

Nose, cancer noli me tangere, on nose cist, jug-c, phyt, thuj

OVARIES, cancer

ars, CON, graph, kreos, lach, med, psor, thuj

PAINS, from cancer

acon, anthr, *apis*, **ARS**, aster, aur, bry, bufo, calc, *calc-ar*, *cadm-s*, carban, **CARC**, cedr, cinnam, cit-ac, *coloc*, *con*, cund, echi, *euph*, **HYDR**, mag-p, merc, morph, op, ph-ac, sil, sol

PALATE, cancer aur, hydr

Palate, cancer hardness, with hydr

PANCREAS, cancer ars, calc-ar, CON, HYDR, phos

PAROTID, cancer merc, phyt PENIS, cancer, glans, epithelioma on excrescences arg-n, ars, con, thuj

PHARYNX, cancer cist

PROSTATE, cancer

carc, CON, crot-h, cop, iod, plb, psor, sel, senec, sil, sulph, THUJ

Prostate, cancer induration, with CON, cop, iod, plb, psor, sel, senec, sil, sulph, THUJ

Prostate, cancer pain, with carc, con, crot-h, sabal

Radiation, sickness, for side effects ars, CADM-S, calc-f, chin, fl-ac, *ip*, nux-v, phos, rad-br, SOL, x-ray

RECTUM, cancer

alum, ars, carb-v, graph, **HYDR,** *kali-c,* laur, lyc, mur-ac, nat-s, *nit-ac,* phyt, *ruta,* sang, *sep,* spig, tub

Scirrhus, cancer

alumn, anac, arg-m, arn, ars, ars-s-f, aster, bell-p, calc-s, calen, **CARB-AN**, carbn-s, carb-v, clem, **CON**, graph, hydr, lap-a, med, nux-v, petr, phos, phyt, sep, **SIL**, squil, staph, sulph

SCROTUM, cancer

ars, carb-an, fuli, ph-ac, thuj

Scrotum, cancer epithelioma, of carb-an, ph-ac

Scrotum, cancer scirrhus carb-an

SKIN, cancer, epithelioma

acet-ac, alum, alumn, arg-m, arg-n, *ars*, **ARS-I**, ars-s-f, aur, aur-ar, *bell*, brom, calc, calc-p, calc-sil, carb-ac, carb-an, *carc*, chr-ac, cic, clem, **CON**, *cund*, euph, fuli, *hydr*, *hydrc*, kali-ar, kali-chl, kali-m, *kali-s*, *kreos*, *lap-a*, **LYC**, mag-m, mag-s, merc, merc-c, nat-m, nectrin, nit-ac, phos, *phyt*, puls, rad-br, rad-br, raja-s, *ran-b*, ran-s, scroph-n, sep, *sil*, strych-g, **SOL**, sulph, *thuj*, uran-n **Skin**, **cancer**, **epithelioma flat**

cund

Smoking, cancer from tobacco

ars, calad, cob, con, phos

SPLEEN, cancer ars, bor, cean

Spleen, cancer scirrhus bor

Sternum, cancer sulph

STOMACH, cancer

acet-ac, am-m, arg-n, **ARS**, ars-i, bar-c, bell, **BISM**, **CADM-S**, calc-f, caps, **CARB-AC**, **CARB-AN**, carb-v, **CON**, crot-h, **CUND**, form-ac, graph, **HYDR**, iris, kali-bi, kali-c, kreos, lach, **LYC**, mag-p, merc-c, mez, nux-v, **PHOS**, plat, plb, sec, sep, sil, staph, sulph

Stomach, cancer hiccough, with carb-an

Stomach, cancer pylorus acet-ac, graph

Stomach, cancer vomiting, from

cadm-s, carb-ac, kreos

Submaxillary glands, cancer

anthr, calc-s, carb-an, ferr-i calen, carc, lac-c, lach

TESTES, cancer

aur, carb-an, clem, CON, fuli, phyt, sil, spong, thuj

THROAT, cancer

carb-an, led, tarent

THYROID, cancer calc-i

TONGUE, cancer

alumn, apis, ars, aur, aur-m, benz-ac, calc, carb-an, caust, con, crot-h, cund, gali, hydr, kalichl, **KALI-CY**, kali-i, lach, mur-ac, nit-ac, phos, phyt, semp, sep, sil, sulph, thuj

Tongue, cancer epithelioma

ars, carb-ac, chr-ac, hydr, kali-cy, mur-ac, thuj

ulcers, cancerous ambr, anthr, ant-c, apis, ARN, ARS, ars-i, ARS-S-F, aster, aur, aurar, aur-i, AUR-S, bell, BUFO, calc, calc-s, carb-ac, carb-an, carbn-s, carb-v, caust, chel, CON, CUND, crot-c, dor, dulc, flchim, chin-s, clem, ferr, HEP, gali, hydr, kali-ar, kali-c, kaliac, fuli, graph, hippoz, lach, LYC, lyss, mang, merc, merc-i-f, mill, i, kreos, mur-ac, nit-ac, petr, phac, phos, phyt, rhus-t, rumx, sars, sep, SIL, spong, squil, staph, SULPH, sul-ac, suli, syph, tarent-c, thuj, zinc

Ulcers, cancerous contusions, from, with burning stitches con

Ulcers, cancerous lancinating

merc

Ulcers, cancerous particularly painful in morning, burning at and in margin ars

UTERUS, cancer

apis, ARS, ARS-I, AUR-Malum, alumn, anan, arg-m, arg-n, aur, carb-ac, carbn-s, Ν, bell, bov, brom, bufo, calc, carb-an, carbv, carc, chin, cic, clem, CON, crot-h, cund, elaps, fuli, GRAPH, HYDR, iod, kali-ar, kaol, KREOS, LACH, lap-a, LYC, mag-m, med, merc, merc-i-f, MURX, nat-c, nat-m, nitac, PHOS, phyt, plat, rhus-t, ruta, sabin, sang, sars, sec, SEP, SIL, staph, sulac, sulph, tarent, THUJ, zinc

Uterus, cancer bleeding, with kreos, med, phos, thlaspi, ust

Uterus, cancer scirrhus

alumn, anan, *arg-m*, *ars*, aur, aur-m-n, **CON**, kreos, lyc, mag-m, phos, *phyt*, rhust, sep, staph

VAGINA, cancer ars, CON, KREOS

Vagina, cancer labia, vulva, cancerous ars, con, thuj

Weakness, with cancer ars, aur, aur-m-n, CADM-S, CARC, CON, HYDR, kreos, phos, phyt

Weakness, with cancer cancer, after ars, carc, hydr

Weakness, with cancer chemotherapy, after cadm-s

Weakness, with cancer radiation therapy, after cadm-s

EMACIATION, body stomach, cancer, with ars, *cund*, **HYDR**, mez

FOOD

APPETITE, diminished cancer patients, in ALF, ars, cadm-s, hydr

Appetite, loss, of appetite cancer patients, in ALF, ars, *cadm-s*, carc, *hydr*

GENERALS

ANXIETY, (physical) pains, from the cancer, of ars, CARC

MIND

ANXIETY, pains, from the cancer, of ars, CARC

DEPRESSION, (sadness), cancer, with

carc, con **FEAR, cancer, of** agar, **ARS,** bar-c, **CALC,** calc-f, calc-p, **CARC,** chin-ar, ign, *kali-ar,* med, *nitac,* **PHOS, PSOR,** ruta

FEAR, death, of cancer, from ars, carc

SUICIDAL, cancer history in family, with carc

(Bold Capital - 3 mark: Italics- 2 mark: Roman- 1 mark)

Materials and Methods

The present study was carried out at the Government Homoeopathic Medical College, Kozhikode, from August 2003 to February 2005. All patients with cancer pain coming to the O.P.D and I.P.D were selected for the study.

Inclusion criteria: Cancer patients who complaints of pain were taken for the study.

The study was made in all age group

The study was made in both sexes.

Exclusion criteria: Cancer patients without pain were excluded

The case history of the patients thus selected were taken according to homoeopathic philosophy with a detailed enquiry in to the pain characteristics like time of occurrence, severity and duration of pain, any radiation of pain, the quality of pain and finally the aggravating and ameliorating factors of pain . After

completing the case the patients were then asked to rate their pain by using verbal rating scale and visual analog scale. Then they were assessed for the quality of life using degree of distress score and W.H.O performance status classification. In the selection of medicines the approach was to individualize each case based on homoeopathic principles. The miasmatic background was given due consideration while selecting the medicine.

Repertorisation was based on Kent's Repertory of the homoeopathic materia medica and Boenninghaussen's Charecterestics materia medica and repertory by C.M.Boger.

Repetition and change in potency was based on homoeopathic principles.

Pain, performance status and degree of distress had assessed objectively and subjectively and scored. Effectiveness has assessed on the basis of change in the scores.

Out patients were reviewed every 2 weeks or until the pain get palliated, and the in patients were reviewed every day till they got discharged.

The method of approach was a clinical study without the use of control.

Assessment tool was developed after literature review. Three major areas were identified as important parameters and each item were rated.

Assessment criteria include:

Ø Pain Sore

Ø Degree of distress score

Ø W.H.O performance status score

Pain scoring:

Pain was measured using verbal rating scale (Simple descriptive pain intensity scale) and visual analog scale or numeric analog scale. Each terminology on the verbal rating scale was explained to the patient before assessment, and they were asked to classify their intensity of pain they experienced daily. They were also explained how to use the visual analog scale to numerically rate their pain as already described. 0 for no pain and 1 to 10 for increasing severity as given by the patient²⁵.

Degree of distress score²⁶:

Questions	Response	Point
1. Did you feel sick to day?	Not at all	1
	Occasionally	2
	A lot	3
	All of the time	4
2. Did you vomit to day?	Not at all	1
	Once	2
	Twice	3
	More than twice	4
3.How good has your	Good	1
Appetite been today?	Fair	2
	Poor	3
	Bad	4

4.How much pain have you	None	1
had today?	A little	2
	Quite a lot	3
	A lot	4
5.How did you sleep last	Very well	1
Night?	Quite well	2
	Badly	3
	Not at all	4
6.How is your bowel	No distress	1
pattern today?	Low distress	2
	Moderate distress	3
	High distress	4
7.How happy have you	Нарру	1
been today?	Fairly happy	2
	Un happy	3
	Very unhappy	4
8.How are you feeling	Well	1
today?	Fair	2
	Poor	3
	Very poor	4
9. What did you do today?	Fully active	1
	Light work/house work	2
	Got up but did nothing	3
	Stayed in bed	4

W.H.O performance Status²⁶

Performance	Grade
Able to carry out all normal activity without restriction	0

Restricted in physically strenuous activity but ambulatory &able to do light work	1
Ambulatory &capable of self care but unable to carry out any work	2
Capable of only limited self care confined to bed or chair 50% or more of the waking hours	3
Completely disabled &cannot carry on any self care	4

This study was carried out in the Govt.Homoeopathic Medical College, Kozhikode during the period from Aug 2003 – to Feb 2005 in 30 patients. The individual observations were given in the distribution table no 1, 2,3,4,5,6,7,8 9 and 10.

The results were categorized under the following headings as follows,

Observation and Discussion

Age Wise Distribution of Patients:

Out of the 30 patients studied 2 patients (6.66%) were between the age group 30 -39. 5 patients (16.66%) were between 40-49, 3 patients (10%) were between 50-59. 11 patients (36.66%) were between the age groups 60-69, 7 patients (23.33%) were between the age group 70-79, and 2 patients (6.66%) were between the age group 80-89.

Distribution of Patients According To Sex:

Chart:

3

Out of the 30 patients studied 16 patients (53.3%) were male patients and 14 patients (46.6%) were female.

Distribution Of Patients According To Religion:

Out of the 30 patients studied 16 patients (53%) were of Hindu religion, 11 patients (37%) were of Muslim religion and 3 patients (10%) belong to Christian religion.

Distribution of Patients according To Intensity of Pain Before and after treatment

Out of the 30 patients studied 8 patients (26.6%) presented with mild pain, 10 patients (33.3%) presented with moderate pain, and 12 patients (40%) presented with severe pain before treatment. After treatment 12 patients (40%) have no pain, 14 patients (46.6%) have mild pain 3 patients (10%) have moderate pain and 1 patient (3.3%) has severe pain.

Distribution of Patients According To the Site of Cancer:

Out of the 30 patients studied 5 patients (16.66%) have esophageal carcinoma, 3 patients each (10%) have bronchogenic carcinoma, bladder carcinoma, metastatic carcinoma with undetected primary and carcinoma of the stomach, 2 patients each (6.66%) presented with oral cancer carcinoma pharynx, larynx

and genital malignancy.1 patient each (3.33%) presented with lymphoma, leukemia, multiple myeloma, liver carcinoma and rectal carcinoma.

Distribution of Medicines Used In the Study:

t of 30 patients studied Lycopodium was found effective in 7 (23.33%) cases. Sulphur and Phosphorous was found effective in 6 (20%)cases each, Ars.alb was found effective in 3(10%) cases, Lachesis was found effective in 2(6.66%)cases, Ars.iod, Calc.iod, Kali.carb, Phytolaca ,China and Hepar.sulph were found to be effective in 1(3.33%) case each .

Distribution of Medicine Used For Acute Management:

Out of 30 patients studied Calc.carb was found to be effective in 9(30%) cases, Euphorbium and Nux.vom was found to be effective in 4(13.33%) cases each, Ars.alb was found to be effective in 3(10%) cases, Colocynth, Terebinth, and Carbo veg was found to be effective in 2 (6.66%) cases each, Spongia and Phytolaca was found to be effective in 1(3.33%) cases each.

Distribution of miasm of the cases:

Out of the 30 patients studied 11 patients (36.66%) are of psoric miasm,9 patients (30%) are psoro syphilitic,3 patients (10%) are psora sycotic and 7 patients (23.3%) have psoric+syphilitic+sycotic miasm.

Distribution of the miasm of Pain of the cases:

Out of the 30 patients studied, the miasm of pain of 27 patients (90%) are psoric, and the 3 patients (10%) pain belongs to syphilitic miasm.

STATISTICAL ANALYSIS

Assessment criteria include:

- Pain Sore
- Degree of distress score
- WHO performance status score

Tests of significance: -

- a) Questions to be answered. Is there any difference in the pain score, degree of distress score and performance status score of the case before and after treatment?
- b) Null hypothesis H_o: no difference in the pain score, degree of distress score and performance status score of the case before and after treatment.

Test of significance determined by using paired t' test.²⁷

$$EQ t = \overline{Z} / Sz / \sqrt{n}$$

	Calculated t value	Tabled t value	Level of significance	Significant or not
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			0.01	
Pain	12.248	2.756	< 0.01	Highly significant
Degree of distress	4.53	2.756	< 0.01	Significant
Performance status	-0.194	2.756	> 0.01	Not significant

The calculated t' value of pain score and degree of distress score are greater than the tabled t' value, so the result is significant Where as the calculated t' value of performance status score is less than the tabled t' value so the result is not significant.

Summary and Conclusion

In the present study 30 patients who attended OPD & IPD of Organon of Medicine from Aug 2003 – to Feb 2005 were included. These patients belonged to various socio-economic status end of age group between 30 - 90 years. The results of the study were evaluated using statistical principles.

Maximum age group affected 60-69 years, and the incidence was more in Hindu religion.

In this study the efficacy of homoeopathic treatment in the management of pain in cancer was evaluated. Assessment was based on the changes in pain score, degree of distress score and WHO performance status score before and after treatment.

After statistical analysis, the calculated value for pain score was 12.248 and degree of distress score was 4.53 which were well above the tabled value at 5% and levels P < 0.01. Thus, this study provides an evidence to say that homoeopathic medicines are effective in managing this condition. But the calculated value for performance status was -0.194 which were less than the tabled value at 5% and levels P < 0.01. Thus, this study provides an evidence to say that homoeopathic medicines are not effective in managing this condition.

Medicinal management was found to be very much effective. Lycopodium was the medicine found effective in 7(23.33%) patients. Sulphur and Phosphorous were found to be effective in 6(20%) patients each. Ars.alb was found effective in 3 (10%) patients. Lachesis was found to be effective in 2(6.66%) patients. Ars.iodi, Calc.iod, Kali.carb, Hepar.sulph and China were found to be effective in 1(3.33%) patient each.

CONCLUSION

The following salient conclusions have been drawn from the present study after summarizing its findings,

- 1) Homoeopathic medicines are effective in the management of pain in cancer.
- 2) Age group mostly affected is between 40 -90 years.
- 3) Males are affected more than females.

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