ONCOLOGY NURSING

Sanksi Pelanggaran Pasal 113
Undang-Undang Nomor 28 Tahun 2014
tentang Hak Cipta

1. Setiap Orang yang dengan tanpa hak melakukan pelanggaran hak ekonomi sebagaimana dimaksud dalam Pasal 9 ayat (1) huruf i untuk Penggunaan Secara Komersial dipidana dengan pidana penjara paling lama 1 (satu) tahun dan/atau pidana denda paling banyak Rp100.000.000 (seratus juta rupiah).
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Dilarang memperbanyak karya tulis ini dalam bentuk
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PREFACE

Cancer is a leading disease, cause of death, and source of morbidity of adults in the Western world. The incidence of cancer increases markedly with advancing age and is strongly affected by gender, lifestyle, ethnicity, infection, and genetics. Cancer refers to any one of a large number of diseases characterized by the development of abnormal cells that divide uncontrollably and have the ability to infiltrate and destroy normal body tissue. Cancer often has the ability to spread throughout your body. Cancer is a collection of more than 100 different diseases, caused by an accumulation of genetic and epigenetic alterations. Environment, heredity and behavior interact to modify the risk of developing cancer and the response to treatment. Improvements in treatment strategies and supportive care, coupled with new, often individualized therapies based on advances in fundamental understanding.

This book is all about Oncology, early detection to cancer plays a very crucial role in order to prevent the worsening of the condition. That is why nurses play big role in educating clients regarding early cancer detection. It is for this reason that cancer risk and screening procedures are hot topics in encountered in the exam.

Ernawaty Siagian

Each student has a destiny---a dream or vision filled with hope for their future---and each has been uniquely gifted and empowered by God to fulfil this dream as he or she connects with God.

EGW

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CHAPTER 1

Concepts of Cell Growth

Cancer is a disorder of altered cell differentiation and growth. Uncoordinated and relatively autonomous growth of neoplasms because they do not have the normal regulatory control over cell growth and division. Getting bigger and continuing to grow after the stimulation has stopped or the organism's needs have been met, this is a neoplasm. Tissue renewal and repair, as well as cancer pathogenesis, involve cell proliferation and differentiation. (Porth, 2015).

1.1 Cell Proliferation Versus Differentiation

The process of increasing the number of cells by mitotic division is called proliferation. The process by which cells become more specialized in structure and function is called differentiation (Robbins et al., 2010; Rubin et al., 2012). A cell that is undifferentiated and has the capacity to produce many different types of cells is called a stem cell. The size of the cell population is determined by the balance of cell proliferation, death by apoptosis, and emergence of new cells that differentiate from stem cells2 occurring in normal tissues (Figure 1-1).

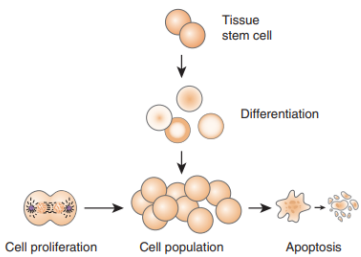


Figure 1.1 Normal tissues cells

Proliferative Capacity of Tissues

Regeneration of body tissue by: (1) continuing to divide, (2) stable, and (3) permanent tissue (Robbins et al., 2010; Rubin et al., 2012). Stem cells are special human cells that are able to develop into many different cell types.

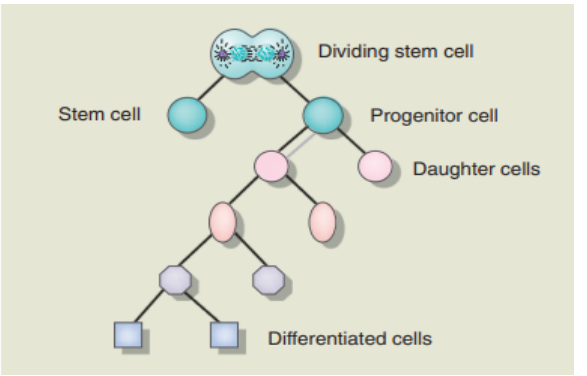


Figure 1.2 Stem cell mediated cell replacement

Understanding the Cell Cycle

1. Synthesis and Mitosis. Phase S, the period of DNA synthesis and chromosome replication takes about 10 to 12 hours. Formation of the mitotic spindle and cell division with the formation of two daughter cells takes less than 1 hour in Phase M.

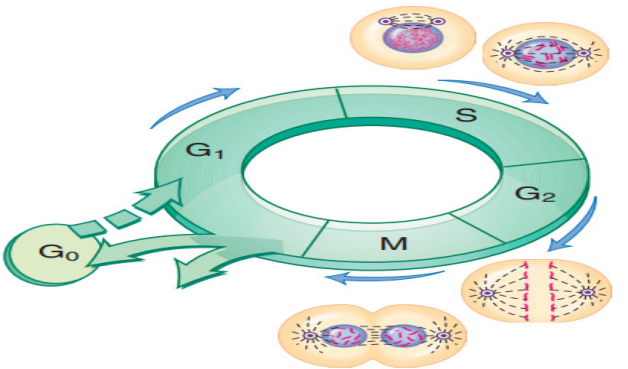


Figure 1.3 Synthesis and Mitosis

2. Gaps 1 and 2. Growing and multiplying the proteins and organelles of cells takes time, extra gaps (G) are inserted into the cell cycle. G1 is the stage during which the cell is starting to prepare for DNA replication and mitosis through protein synthesis and an increase in organelle and cytoskeletal elements. G2 is the premitotic phase. During this phase, enzymes and other proteins needed for cell division are synthesized and moved to their proper sites.

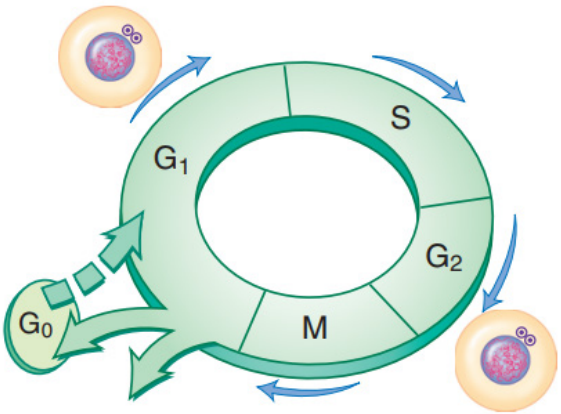


Figure 1.4 Gaps 1 and 2

3. Gap 0. After mitosis where the cell can leave the cell cycle and remain in an inactive state or re-enter the cell cycle at a later time is G0. Labile cells, do not enter G0 but continue cycling. Stable cells, such as hepatocytes, enter G0 after mitosis but can re-enter the cell cycle when stimulated by the loss of other cells. Permanent cells, such as neurons that become terminally differentiated after mitosis, leave the cell cycle and are no longer capable of cell renewal.

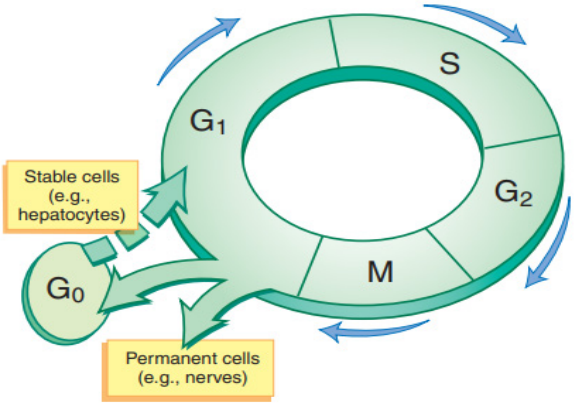


Figure 1.5 Gap 0

4. Checkpoints and Cyclins. In the cell cycle there are several checks, where the cycle can be terminated if previous events have not completed. For example, the G1/S checkpoint monitors whether the DNA in the chromosomes is damaged by radiation or chemicals, and the G2/M checkpoint prevents entry into mitosis if DNA replication is not complete. The cyclins are a family of proteins that control entry and progression of cells through the cell cycle. They function by activating proteins called cyclin-dependent kinases (CDKs). Different combinations of cyclins and CDKs are associated with each stage of the cell cycle. In addition to the synthesis and degradation of the cyclins, the cyclin–CDK complexes are regulated by the binding of CDK inhibitors. The CDK inhibitors are particularly important in regulating cell cycle checkpoints during which mistakes in DNA replication are repaired.

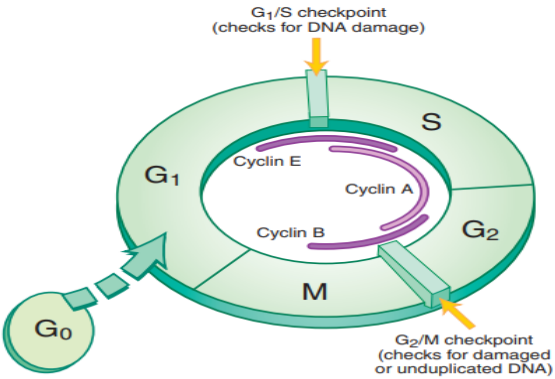


Figure 1.6 Checkpoints and Cyclins

1.2 Characteristics of Benign and Malignant Neoplasms

Differentiated based on benign and malignant neoplasms (1) degree of differentiation, (2) growth rate, (3) local invasion, (4) ability to metastasize and spread to other parts of the body, and (5) potential to cause death.

Table 1.1 Benign and Malignant Tumors According to Tissue Types

Tissue Type	Benign Tumors	Malignant Tumors
Epithelial		
Surface	Papilloma	Squamous cell carcinoma
Glandular	Adenoma	Adenocarcinoma
Connective		
Fibrous	Fibroma	Fibrosarcoma
Adipose	Lipoma	Liposarcoma
Cartilage	Chondroma	Chondrosarcoma
Bone	Osteoma	Osteosarcoma
Blood vessels	Hemangioma	Hemangiosarcoma
Lymph vessels	Lymphangioma	Lymphangiosarcoma
Lymph tissue		Lymphosarcoma
Muscle		
Smooth	Leiomyoma	Leiomyosarcoma
Striated	Rhabdomyoma	Rhabdomyosarcoma
Neural Tissue		
Nerve cell	Neuroma	Neuroblastoma
Glial tissue	Glioma	Glioblastoma, astrocytoma, medulloblastoma, oligodendroglioma
Nerve sheaths	Neurilemmoma	Neurilemmal sarcoma
Meninges	Meningioma	Meningeal sarcoma
Hematologic		
Granulocytic		Myelocytic leukemia
Erythrocytic		Erythrocytic leukemia
Plasma cells		Multiple myeloma
Lymphocytic		Lymphocytic leukemia or lymphoma
Monocytic		Monocytic leukemia
Endothelial Tissue		
Blood vessels	Hemangioma	Hemangiosarcoma
Lymph vessels	Lymphangioma	Lymphangiosarcoma

Table 1.2 Characteristics of Benign and Malignant Neoplasms

Characteristics	Benign	Malignant
Cell characteristics	Well-differentiated cells that resemble cells in the tissue of origin	Cells are undifferentiated, with anaplasia and atypical structure that often bears little resemblance to cells in the tissue of origin
Rate of growth	Usually progressive and slow; may come to a standstill or regress	Variable and depends on level of differentiation; the more undifferentiated the cells, the more rapid the rate of growth
Mode of growth	Grows by expansion without invading the surrounding tissues; usually encapsulated	Grows by invasion, sending out processes that infiltrate the surrounding tissues
Metastasis	Does not spread by metastasis	Gains access to blood and lymph channels to metastasize to other areas of the body

Benign Neoplasms

Well-differentiated cells that resemble the cells of tissue of origin with a slow and progressive growth rate that may atrophy or regress (Robbins et al., 2010; Rubin et al., 2012).

Malignant Neoplasms

In contrast to benign tumors, malignant neoplasms tend to grow rapidly, invade and infiltrate nearby tissue, and spread to other parts of the body. They lack a well-defined capsule and their margins are not clearly separated from the normal surrounding tissue(Robbins et al., 2010; Rubin et al., 2012).

Differentiation and Anaplasia

Differentiation refers to the extent to which the parenchymal (specific organ versus supportive tissue) cells of a tumor resemble their normal forbearers morphologically and functionally (Robbins et al., 2010; Rubin et al., 2012).

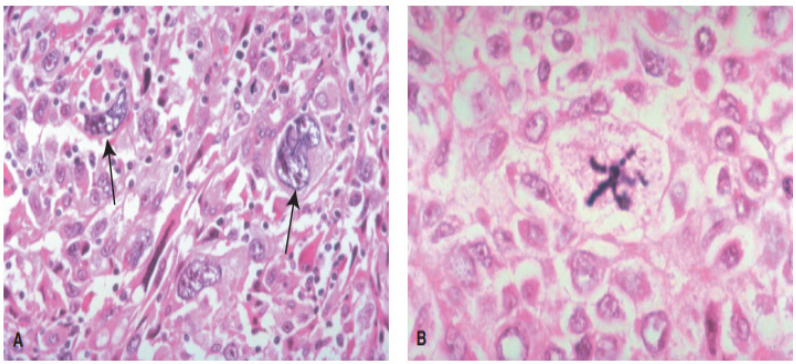


Figure 1.7 Anaplastic features of malignant tumors. (A) The cells of this anaplastic carcinoma are highly pleomorphic (i.e., they vary in size and shape). The nuclei are hyperchromatic and are large relative to the cytoplasm. Multinucleated tumor giant cells are present (arrows). (B) A malignant cell in metaphase exhibits an abnormal mitotic figure.

Tumor Growth

The growth of normal and cancerous tissue is based on: (1) the number of actively dividing cells, (2) the duration of the cell cycle, and (3) the number of cells lost relative to the number of new cells produced.

Metastatic Spread

Development of a secondary tumor at a site distant from the primary tumor represents metastasis (Robbins et al., 2010; Rubin et al., 2012). The spread of malignant tumors is the lymphatic channels or blood vessels (Robbins et al., 2010).

Lymphatic Spread

Through the lymphatic ducts metastases occur, the first time tumor cells lodge in the lymph nodes there cells can die from the right environment, or grow into a mass that can be seen. (Chen et al., 2006).

Hematogenous Spread

Capillaries and venules where cancer cells invade, thick walls are relatively resistant to arterioles and arteries. In venous invasion, venous blood from the digestive tract, pancreas, and spleen flows via the portal vein to the liver, and all of the vena cava blood flows to the lungs, liver and lungs which are metastatic sites for hematogenous spread (Robbins et al., 2010; Rubin et al., 2012).

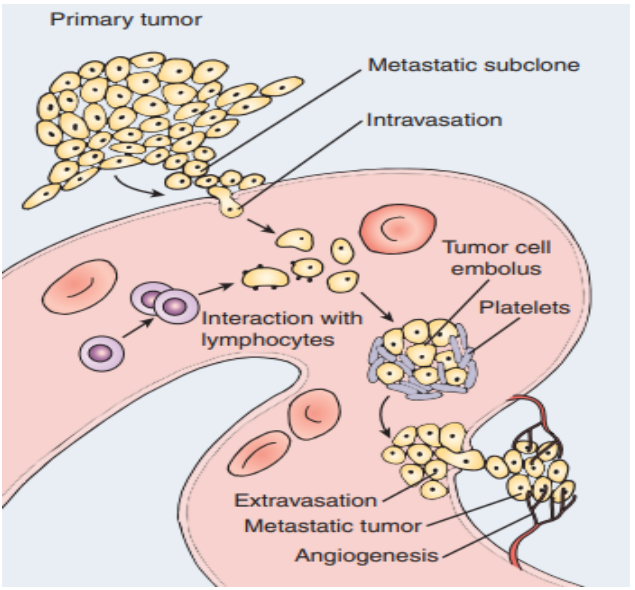


Figure 1.8 The pathogenesis of metastasi

CHAPTER 2
Cancer Epidemiology

Any of the following factors may contribute to the development of cancer (Cogliano et al., 2011; Institute of Medicine, 2001; National Toxicology Program, 2011):

a. Lifestyle (nutritional intake, smoking, alcohol use)
b. Environment (sun exposure, natural and medical radiation, occupational exposure, and unknown exposure)
c. Obesity and physical inactivity
d. Sexual practices
e. Drugs
f. Socioeconomic influences on exposure and vulnerability
g. Carcinogenic substances (air, water and soil)

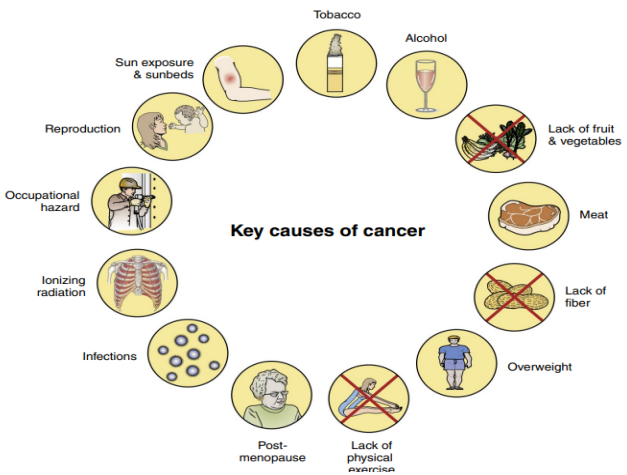


Figure 2.1 Causes of Cancer. (Colditz et al., 2012; National Cancer Institute, 2010; Parkin et al., 2011).

Early life influences later susceptibility to certain chronic diseases (Gluckman et al., 2008). The placenta has a major role in controlling growth and development throughout uterine development (Koukoura et al., 2012)

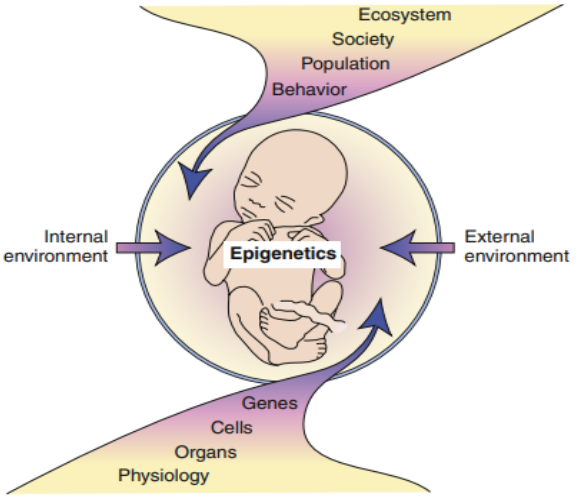


Figure 2.2 The fetus is vulnerable to the External and Internal Environment.

Tobacco Use

Second hand smoke, also called environmental tobacco smoke (ETS), is the combination of side stream smoke (burning end of a cigarette, cigar, or pipe) and mainstream smoke (smoke exhaled by the smoker). More than 4000 chemicals have been identified in mainstream tobacco smoke (250 chemicals as toxic), of which 60 are considered carcinogenic (Center For Disease Control and Prevention, 2006). Non-smokers who live with smokers are at greatest risk for lung cancer as well as numerous noncancerous conditions (National Cancer Institute, 2018). One in vitro study supported a relationship between nicotine and genotoxic effects in fetal cells (National Center for Environmental Health, 2011). United Kingdom researchers reported for the first time that starting smoking results in

epigenetic changes associated with the development of cancer (Ma et al., 2011). These researchers showed that smoking increased DNA methylation. Figure 2-4 illustrates a working model of carcinogenesis by cigarette smoke. Measures that prevent young adults from starting smoking would substantially avoid future disease burden. A strong public health approach is one that prevents young people from starting smoking and helps others stop smoking.

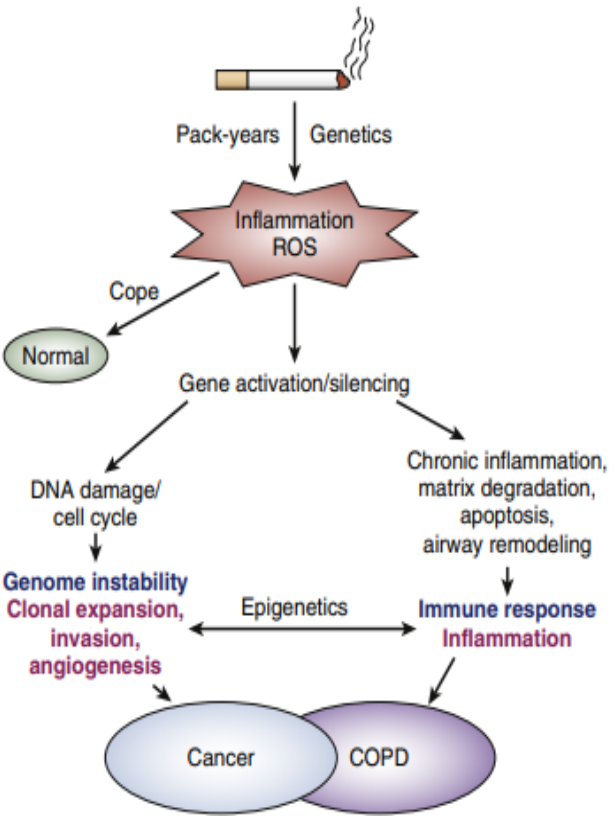


Figure 2.3 Working Model of Carcinogenesis by Cigarette Smoke.

Diet

Food acts directly as a mutagen or interferes with the elimination of the mutagen. (Figure 2-4).

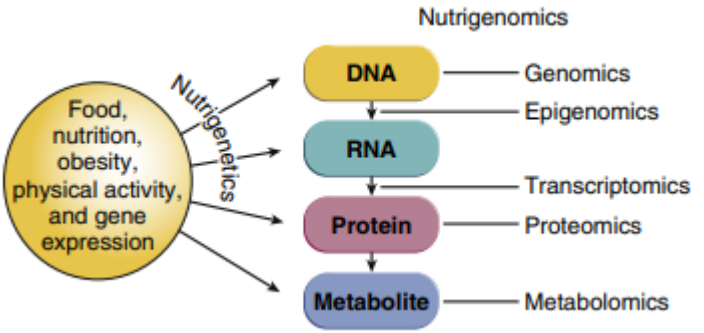


Figure 2.4 Food acts directly as a mutagen or interferes with the elimination of the mutagen

Nutrition

Nutrients affect cellular processes (Fig.2-5)

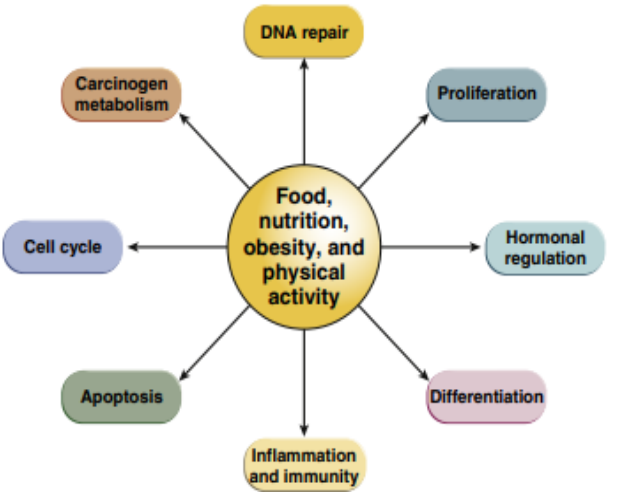


Figure 2.5 Nutrients affect cellular processes

Obesity.
Health conditions linked to obesity and physical inactivity. This requires a comprehensive approach including the involvement of knowledgeable health workers, provision of appropriate education from schools, access to healthy food and drink choices, and promotion of physical activity.

Mechanisms Associated with Energy Balance and Obesity. A simple model based on energy balance and energy expenditure was utilized. Energy balance measures intake-absorbable energy against the energy demands of the body. Energy expenditure is comprised of: (1) the resting metabolic rate (RMR), the energy required for normal body functions, which constitutes the majority of energy needs; (2) the thermic effect of food, or the amount of energy needed to digest and metabolize food; and (3) physical activity, a moderate and modifiable part of energy expenditure. This model does not include body composition (i.e., lean tissue, adipose tissue) or the dynamic state of body composition.

Alcohol Consumption. The human carcinogen is alcohol. contains ethanol which binds to DNA and causes cells to replicate incorrectly. and affects hormone levels that change the way cells grow and divide. Another ingredient is acetaldehyde which causes permanent DNA damage and triggers cancer.

Physical Activity. Studies suggest that regular exercise decreases the risk of breast cancer, colon cancer, and endometrial cancer, independent of weight changes.

The World Cancer Research Fund summarized the effects as convincing for cancers of the colon and probable for postmenopausal breast cancer and

endometrial cancer. Evidence of a reduction in breast cancer risk in postmenopausal women was associated with higher levels of activity (dose-response effect).

CHAPTER 3

CANCER

Cancer of The Digestive System

Cancer occurs throughout the alimentary tract and the accessory organs of digestion. A genetic predisposition is being evaluated.

Table 3.1 Cancer of the Gut, Liver, and Pancreas

Organ	**Risk**	**Cell Type**	**Common Manifestations**
Esophagus	Malnutrition Alcohol Tobacco Chronic reflux	Squamous cell Adenocarci-noma	Chest pain Dysphagia
Stomach	Salty food Nitrates and nitrosamines Gastric atrophy	Adenocarci-noma Squamous cell	Anorexia Malaise Weight loss Upper abdominal pain Vomiting Occult blood

Colorectal	Polyps Ulcerative colitis Diverticulitis High–refined-carbohydrate, low-fiber, high-fat diet	Adenocarcinoma (left colon grows in ring; right colon grows as mass)	Pain Mass Anemia Bloody stool Obstruction Distention
Liver	Hepatitis B, C, and D viruses Cirrhosis Intestinal parasite Aflatoxin from moldy peanuts	Hepatomas Cholangiomas	Pain Anorexia Bloating Weight loss Portal hypertension Ascites ± jaundice
Pancreas	Chronic pancreatitis Cigarette smoking Alcohol (?) Diabetic women	Adenocarcinoma (exocrine part of gland, ductal epithelium)	

Amended with permission from the American Cancer Society: Cancer facts and figures, 2009, Atlanta, GA, 2009, Author

3.1 Cancer of the Oesophagus

In Indonesia there were 1,327 new cases of oesophageal cancer and 1,283 deaths due to oesophageal cancer. This cancer is generally more common in men than women (Globocan,2020). Squamous cell carcinoma is found in the

upper two thirds of the oesophagus and is associated with smoking and alcohol ingestion. Adenocarcinoma accounts for about 58% of oesophageal carcinoma casesin the United States and is increasing. Adenocarcinoma is found in the distal one third of the oesophagus and is associated with risk factors that include smoking, abdominal obesity, reflux esophagitis, and sliding hiatal hernia. Carcinomas are most common at the gastroesophageal junction (Mawhinney & Glasgow, 2012).

Etiology and Pathophysiology. History of alcohol abuse; ingestion of hot, spicy foods; smoking; GERD; or Barrett oesophagus (oesophageal damage from acid reflux). The use of tobacco to alcohol, causes oesophageal cancer by damaging the DNA in the cells that line the inside of the oesophagus.

Pathogenesis

The pathogenesis of oesophageal carcinoma is facilitated by (1) chronic inflammation, metaplasia, and dysplasia caused by gastroesophageal reflux (Barrett oesophagus); and (2) long-term exposure to irritants, such as alcohol and tobacco, that cause neoplastic transformation. Both genomic and epigenomic events are associated with Barrett oesophagus and mutation of the TP53 gene is an early event (Kaz et al., 2012; Reid et al., 2011). The CagA-positive strain of H. pylori may be a protection against oesophageal adenocarcinoma (Islami & Kamangar, 2008).

Clinical Manifestations

The type of pain is heartburn (pyrosis). Pain in the throat or back, behind the breastbone, or between the shoulder blades. Vomiting or coughing up blood. Hoarse voice or chronic cough. Unintentional weight loss, bad breath and hiccups occur when the tumor spreads to the diaphragm. Clinical findings. esophagogastroduodenoscopy (EGD) with biopsy and brush showing malignant cells; Barium swallow shows a mass.

Evaluation and Treatment

Clients with dysphagia undergo endoscopy so that the specimen can be examined for neoplastic changes and the type of carcinoma. Thoracic CT study for diagnosis. Prevention of Barrett's esophagus is accomplished by treating gastroesophageal reflux. Barrett's esophagus with high-grade dysplasia is treated with endoscopic radiofrequency ablation, cryotherapy, or resection (Gaddam & Wani, 2013).

At the time of diagnosis, 50% of oesophageal cancers present with metastatic disease. The lymphatic vessels of the oesophagus are continuous with vital mediastinal structures (trachea, heart, and great vessels) and drain to the celiac lymph nodes, making it impossible to remove all the lymph nodes with the tumor. Removal of the primary lesion and the local lymph nodes, however, can benefit the individual with oesophageal cancer and cure is likely if there is no metastasis. If spread has occurred, treatment is combined radiation, chemotherapy, and palliative care (e.g., self-expanding metal stents) (Sgourakis et al., 2012).

Table 3.2 Food, Nutrition, Physical Activity, and Cancer Of The Oesophagus (American Institute for Cancer Research, 2007). American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington, DC: AICR, 2007.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE OESOPHAGUS

In the judgement of the Panel, the factors listed below modify the risk of cancer of the oesophagus. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		**Alcoholic drinks** **Body fatness**[1]
Probable	**Non-starchy vegetables**[2] **Fruits**[2] **Foods containing beta-carotene**[3] **Foods containing vitamin C**[3]	**Maté**[4]
Limited — suggestive	Foods containing dietary fibre[3] Foods containing folate[3] Foods containing pyridoxine[3,5] Foods containing vitamin E[3]	Red meat[6] Processed meat[7] High-temperature drinks
Limited — no conclusion	Cereals (grains) and their products; starchy roots, tubers, and plantains; pulses (legumes); soya and soya products; herbs, spices, and condiments; poultry; fish; eggs; milk and dairy products; total fat; saturated fatty acids; monounsaturated fatty acids; polyunsaturated fatty acids; sugary foods and drinks; salt; salting; fermenting; pickling; smoked and cured foods; nitrates and nitrites; frying; grilling (broiling) and barbecuing (charbroiling); protein; vitamin A; retinol; thiamin; riboflavin; calcium; iron; zinc; pro-vitamin A carotenoids; beta-cryptoxanthin; Seventh-day Adventist diets; adult attained height; energy intake	
Substantial effect on risk unlikely	None identified	

3.2 Cancer of the Stomach

The American Cancer Society's estimates for stomach cancer (also known as gastric cancer) in the United States for 2023 are: About 26,500 new cases of stomach cancer (15,930 in men and 10,570 in women) (American Cancer Society, 2021).

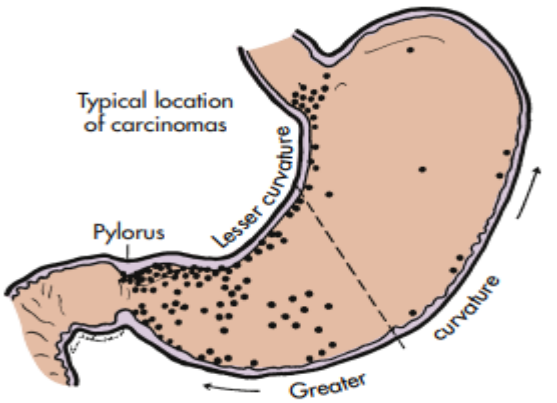


Figure 3.1 Typical Sites of Stomach Cancer

The most important environmental risk factors in causing gastric cancer are: (1) infection with H. pylori that carries the CagA gene product cytotoxin-associated antigen A (80% of cases); (2) dietary factors, such as salt added to food, food additives (e.g., nitrates) in pickled or salted foods (e.g., bacon), and low intake of fruits and vegetables; and (3) lifestyle, such as alcohol consumption and cigarette smoking. Infection with H. pylori and severe chronic gastritis change the mucosal cell proliferation pattern, destroy cell junctions, inhibit cell proliferation, and promote cell invasive ability, increasing the risk for gastric and duodenal carcinoma (Bornschein et al., 2011; Compare et al., 2010; P. Wang et al., 2012).

Pathogenesis

In the gastric mucosa gland adenocarcinoma begins. Gastric cancer develops in the prepyloric antrum in about 50%. (Figure 3-1). The development of gastric cancer is strongly associated with atrophic gastritis and intestinal metaplasia. A relatively alkaline environment is created which allows bacteria to multiply and act on nitrates due to insufficient acid secretion by the atrophic mucosa. Deoxyribonucleic acid (DNA) damage to mucosal cells further promotes metaplasia and neoplasia due to the increased nitrosoamines produced. Intestinal metaplasia

occurs due to the contribution of duodenal reflux (Gigek et al., 2012; Ness et al., 2012)

Clinical findings. Subjective: lack of interest in food (anorexia); nauseous; belching (eructation); heartburn (dyspepsia). Goals: weight loss; stool positive for occult blood; anemia; absence of hydrochloric acid; pale skin and acanthosis nigricans (hyperpigmentation, a velvety thickening of the skin on the neck, armpits, and groin)

Evaluation and Treatment

Direct endoscopic visualization and biopsy usually confirm the diagnosis. Surgery is the treatment for stomach cancer. Staging is determined by pathological findings after resection. Chemotherapy combined with chemoradiotherapy can provide the best postoperative results. The five-year survival is less than 20%. Screening and eradication of H. pylori infection is the best preventive approach for gastric cancer and gastric MALT lymphoma remission (Yahalom, 2011). Dietary modifications include high intakes of fruits and vegetables, vitamin C, carotenoids, and fiber as well as reducing intake of salt, salty foods, and red meat (Gonzalez & Riboli, 2010).

Table 3.3 Food, Nutrition, Physical Activity, and Cancer of The Stomach (American Institute for Cancer Research, 2007). American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington, DC: AICR, 2007.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE STOMACH

In the judgement of the Panel, the factors listed below modify the risk of cancer of the stomach. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		
Probable	**Non-starchy vegetables[1]** **Allium vegetables[1]** **Fruits[1]**	**Salt[2]** **Salted and salty foods**
Limited — suggestive	Pulses (legumes)[3] Foods containing selenium[4]	Chilli[1] Processed meat[5] Smoked foods[6] Grilled (broiled) or barbecued (charbroiled) animal foods[6]
Limited — no conclusion	Cereals (grains) and their products; dietary fibre; potatoes; starchy roots, tubers, and plantains; nuts and seeds; herbs, spices, and condiments; meat (unprocessed); poultry; eggs; milk and dairy products; fats and oils; total fat; fatty acid composition; cholesterol; sugars; sugar (sucrose); fruit juices; coffee; tea; alcohol; dietary nitrate and nitrite, *N*-nitrosodimethylamine; drying or dried food; protein; thiamin; riboflavin; vitamin C; vitamin D; multivitamin/mineral supplements; calcium; iron; selenium supplements; carotenoids; culturally defined diets; meal frequency; eating speed; body fatness; energy intake	
Substantial effect on risk unlikely	None identified	

3.3 Cancer of the Colon and Rectum

In 2020, there will be an estimated 104,610 new cases of colon cancer and 43,340 cases of rectal cancer diagnosed in the US. Although the majority of CRCs are in adults ages

50 and older, 17,930 (12%) will be diagnosed in individuals younger than age 50, the equivalent of 49 new cases per day (Global Headquarters: American Cancer Society, 2020). CRC develops in individuals with an acquired or inherited genetic predisposition who are exposed to a combination of environmental risk factors (Table 3-4).

Table 3-4 Selected Risk Factors and Colorectal Cancer

SELECTED RISK FACTORS AND COLORECTAL CANCER
Higher Risk Family history of colorectal cancer, familial adenomatous polyposis, Inflammatory bowel disease, Smoking or chewing tobacco Obesity Alcohol consumption Red meat consumption Type 2 diabetes mellitus High-fat, low-fiber diet **Lower Risk** Diets high in cereal grains, vegetables, milk; fish; folic acid, calcium, and vitamin D; magnesium and selenium Postmenopausal estrogen use Physical activity Use of NSAIDs

Yuhara H et al: Am J Gastroenterol 106(11):1911–1921, 2011; Zhang X, Giovannucci E: Best Pract Res Clin Gastroenterol 25(4-5):485–494, 2011

Pathogenesis

About 75% of CRC is sporadic (nonhereditary or acquired) and the environment contributes to multiple somatic mutations.

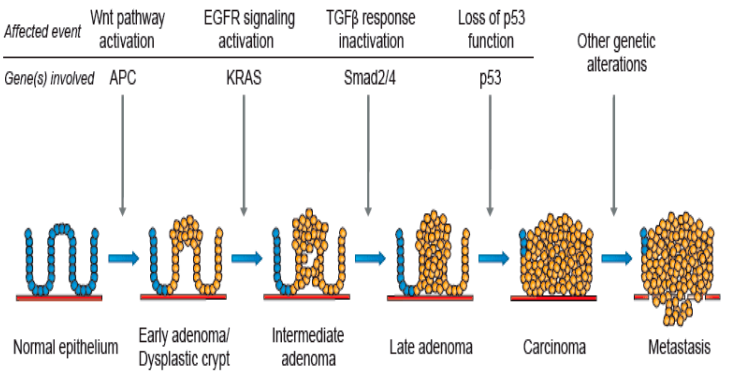


Figure 3.2 Multistage Development of Colonic Cancer

Most CRC develops from adenomatous polyps (Figure 3-3). A polyp is a mass or finger-like projection arising from the intestinal mucosal epithelium. Histologically, they are classified as tubular with branched tubular glands (the most prevalent), villous with finger-like projections of the epithelium (more related to CRC), or tubulovillous adenomas. Sessile serrated polyps have a serrated crypt and include hyperplastic polyps. They generally lack atypia but when they are large (greater than 1 cm), numerous (more than 20), and located in the right colon they are associated with cancer. The adenomatous polyp forms in an area of epithelial cell hyperproliferation and crypt dysplasia. Once the adenoma traverses the muscularis mucosae, it becomes invasive and highly malignant. Adenomas can be detected early, however, and the submucosa may not be penetrated for several years. The larger the polyp and the greater the degree of dysplasia, the greater the risk of CRC. Thus, screening colonoscopy with polypectomy is important when polyps are found. Most colorectal cancers are moderately differentiated adenocarcinomas. These tumors have a long preinvasive phase, and when they invade they tend to grow

slowly (10 to 15 years). Because the lymphatic channels are located underneath the muscularis mucosae, the lesions must traverse this layer before metastasis can occur. Systemic lymphatic spread occurs along the aorta to the mesenteric and pancreatic lymph nodes. Liver metastasis follows invasion of the mesenteric veins (left colon) or superior veins (right colon), which drain into the portal circulation.

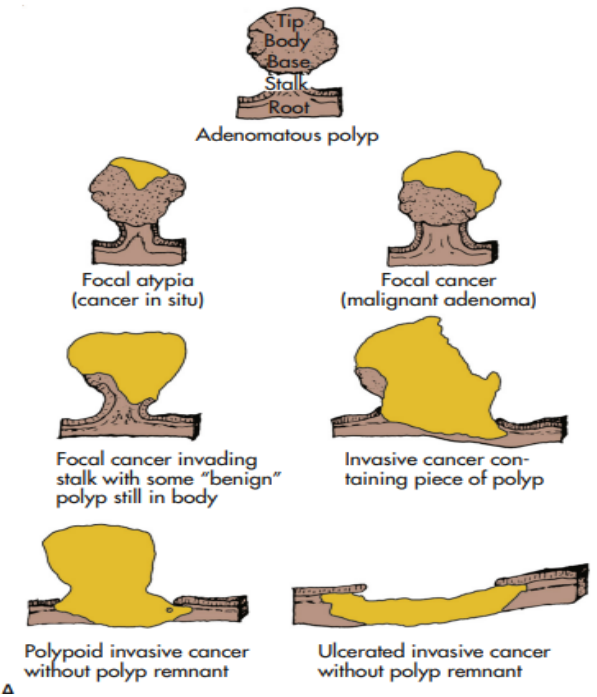


Figure 3.3 Development of Cancer of the Colon from Adenomatous Polyps. The tumor becomes invasive if it penetrates the muscularis mucosae and enters the submucosal layer (McCance & Huether, 2019)**.** McCance, Kathryn, Huether. Pathophysiology: The Biologic Basis for Disease in Adults and Children, Eight Edition. St. Louis, Missouri: Elsevier

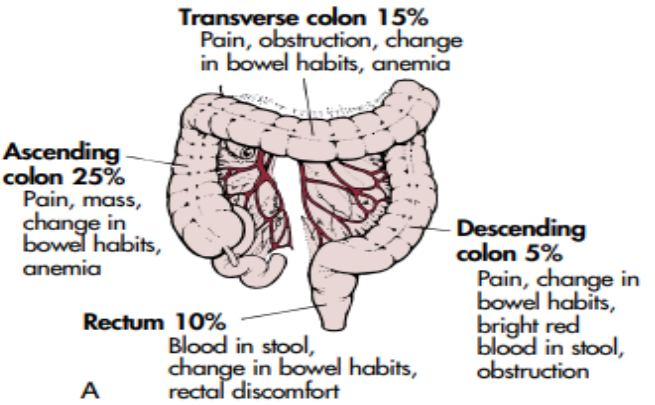


Figure 3.4 Colorectal Cancer Based on Primary Lesion Location.

Clinical Manifestations

Symptoms of colorectal cancer depend on the location, size, shape, and metastases of the lesion. Generalized obstruction, progressive abdominal distension, pain, vomiting, constipation, cramping, and bright blood on the surface of the stool, stool becomes narrow and pencil-shaped (De Salvo et al., 2004).

Evaluation and Treatment

Individuals with hereditary polyposis or a strong family history of CRC should begin screening at an early age (10 to 12 years) using colonoscopy and biopsy with a consideration of prophylactic surgery (Al-Sukhni et al., 2008). Screening for nonhereditary CRC in asymptomatic individuals older than age 50 years includes fecal occult blood and immunochemical tests, stool DNA and sigmoidoscopy, colonoscopy, virtual colonoscopy, or doublecontrast barium enema (Logan, 2012; National Cancer Institute, 2023b). The staging of colorectal cancer involves preoperative testing and operative exploration. Preoperative testing begins with physical examination of the abdomen to detect liver enlargement and ascites and palpation of appropriate lymph nodes. Elevations in the level of carcinoembryonic antigen (CEA) are often detected in the

sera of individuals with colorectal carcinoma. The amount of CEA in the serum is a function of the stage of the disease and the type of tumor. Operative staging consists of careful exploration during surgery and biopsy of possible metastases. The National Cancer Institute (National Cancer Institute, 2023a) classification is widely used for staging of colorectal cancer and is as follows:

a. Stage 0 (carcinoma in situ): involves only the mucosal lining; also known as carcinoma in situ
b. Stage I: extension of cancer to the middle layers of the colon wall, no spread to lymph nodes; stage I colon cancer is sometimes called Dukes' A colon cancer
c. Stage II: extension beyond the colon wall to nearby tissues around the colon or rectum, and/or through the peritoneum; stage II colon cancer is sometimes called Dukes' B colon cancer
d. Stage III: spread beyond the colon into lymph nodes and nearby organs and/or through the peritoneum; stage III colon cancer is sometimes called Dukes' C colon cancer
e. Stage IV: spread to nearby lymph nodes and has spread to other parts of the body, such as the liver or lungs; stage IV colon cancer is sometimes called Dukes' D colon cancer

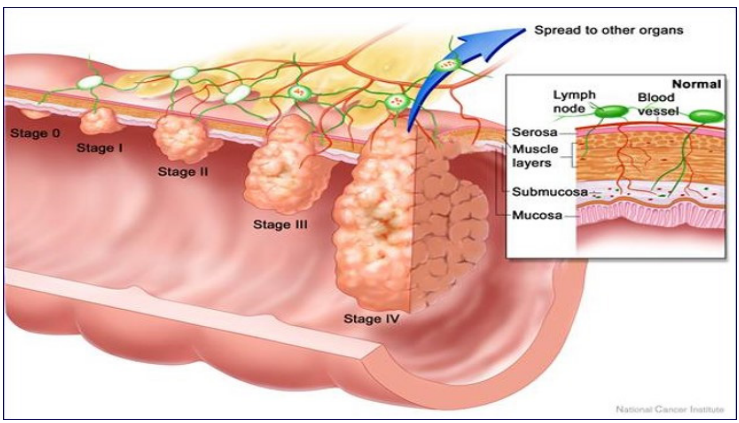
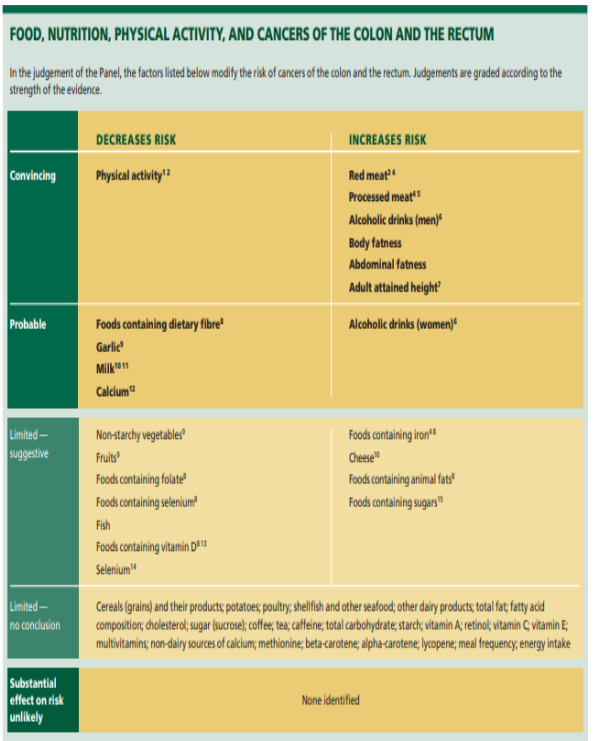


Figure 3.5 Stages of colorectal cancer (CRC).

Table 3.5 Food, Nutrition, Physical Activity, and Cancers of The Colon and The Rectum.



FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCERS OF THE COLON AND THE RECTUM

In the judgement of the Panel, the factors listed below modify the risk of cancers of the colon and the rectum. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing	**Physical activity**[1,2]	**Red meat**[2,4] **Processed meat**[4,5] **Alcoholic drinks (men)**[6] **Body fatness** **Abdominal fatness** **Adult attained height**[7]
Probable	**Foods containing dietary fibre**[3] **Garlic**[8] **Milk**[10,11] **Calcium**[12]	**Alcoholic drinks (women)**[6]
Limited — suggestive	Non-starchy vegetables[9] Fruits[9] Foods containing folate[8] Foods containing selenium[8] Fish Foods containing vitamin D[8,13] Selenium[14]	Foods containing iron[4,8] Cheese[15] Foods containing animal fats[8] Foods containing sugars[15]
Limited — no conclusion	Cereals (grains) and their products; potatoes; poultry; shellfish and other seafood; other dairy products; total fat; fatty acid composition; cholesterol; sugar (sucrose); coffee; tea; caffeine; total carbohydrate; starch; vitamin A; retinol; vitamin C; vitamin E; multivitamins; non-dairy sources of calcium; methionine; beta-carotene; alpha-carotene; lycopene; meal frequency; energy intake	
Substantial effect on risk unlikely	None identified	

3.4 Cancer of the Liver

Hepatocellular carcinoma (HCC) is a primary tumor of the liver. Hepatocellular carcinoma occurs in approximately 85% of patients diagnosed with cirrhosis (Ioannou et al., 2007). The second leading cause of cancer death after lung cancer in men is HCC (Ferlay et al., 2015). Five-year survival of HCC is 18% and second to pancreatic cancer (Jemal et al., 2017). Significant risk factors for hepatocellular carcinoma include viral hepatitis (hepatitis B and hepatitis C), alcoholic liver disease, and non-alcoholic liver steatohepatitis/non-alcoholic fatty liver disease. HCC occurs in 80%-90% of patients with cirrhosis. The annual incidence of HCC in patients with cirrhosis is 2-4% (Ioannou et al., 2007). Cancer in the liver is usually caused by metastatic spread from a primary site elsewhere in the body.

Risk factors for primary liver cancer include the following (Blonski et al., 2010; El-Serag, 2011; McGlynn & London, 2011; Trichopoulos et al., 2011) :

Infection with HBV, HCV, and HDV (Arzumanyan et al., 2013)

Chronic alcoholic liver disease and non-alcoholic liver disease that results in cirrhosis

Exposure to mycotoxins

Long duration of heavy smoking (greater than 20 years)

Nonalcoholic fatty liver disease 6. Hepatic iron overload

Pathogenesis

Hepatocellular carcinoma develops in hepatocytes. Hepatocellular carcinoma (hepatocarcinoma) (HCC) can be nodular, massive, or diffuse. HCC is a primary liver cancer associated with cirrhosis. HCC repetitive cell proliferation that occurs in the inflamed liver in response to growth factors and cytokine stimulation and DNA damage due to oxidative stress elicited by Hepatitis B, C and cirrhosis (Tyson & El-Serag, 2011).

Clinical Manifestations

Characterized by nausea and vomiting, fullness, pressure, dull pain in the right hypochondrium, and weight loss. Sudden worsening of portal hypertension and development of ascites due to tumor obstruction

Evaluation and Treatment

There is no specific test for the diagnosis of liver cancer. For HCC high-risk individuals, alpha fetoprotein and abdominal ultrasound are common screening tools and can be used for screening in individuals with cirrhosis (Forner et al., 2018). Additional serum markers are being evaluated (Masuzaki et al., 2012). The diagnosis is based on additional laboratory findings, radiologic examination, biopsy findings, and exploratory laparotomy. Serum levels of alkaline phosphatase, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) are commonly elevated in individuals with HCC. Transarterial

embolization and radiation is used for management of pain and to reduce tumor size (Lencioni, 2012). Surgical resection or liver transplantation is the only alternative for cure; however, very early stage HCC is difficult to diagnose (Forner et al., 2018). Primary prevention of HCC is vaccination against HBV and antiviral therapy for HCV (Cabibbo et al., 2012).

Table 3.6 Food, Nutrition, Physical Activity, and Cancer of The Liver.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE LIVER

In the judgement of the Panel, the factors listed below modify the risk of cancer of the liver. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		**Aflatoxins[1]**
Probable		**Alcoholic drinks[2]**
Limited — suggestive	Fruits[3]	Body fatness
Limited — no conclusion	Cereals (grains) and their products[1]; non-starchy vegetables; peanuts (groundnuts)[1]; fish; salted fish; water source; coffee; tea	
Substantial effect on risk unlikely	None identified	

3.5 Cancer of the Gallbladder

In 2023, an estimated 12,220 adults (5,750 men and 6,470 women) in the United States will be diagnosed with gallbladder and other biliary cancers. About 4 out of 10 are specifically gallbladder cancers. Worldwide, an estimated 115,949 people were diagnosed with gallbladder cancer in 2020. It is estimated that 4,510 deaths (1,900 men and 2,610 women) from gallbladder and other biliary cancers will occur in the United States in 2023. In 2020, an estimated 84,695 people worldwide died from gallbladder cancer (American Society of Clinical Oncology, 2023d). It rarely occurs before age 40.Risk

factors include gallstones, advancing age, female gender (2:1), anomalous pancreaticobiliary ductal junction, and obesity. Native populations in North and South America have greater risk of gallbladder cancer, and it is more common in Chile, Poland, India, Japan, and Israel (Dutta, 2012; Lowenfels et al., 1999; Pandey, 2003; F. Wang et al., 2012). Most cancerous tumors in the gallbladder are caused by metastasis.

Pathogenesis

Most primary cancers of the gallbladder are adenocarcinomas and, less commonly, squamous cell carcinomas. The mechanisms of tumorigenesis are not clear. Multiple genes and oncogenes are involved in the initiation and progression of gallbladder cancer. Research is in progress to target these genes for diagnosis and treatment (Andrén-Sandberg, 2012). Infiltrative tumors are associated with gallstones, and invasion of the liver and lymph nodes occurs early. Spreading extends to the pancreas and retroperitoneal lymph nodes. Direct invasion of the stomach and the duodenum can cause pyloric obstruction. Infection often accompanies cancer of the gallbladder. Generalized peritonitis, gangrene, perforation, and liver abscesses are potential complications of infection.

Clinical Manifestations

Early stages of gallbladder carcinoma are asymptomatic. A typical presentation is steady upper right quadrant pain for about 2 months. Other symptoms mimic benign gallbladder disease, including diarrhea, belching, weakness, loss of appetite, weight loss, and vomiting. Obstructive jaundice can occur if an enlarging tumor presses on the extrahepatic ducts.

Evaluation and Treatment

Early diagnosis of gallbladder cancer is rare and the disease is often found incidentally when removing gallstones or when an individual presents with an advanced stage of disease (Fuks et al., 2011). Individuals with gallstones, especially older women, are evaluated carefully. Inflammatory disorders, such

as cholangitis (bile duct inflammation) and peritonitis, often obscure an underlying malignancy. The most specific diagnostic procedures include ultrasonography, CT, and MRI. Complete surgical resection of the gallbladder is the only effective treatment. Because advanced malignancies cannot be resected, gallbladders containing stones are removed as a preventive measure. Palliative chemotherapy or chemoradiation provides symptom improvement but does not improve survival (Thomas, 2008). The prognosis of gallbladder cancer is extremely poor; most individuals die within 1 to 2 years after surgery (Gore & Shelhamer, 2007; Marsh et al., 2012).

Table 3.7 Food, Nutrition, Physical Activity, and Cancers of The Gallbladder.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE GALLBLADDER

In the judgement of the Panel, the factors listed below modify the risk of cancer of the gallbladder. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		
Probable		**Body fatness**[1]
Limited — suggestive		
Limited — no conclusion	Peppers (capsicums); fish; coffee; tea; alcohol; vitamin C.	
Substantial effect on risk unlikely	None identified	

3.6 Cancer of the Pancreas

Worldwide, an estimated 495,773 people were diagnosed with pancreatic cancer in 2020. It is estimated that 49,830 deaths (25,970 men and 23,860 women) from this disease will occur in the United States this year. It is the fourth leading cause of cancer death in both men and women. Pancreatic cancer accounts for 7% of all cancer deaths. The death rate has very

slowly increased each year since the mid-2000s. In 2020, an estimated 466,003 people worldwide died from pancreatic cancer (American Society of Clinical Oncology, 2023h). Risk factors include cigarette smoking, heavy alcohol use, family history of pancreatic cancer, and non-O blood group (Duell, 2012). Chronic pancreatitis is associated with about 4% to 5% of pancreatic cancers (Kudo et al., 2011; Raimondi et al., 2010).

Pathogenesis

Cancer of the pancreas can arise from exocrine or endocrine cells. Most pancreatic tumors arise from exocrine cells in the ducts and are called ductal adenocarcinomas. Tumors arising in small ducts invade nearby glandular tissue, penetrate the covering of the pancreas, and extend into surrounding tissues. Pancreatic canceris a disease of inherited and acquired mutations in cancer-related genes associated with chronic inflammation (Klöppel & Lüttges, 2004). Growth factors are overexpressed in ductal cancer (Birnbaum et al., 2012; Macgregor-Das & Lacobuzio-Donahue, 2013).

Clinical Manifestations

Cancer of the body and tail of the pancreas is generally asymptomatic until there is intraductal obstruction or the tumor invades adjacent tissue. Often vague, abdominal or mid-back pain is an initial symptom (Lomberk, 2008). Jaundice develops in most cases, usually caused by obstruction of the bile duct. Because obstruction impairs enzyme secretion and flow to the duodenum, pancreatic cancer causes fat and protein malabsorption, resulting in weight loss. Some individuals develop diabetes mellitus. Distant metastases are found in the cervical lymph nodes, the lungs, and the brain. Most individuals die of hepatic failure, malnutrition, or systemic complications.

Evaluation and Treatment

Pancreatic cancer is usually diagnosed at an advanced stage and has a poor prognosis. The most effective screening method

for those at risk is endoscopic ultrasound because it detects small lesions; the search for biomarkers is continuing (Stoita et al., 2011). Contrast CT scans, contrast MRI, and abdominal and endoscopic ultrasound (EUS) are used for initial diagnosis. EUS also provides opportunity for biopsy, or CT-guided fine needle aspiration can confirm diagnosis although this is not necessary when cancer suspicion is high (Tummala et al., 2011). Laparotomy is used to establish a definitive diagnosis, evaluate the extent of disease, and determine whether palliative bypass surgery (i.e., cholecystojejunostomy and gastrojejunostomy) is needed. Individuals with small tumors and complete resection have the best possibility of cure (15% to 20% of cases). Pancreaticoduodenectomy (Whipple procedure) is the most common procedure and portal or superior mesenteric vein resection and reconstruction may be performed. Some surgeons recommend a total pancreatectomy because cancer of the pancreas seldom consists of a single lesion (Casadei et al., 2010). The dense stromal structure of the tumor impairs chemotherapeutic drug delivery, resulting in poor response to chemotherapy (Corbo et al., 2012). Adjuvant chemotherapy is used for curative attempts in resected lowgrade tumors. Chemoradiation therapy is used for advanced disease in some centers (Herreros-Villanueva et al., 2012; Vincent et al., 2011; Yee, 2013). Palliative care, including pain management and nutrition, are important (Fazal & Saif, 2007).416

Table 3.8 Food, Nutrition, Physical Activity, and Cancer of The Pancreas.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE PANCREAS

In the judgement of the Panel, the factors listed below modify the risk of cancer of the pancreas. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		**Body fatness**
Probable	**Foods containing folate[1]**	**Abdominal fatness** **Adult attained height[2]**
Limited — suggestive	Fruits[3] Physical activity[4]	Red meat[5]
Limited — no conclusion	Cereal (grains) and their products; dietary fibre; vegetables; pulses (legumes); soya and soya products; processed meat; poultry; fish; eggs; milk and dairy products; total fat; butter; plant oils; margarine; cholesterol; sugar (sucrose); black tea; green tea; alcohol; nitrate and nitrite; total carbohydrate; folic acid supplements; vitamin C; vegetarianism; age at menarche; lactation; energy intake	
Substantial effect on risk unlikely	Coffee	

3.7 Renal Tumor

In the United States, kidney cancer is the sixth most common cancer for men. It is the ninth most common cancer for women. The average age at diagnosis for people with kidney cancer is 64, and most people are diagnosed between the ages of 65 and 74. Pathogenesis. Adenocarcinoma is renal cell carcinoma. Clinical Manifestations. Things that are found on the client are hematuria, pelvic pain, soreness. while in the advanced stages you will find decreased body weight, hypertension, fatigue and fever.

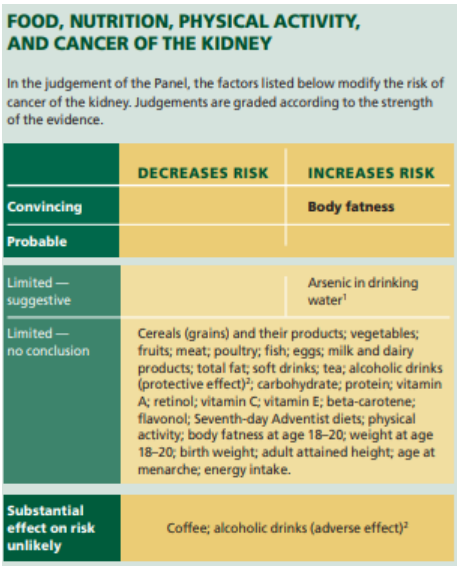
Table 3.9 Renal Cell carcinoma (TNM System)

STAGE	METASTASIS
I	Tumor confined within kidney capsule ≤7 cm in size.
II	Invasion through renal capsule and renal vein but within surrounding fascia ≥7 cm in size.
III	Involvement of adrenal glands and vena cava and one nearby lymph node:
	T3a-T3c, N0, M0: The main tumor has reached the adrenal gland, the fatty tissue around the kidney, the renal vein, and/or the large vein (vena cava) leading from the kidney to the heart. It has not spread beyond Gerota's fascia. There is no spread to lymph nodes or distant organs.
	T1a-T3c, N1, M0: The main tumor can be any size and may be outside the kidney, but it has not spread beyond Gerota's fascia. The cancer has spread to one nearby lymph node but has not spread to distant lymph nodes or other organs.
IV	Distant metastases (e.g., liver, lung, bones, brain) and more than one lymph node:
	T4, N0-N1, M0: The main tumor has invaded beyond Gerota's fascia. It has spread to no more than one nearby lymph node. It has not spread to distant lymph nodes or other organs.
	Any T, N2, M0: The main tumor can be any size and may be outside the kidney. The cancer has spread to more than one nearby lymph node but has not spread to distant lymph nodes or other organs.
	Any T, Any N, M1: The main tumor can be any size and may be outside the kidney. It has spread to distant lymph nodes and/or other organs.

Evaluation and Treatment

Ultrasound, CT scan, MRI, Biopsy, and laboratory tests. Surgical removal of the affected kidney (radical nephrectomy) or partial nephron sparing nephrectomy for smaller tumors. Surgery is combined with chemotherapeutic agents (Dutcher et al., 2012).

Table 3.10 Food, Nutrition, Physical Activity, and Cancer of The Kidney.



FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE KIDNEY

In the judgement of the Panel, the factors listed below modify the risk of cancer of the kidney. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		**Body fatness**
Probable		
Limited — suggestive		Arsenic in drinking water[1]
Limited — no conclusion	Cereals (grains) and their products; vegetables; fruits; meat; poultry; fish; eggs; milk and dairy products; total fat; soft drinks; tea; alcoholic drinks (protective effect)[2]; carbohydrate; protein; vitamin A; retinol; vitamin C; vitamin E; beta-carotene; flavonol; Seventh-day Adventist diets; physical activity; body fatness at age 18–20; weight at age 18–20; birth weight; adult attained height; age at menarche; energy intake.	
Substantial effect on risk unlikely	Coffee; alcoholic drinks (adverse effect)[2]	

3.8 Bladder Tumors

Approximately 90% to 95% of bladder cancers are derived from the transitional epithelial (urothelial) cells that line the bladder.

Etiology and Pathogenesis

There are local influences, such as carcinogens excreted in the urine and stored in the bladder. 30% to 50% of all bladder cancers among men are current or past smokers. Presence of arsenic in drinking water and industrial exposure to the decomposition products of aromatic amines used in the dyes industry and chemicals used in the manufacture of rubber, textiles, paints and petroleum products.

Clinical Manifestations

Frequent urination at night, increased frequency of urination, difficulty holding urination (urinary incontinence), pain or a burning sensation when urinating and frequent urge to urinate suddenly.

Table 3.11 Staging of Bladder Carcinoma

T – primary tumour	
Tx	Primary tumour cannot be assessed
T0	No evidence of primary tumour
Ta	Noninvasive papillary carcinoma
Tis	Carcinoma in situ
T1	Tumour invades subepithelial connective tissue (lamina propria)
T2	Tumour invades muscle (muscularis propria) • T2a tumour invades superficial muscle (inner half) • T2b tumour invades deep muscle (outer half)
T3	Tumour invades perivesical tissue • T3a miscroscopic • T3b macroscopic (extravesical mass)
T4	Invasion of adjacent structures • T4a tumour invades prostate, uterus, vagina • T4b tumour invades pelvic or abdominal wall
N – lymph nodes	
Nx	Regional nodes cannot be assessed
N0	No regional lymph node disease
N1	Metastasis in single node 2 cm or less
N2	Metastasis in single or multiple nodes between 2–5 cm
N3	Metastasis in lymph node greater than 5 cm
M – metastasis	
Mx	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Diagnosis and Treatment

Cytology, excretory urography, cystoscopy and biopsy. Ultrasound, CT scan, and MRI to determine the stage of the tumor. Endoscopic resection is performed as a treatment for superficial lesions. Initial diagnostic transurethral resection for non-high-grade small papillary tumors. Segmental surgical resection is used to remove large single wounds.

Table 3.12 Food, Nutritional, Physical Activity, and Cancer of The Bladder.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE BLADDER

In the judgement of the Panel, the factors listed below modify the risk of cancer of the bladder. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		
Probable		
Limited — suggestive	Milk[1]	Arsenic in drinking water[2]
Limited — no conclusion	Cereals (grains) and their products; vegetables; fruits; pulses (legumes); meat; poultry; fish; eggs; total fat; butter; dietetic foods; soft drinks; diet drinks; fruit juices; coffee; tea; caffeine; alcohol; chlorinated surface water; total fluid intake; sweeteners; frying; carbohydrate; protein; vitamin A; folate; vitamin C; vitamin E; multivitamin supplements; selenium; beta-carotene; alpha-carotene; lycopene; beta-cryptoxanthin; lutein; zeaxanthin; flavonoids; physical activity; body fatness; energy intake	
Substantial effect on risk unlikely	None identified	

3.9 Skin Cancer

The most serious and most common cause of death from skin cancer is malignant melanoma. This is caused by chronic ultraviolet (UV) radiation. Basal Cell Carcinoma, The most serious cause of death from skin cancer is malignant melanoma. Skin cancer is caused by ultraviolet radiation. Basal cells contain the pigment melanin, a protective factor against sun exposure found in people with dark skin. Squamous Cell Carcinoma. Is an epidermal tumor, there is a change in DNA that is triggered by UV radiation.

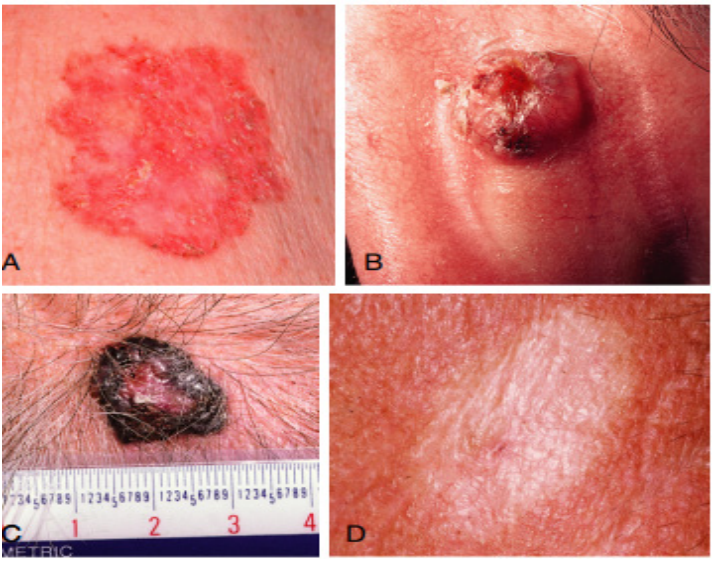


Figure 3.6 Types of Basal Cell Carcinoma. A, Superficial. B, Nodular. C, Pigmented. D, Morpheaform—recurrent tumor. (A and D from Bolognia JL, Jorizzo JL, Schaffer JV: Dermatology, ed 3, Philadelphia, 2012, Saunders; B and C from James WD, Berger TG, Elston DM: Andrews' diseases of the skin: clinical dermatology, ed 11, Philadelphia, 2011, Saunders.)

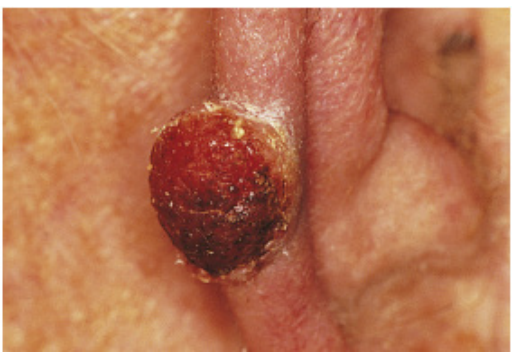


Figure 3.7 Squamous Cell Carcinoma. The sun-exposed ear is a common site for squamous cell carcinoma. (Courtesy Department of Dermatology, School of Medicine, University of Utah.

Cutaneous Melanoma

Cutaneous melanoma is a malignant tumor of the skin originating from melanocytes, or cells that synthesize the pigment melanin. The incidence of melanoma is increasing, and young to middle-age adults are at highest risk. Risk factors implicated in melanoma induction include genetic predisposition, exposure to ultraviolet light (solar and artificial), acquired melanocytic nevi, family history of melanoma, fair hair, light skin with a propensity to sunburn, and the presence of susceptibility genes (Mehnert & Kluger, 2012; Russak & Rigel, 2012). The relationship between nevi and melanoma makes it important for the clinician to understand the various neval forms (Table 3-12). Early recognition of cutaneous melanomas can have a major effect on achieving a surgical cure. Prevention of melanoma includes avoidance of UV radiation exposure through use of protective clothing and sunscreens, and avoidance of artificial UV radiation exposure.

Table 3.13 Classification of Nevi

NEVI	COMMON CHARACTERISTICS
Junctional nevus	Flat, well circumscribed, vary in size up to 2 cm, dark color, hairs may be present; originate in basal layer of epidermis and can eventually reach the cutaneous surface; rarely develop into a melanoma
Compound nevus	Most common in adolescents; the majority of pigmented lesions are in children; rarely develops into melanoma; usually 1 cm in size; hairs may be present; surface is elevated and smooth
Intradermal nevus	Small (less than 1 cm) with regular edges and bristle-like hairs; color ranges from skin tone to light brown; has a slight likelihood of developing into a melanoma

Table 3.14 TNM staging Criteria for Melanoma

Stage 0: carcinoma in situ (TisN0M0)
Stage I A/B: includes lesions up to 2 mm with no nodal or distant metastases (T1aN0M0, T1bN0M0, T2aN0M0).
Stage II A/B/C: includes larger lesions, greater than 2 mm without positive nodes or distant metastases (T2bN0M0, T3aN0M0, T3bN0M0, T4aN0M0, T4bN0M0).
Stage III: includes lesions of any size with positive lymph nodes (TxN1M0, TxN2M0, TxN3M0).
Stage IV: includes lesions of any size with distant metastases (TxNxM1).

Data from Piris A, Lobo AC, Duncan LM: Melanoma staging: Dermatol Clin 30(4):581–592, v, 2012.

Table 3.15 Food, nutrition, Physical Activity, and cancer of The Skin.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE SKIN

In the judgement of the Panel, the factors listed below modify the risk of cancer of the skin. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		
Probable		**Arsenic in drinking water[1]**
Limited — suggestive	Retinol[2]	Selenium supplements[3]
Limited — no conclusion	Potatoes; non-starchy vegetables; fruits; fish; eggs; milk; total fat; cholesterol; coffee; tea; alcohol; protein; vitamin A; retinol (foods); folate; vitamin C; vitamin D; vitamin E; multivitamins; selenium; carotenoids; beta-carotene (melanoma); alpha-carotene; lycopene; physical activity; body fatness; energy intake	
Substantial effect on risk unlikely	Beta-carotene[4] (non-melanoma)	

3.10 Cervical Cancer

Cervical cancer ranks second after breast cancer as the most common type of cancer of all cancer cases in 2020 in Indonesia. There have been more than 36,000 cases and 21,000 deaths

from this cancer.

Pathogenesis

It is established that cervical cancer is almost exclusively caused by cervical human papillomavirus (HPV) infection. Infection with "high-risk" (oncogenic) types of HPV (predominantly 16 and 18) is a necessary precursor to development of the precancerous cell changes, known as dysplasia, of the cervix that leads to invasive cancer. Anything that affects the integrity of the immune system may affect the later risk of cervical cancer including poor nutrition and chronic stress (CA et al., 2011). HIV infection greatly increases the risk that women infected with HPV will develop cervical cancer, and women with HIV should be screened for cervical cancer more frequently than women without HIV (Curry et al., 2018).

Clinical Manifestations

Cervical neoplasms are predominantly asymptomatic; therefore, regular Pap test or HPV screening is necessary for early detection. About 90% of cervical cancer cases can be detected through early use of regular screening tests. If symptoms exist, they may include vaginal bleeding or abnormal discharge. Bleeding is variable and may occur after intercourse or between menstrual periods. Vaginal discharge is a less common presenting symptom and may be serosanguineous or yellowish with a foul odour.

Evaluation and Treatment

HPV testing is now recommended at the same time as the Pap smear because it is non-invasive and identifies women at later risk for cellular abnormalities leading to cancer. HPV is often detectable for more than a decade prior to any noticed cellular changes (Sankaranarayanan et al., 2009). Colposcopy involves examining the cervix visually and taking needed biopsies. An acetic acid (vinegar) solution is applied to the cervix, making areas of HPV infection stand out in a white colour, known as aceto-white. The cervix is then viewed under magnification for

aceto-white areas, changes in the epithelium, and the presence of abnormal vascular patterns (Carcio, 2011). Abnormal areas of the cervix are biopsied. Because the vulnerable transformation zones move into the cervical canal as a woman ages, the endocervix is sampled using curettage for diagnosis (Table 3-13)

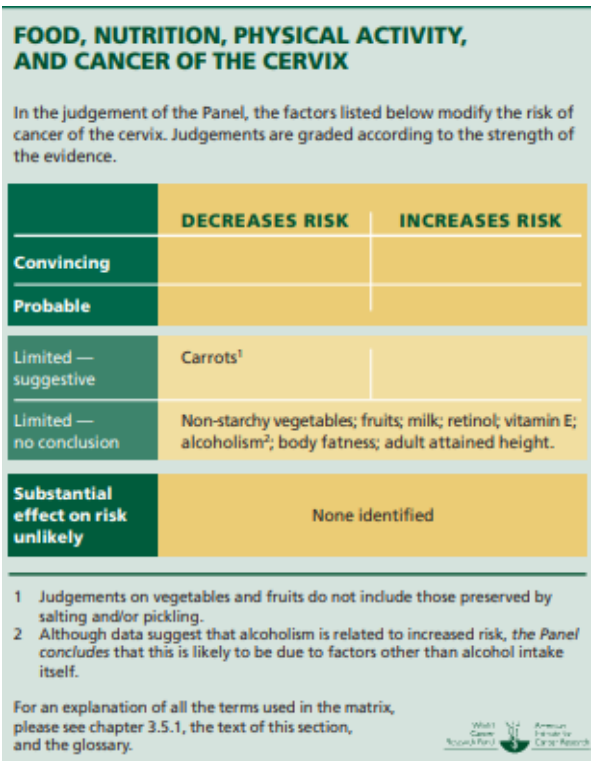
Table 3- 16 Clinical Staging for Cancer of the Cervix. (Society of Gynecologic Oncology, 2014)

CARCINOMA OF THE CERVIX	
Stage I: Cancer confined to the cervix	
IA	Microscopic cancer depth ≤5 mm and extension ≤7 mm
IA1	Stromal invasion ≤3 mm and extension ≤7 mm
IA2	Stromal invasion >3 mm and ≤5 mm with extension ≤7 mm
IB	Cancer confined to the cervix clinically visible or exceeds the dimensions for IA
IB1	≤4 cm in greatest diameter
IB2	>4 cm in greatest diameter
Stage II: Cancer extends beyond cervix but not to pelvic wall or lower third of vagina	
IIA	Without parametrial invasion
IIA1	≤4 cm in greatest diameter
IIA2	>4 cm in greatest diameter
IIB	With parametrial invasion
Stage III: Cancer extends to pelvic wall and/or involves lower third of vagina and/or causes hydronephrosis or non-functioning kidney	
IIIA	Tumor involves lower third of vagina, no extension to pelvic wall
IIIB	Extension to pelvic wall and/or hydronephrosis or non-functioning kidney
Stage IV: Cancer extends beyond true pelvis or has involved (biopsy proven) mucosa of bladder or rectum	
IVA	Cancer spread to adjacent organs
IVB	Cancer spread to distant organs
Note: Lymph vascular space invasion (LVSI) is not part of the staging, but should be reported.	

Invasive cervical cancer is most often discovered through Pap smears, tentatively diagnosed with a biopsy during a colposcopy, and then further diagnosed using surgery and lymphangiography, CT scan, MRI, ultrasonography, or radio immunodetection methods. The staging system is shown in table 3-16.

Treatment depends on the degree of neoplastic change, the size and location of the lesion, and the extent of metastatic spread. For premalignant cellular changes and CIS, the goal is to kill or remove abnormal cells; these procedures can be done in a clinic with local anesthetic. Invasive carcinoma requires surgery, including removal of the cervix and other affected tissues. Common treatments can be classified as ablative, when the cells are killed without being removed, or excisional in which the abnormal cells are physically removed from the cervix. Ablative therapies are appropriate for lower levels of cervical dysplasia because the treatment does not produce a sample for analysis. However, ablative therapies leave the cervix intact, which may be beneficial for later childbearing. Ablative surgeries include cryotherapy and cold coagulation in which extreme cold is applied to the surface of the cervix. Carbon dioxide laser and electrocoagulation also are used to kill abnormal cells and coagulate vessels supporting their growth. Excisional therapies are appropriate if a more advanced lesion is suspected because they produce tissue for analysis. Excisional therapies include conization, in which a cone-shaped portion of the cervix is removed, and the loop electrosurgical excision procedure (LEEP), in which a small looped wire with electric current generates heat and burns off cancer cells. Heat, cold, or lasers are used in excisional procedures to simultaneously excise the tissue and obliterate abnormal blood vessels.

Table 3.17 Food, Nutrition, Physical Activity, and Cancer of the Cervix.



FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE CERVIX

In the judgement of the Panel, the factors listed below modify the risk of cancer of the cervix. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		
Probable		
Limited — suggestive	Carrots[1]	
Limited — no conclusion	Non-starchy vegetables; fruits; milk; retinol; vitamin E; alcoholism[2]; body fatness; adult attained height.	
Substantial effect on risk unlikely	None identified	

1 Judgements on vegetables and fruits do not include those preserved by salting and/or pickling.
2 Although data suggest that alcoholism is related to increased risk, *the Panel concludes* that this is likely to be due to factors other than alcohol intake itself.

For an explanation of all the terms used in the matrix, please see chapter 3.5.1, the text of this section, and the glossary.

3.11 Ovarian Cancer

Ovarian cancer ranks second after endometrial cancer in the United States. With age, especially in women over the age of 50, the mortality rate increases. Ovarian cancer originates from epithelial cells about 90%.

Pathogenesis

The 5% to 10% that are familial, the majority are associated with the breast can cersusceptibility gene 1 (BRCA1) and a smaller number with mutations of BRCA2 or mismatched repair genes (HNPCC syndrome). Women and families who are more susceptible to cancer may have errors in the ability to repair cellular DNA (Loveday et al., 2011), which allows aberrant cellular proliferation that occurs with repetitive

ovulatory tissue repair in the ovary. In sporadic ovarian cancer, BRCA1 and BRCA2 are rarely mutated. Cells from a variety of intra-abdominal locations, including endometrial tissue and epithelium of the fallopian/ uterine tubes, can attach to the ovary (Figure 3-8). The local ovarian environment, including the ovarian stroma, may then interact with the transplanted cells to enhance cellular growth and encourage metastasis (Figure 3-9) (Schauer et al., 2011).

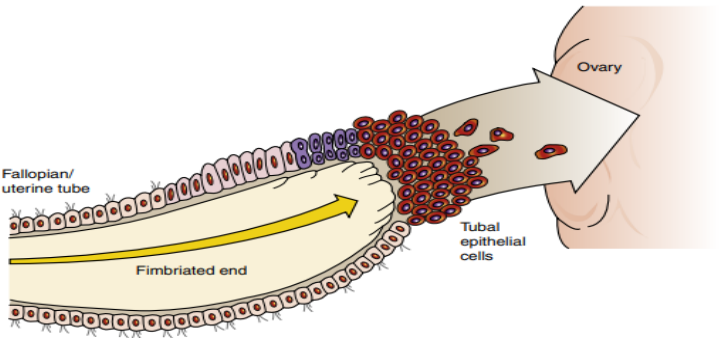


Figure 3.8 Migration of epithelial cells from the fallopian/ uterine tubes to the ovary. (Adapted from Kurman RJ, Shih IM: Am J Surg Pathol 34[3]:433, 2010.

Clinical Manifestations

Ovarian cancer is commonly asymptomatic until the tumors have grown very large. Common first symptoms of ovarian cancer are vague and include persistent abdominal distention, loss of appetite due to early satiety, and pelvic pain. Screening is warranted if women have a new onset of these symptoms that persist for more than 12 days each month. However, many women fail to notice the very first signs of ovarian cancer because they are vague and fairly common in older women. The disease is most commonly diagnosed after metastasis has occurred. Consequently, ovarian cancer is often termed the silent killer. Symptoms of advanced disease include pain and abdominal swelling from the primary ovarian mass or ascites and abdominal distention. Gastrointestinal manifestations may

include dyspepsia, vomiting, and alterations in bowel habits caused by mechanical obstruction. Abnormal vaginal bleeding may occur if the postmenopausal endometrium is stimulated by a hormone-secreting tumor. The tumor also may cause ulcerations through the vaginal wall that result in bleeding. There also can be a feeling of pressure in the pelvis and leg pain. Tumor obstruction of vascular channels can cause venous and, occasionally, arterial thrombosis. Alterations in coagulability also occur, contributing to clot formation. Metastasis often causes pleural effusion.

Evaluation and Treatment

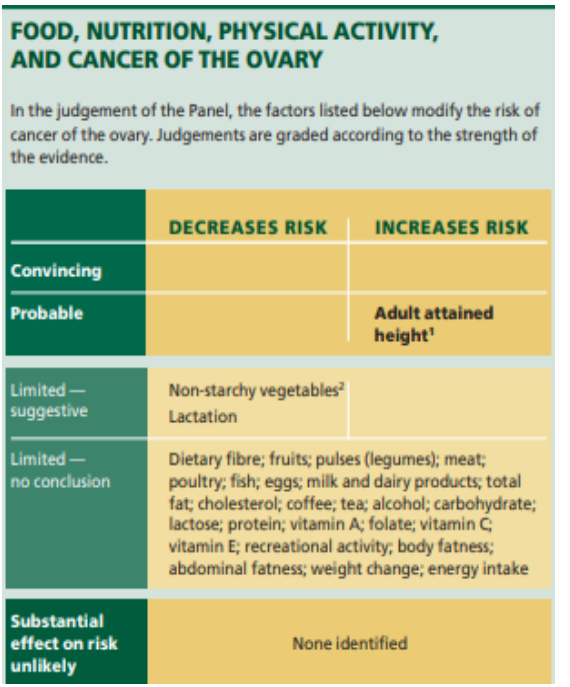
Women with symptoms of disease, as outlined earlier, should be assessed with minimally invasive tests first. Screening commonly begins with a CA-125 blood test looking for specific cancer markers and a transvaginal ultrasound. Diagnosis is confirmed by biopsy and extent of the disease is determined by ultrasound, CT, MRI, or other imaging techniques. Women undergoing surgery for ovarian cancer staging receive a thorough assessment for metastasis. These include an upper gastrointestinal series, barium enema, intravenous pyelogram (IVP), mammography, and lymphography. The initial approach to treatment is surgery, which is performed to determine the stage of disease and to remove as much of the tumor as possible. Future treatment is then customized based on the clinical stage of the cancer, the woman's desires, and the cell type and sensitivity of the cancer cells. Ideally treatment plans are developed and Implemented by a multidisciplinary team from a variety of Disciplines including surgeons, pathologists, and Oncologists.158Radiation therapy and chemotherapy with an agent containing platinum are common treatments.159 Even after initially effective treatment, 55% to 75% of women relapse, and less than 20% survive long term with stage III or IV disease.

Table 3.18 FIGO Staging of Carcinoma of the Ovary

STAGE	CHARACTERISTICS
I	Growth limited to the ovaries
IA	Growth limited to one ovary; no ascites
IA1	No tumor on the external surface; capsule intact (90% 5-year survival with treatment)
IA2	Tumor present on the external surface, or capsule(s) ruptured, or both
IB	Growth limited to both ovaries; no ascites
IB1	No tumor on the external surface; capsule intact
IB2	Tumor present on the external surface, or capsule(s) ruptured, or both
IC	Tumor either stage IA or stage IB, with ascites present or with positive peritoneal washings
II	Growth involving one or both ovaries with pelvic extension
IIA	Extension and/or metastases to the uterus and/or tubes
IIB	Extension to other pelvic tissues
IIC	Tumor either stage IIA or stage IIB but with ascites present or with positive peritoneal washings
III	Growth involving one or both ovaries with intraperitoneal metastases outside the pelvis, or positive retroperitoneal nodes, or both; tumor limited to the true pelvis with histologically proven malignant extension to small bowel or omentum
IV	Growth involving one or both ovaries with distant metastases; if pleural effusion is present, there must be positive cytology to allot a case to stage IV; parenchymal liver metastases indicate stage IV
Special category	Unexplored cases that are thought to be ovarian carcinoma

FIGO, International Federation of Gynecologists and Obstetricians.

Table 3-.19 Food, Nutrition, physical Activity, and Cancer of The Ovary.



FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE OVARY

In the judgement of the Panel, the factors listed below modify the risk of cancer of the ovary. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		
Probable		**Adult attained height[1]**
Limited — suggestive	Non-starchy vegetables[2] Lactation	
Limited — no conclusion	Dietary fibre; fruits; pulses (legumes); meat; poultry; fish; eggs; milk and dairy products; total fat; cholesterol; coffee; tea; alcohol; carbohydrate; lactose; protein; vitamin A; folate; vitamin C; vitamin E; recreational activity; body fatness; abdominal fatness; weight change; energy intake	
Substantial effect on risk unlikely	None identified	

3.12 Endometrial Cancer

Epidemiology and Pathogenesis. In postmenopausal women (peak age 55 to 65 years). Women with excess estrogen are described as type I (endometrioid carcinoma), and women with endometrial atrophy as type II. Endometrioid carcinoma is associated with prolonged estrogen stimulation and mutations in the PTEN, a tumor suppressor gene. other risks are obesity, diabetes, nullipara (not giving birth to children), early menarche, and late menopause. Type II occurs in women who do not show elevated estrogen levels and who are older and in a state of endometrial atrophy rather than hyperplasia. Endometrial cancer is associated with a poorer prognosis than type I carcinoma.

Clinical Features

Bleeding between periods or excessive and prolonged menstrual flow in menstruating women. Any bleeding is not normal in postmenopausal women. Cramps, pelvic discomfort, postcoital bleeding, lower abdominal discomfort, and enlarged lymph nodes. Endometrial biopsy, Direct visualization of the endometrium by hysteroscopy and cervical dilation and curettage of the uterine cavity (D&C). Transvaginal ultrasound is used to determine endometrial thickness as an indicator of hypertrophy and possible neoplastic changes. (Hedrick Ellenson & Pirog, 2021).

Table 3.20 Food, Nutrition, Physical Activity, and Cancer of The Endometrium.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE ENDOMETRIUM

In the judgement of the Panel, the factors listed below modify the risk of cancer of the endometrium. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		**Body fatness**
Probable	**Physical activity[1]**	**Abdominal fatness**
Limited — suggestive	Non-starchy vegetables[2]	Red meat[3] Adult attained height[4]
Limited — no conclusion	Cereals (grains) and their products; dietary fibre; fruits; pulses (legumes); soya and soya products; poultry; fish; eggs; milk and dairy products; total fat; animal fats; saturated fatty acids; cholesterol; coffee; alcohol; carbohydrates; protein; retinol; vitamin C; vitamin E; beta-carotene; lactation; energy intake	
Substantial effect on risk unlikely	None identified	

3.13 Breast Cancer

Worldwide, female breast cancer has now surpassed lung cancer as the most commonly diagnosed cancer. An estimated 2,261,419 new cases of breast cancer were diagnosed in women across the world in 2020. More women in the United States are diagnosed with breast cancer than any other type of cancer, besides skin cancer. The disease accounts for 1 in 3 of new female cancers annually (American Society of Clinical Oncology, 2023b).

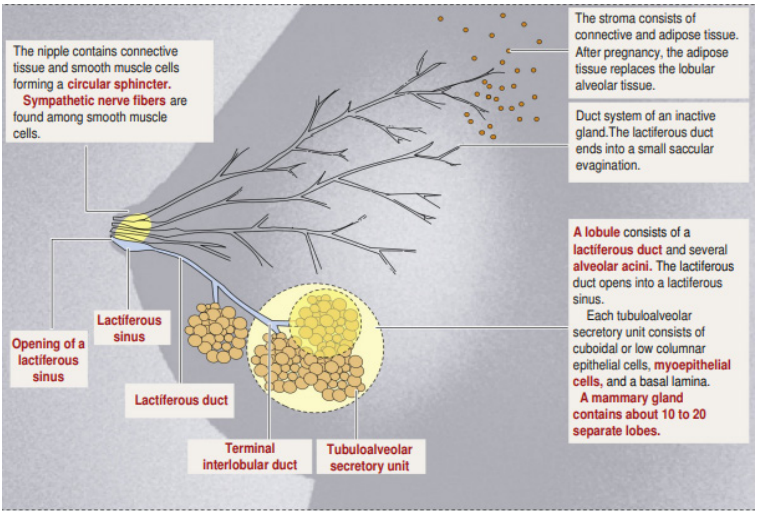
Risk factors include increasing age, personal or family history of breast cancer, history of benign breast disease (ie, primary "atypical" hyperplasia), and hormonal factors, early menarche, late menopause, and not getting pregnant or having a first child after age 30. Obesity (especially after menopause), lack of physical activity, caffeine, moderate to heavy alcohol consumption, smoking, and long-term use of postmenopausal hormone therapy (especially combined estrogen and progestin) as modifiable risk factors. (Lester, 2010).

Detection

Clinically as a mass, puckering, nipple retraction, or unusual discharge, visible thickening or slight changes in the contour of the breast. All women are expected to have an awareness of what their normal breasts look and feel like. Mammography is an effective screening technique for early detection of breast lesions.

Diagnosis and Classification. Physical examination and supporting examinations are mammography, mammary ultrasound, MRI and biopsy. Treatment. Management of breast cancer can be in the form of surgery, radiotherapy, chemotherapy, targeted therapy, or hormonal therapy.

A



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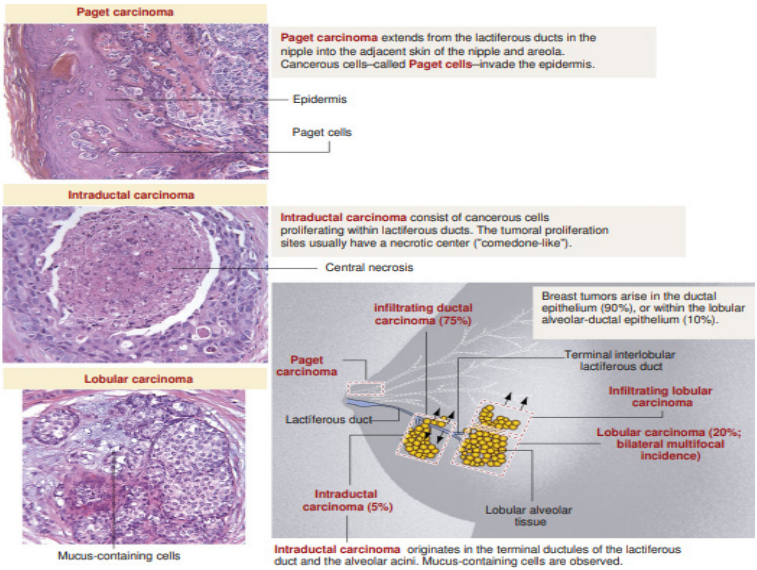

Figure 3.9 Normal Breast and Breast Cancer. A, Normal breast. B, Breast cancer.

Table 3.21 Clinical manifestations of Breast Cancer

CLINICAL MANIFESTATION	PATHOPHYSIOLOGY
Chest pain	Metastasis to the lung
Dilated blood vessels	Obstruction of venous return by a fast-growing tumor; obstruction dilates superficial veins
Dimpling of the skin	Can occur with invasion of the dermal lymphatics because of retraction of Cooper ligament or involvement of the pectoralis fascia
Edema	Local inflammation or lymphatic obstruction
Edema of the arm	Obstruction of lymphatic drainage in the axilla
Hemorrhage	Erosion of blood vessels
Local pain	Local obstruction caused by the tumor
Nipple/areolar eczema	Paget disease
Nipple discharge in a nonlactating woman	Spontaneous and intermittent discharge caused by tumor obstruction
Nipple retraction	Shortening of the mammary ducts
Pitting of the skin (similar to the surface of an orange [peau d'orange])	Obstruction of the subcutaneous lymphatics, resulting in the accumulation of fluid
Reddened skin, local tenderness, and warmth	Inflammation
Skin retraction	Involvement of the suspensory ligaments
Ulceration	Tumor necrosis

Table 3.22 Food, Nutrition, Physical Activity and cancer The Breast.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE BREAST (PREMENOPAUSE)

In the judgement of the Panel, the factors listed below modify the risk of cancer of the breast (premenopause). Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing	Lactation	Alcoholic drinks
Probable	Body fatness	Adult attained height[1] Greater birth weight
Limited — suggestive	Physical activity[2]	
Limited — no conclusion	Cereals (grains) and their products; dietary fibre; potatoes; vegetables; fruits; pulses (legumes); soya and soya products; meat; poultry; fish; eggs; milk and dairy products; fats and oils; total fat; vegetable fat; fatty acid composition, trans-fatty acids; cholesterol; sugar (sucrose); other sugars; sugary foods and drinks; coffee; tea; carbohydrate; starch; glycaemic index; protein; vitamin A; riboflavin; vitamin B6; folate; vitamin B12; vitamin C; vitamin D; vitamin E; calcium; iron; selenium; carotenoids; isoflavones; dichlorodiphenyldichloroethylene; dichlorodiphenyltrichloroethane; dieldrin; hexachlorobenzene; hexachlorocyclohexane; *trans*-nonachlor; polychlorinated biphenyls; dietary patterns; culturally defined diets; adult weight gain; energy intake; being breastfed	
Substantial effect on risk unlikely	None identified	

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE BREAST (POSTMENOPAUSE)

In the judgement of the Panel, the factors listed below modify the risk of cancer of the breast (postmenopause). Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing	Lactation	Alcoholic drinks Body fatness Adult attained height[1]
Probable	Physical activity[2]	Abdominal fatness Adult weight gain
Limited — suggestive		Total fat
Limited — no conclusion	Cereals (grains) and their products; dietary fibre; potatoes; vegetables and fruits; pulses (legumes); soya and soya products; meat; poultry; fish; eggs; milk and dairy products; fats and oils; vegetable fat; fatty acid composition; cholesterol; sugar (sucrose); sugary foods and drinks; coffee; tea; carbohydrate; starch; glycaemic index; protein; vitamin A; riboflavin; vitamin B6; folate; vitamin B12; vitamin C; vitamin D; vitamin E; calcium; iron; selenium; carotenoids; isoflavones; dichlorodiphenyldichloroethylene; dichlorodiphenyltrichloroethane; dieldrin; hexachlorobenzene; hexachlorocyclohexane; *trans*-nonachlor; polychlorinated biphenyls; dietary patterns; culturally defined diets; birth weight; birth length; energy intake; being breastfed	
Substantial effect on risk unlikely	None identified	

3.14 Cancer of the Prostate

The most common cancer among men is prostate cancer. It is the fourth most frequently diagnosed cancer in the world. The mean age at diagnosis is 66 years in approximately 60% of cases. The number of new cases diagnosed in black men is 73% higher than the number of new cases diagnosed in white men (American Society of Clinical Oncology, 2023i).

Etiology and Pathogenesis

There is a gradual process involving genes that control cell differentiation and growth. (Damajanov, 2008; Epstein, 2010b; Nelson et al., 2003). Due to the amplification or mutation of the androgen receptor, prostate cells continue to proliferate without being followed by the rate of apoptosis. Age, ethnicity, and genetic factors are risks.

Clinical Features.

Urgency, frequency, nocturia, hesitancy, dysuria, hematuria, or blood in the ejaculate. The prostate may be nodular and fixed on digital rectal examination. Bone metastases are often characterized by low back pain

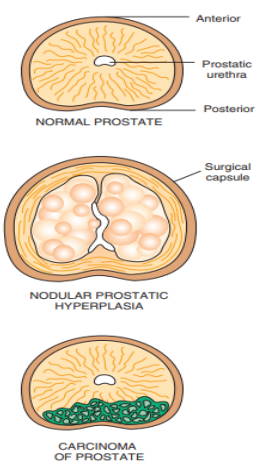

Figure 3.10 Normal prostate, nodular benign prostatic hyperplasia, and cancer of the prostate.

Diagnosis

Physical examination and confirmed through the biopsy method (Cooperberg et al., 2013; Cornett & Dea, 2013). Transrectal ultrasound, the currently available screening tests are digital rectal examination, PSA test, and transrectal ultrasound. Treatment Surgery, radiation therapy, and hormonal manipulation.

Table 3.23 Food, Nutrition, Physical Activity, and Cancer of The Prostate.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE PROSTATE

In the judgement of the Panel, the factors listed below modify the risk of cancer of the prostate. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		
Probable	**Foods containing lycopene[1,2]** **Foods containing selenium[1]** **Selenium[3]**	**Diets high in calcium[4,5]**
Limited — suggestive	Pulses (legumes)[6] Foods containing vitamin E[1] Alpha-tocopherol[7]	Processed meat[8] Milk and dairy products[5]
Limited — no conclusion	Cereals (grains) and their products; dietary fibre; potatoes; non-starchy vegetables; fruits; meat; poultry; fish; eggs; total fat; plant oils; sugar (sucrose); sugary foods and drinks; coffee; tea; alcohol; carbohydrate; protein; vitamin A; retinol; thiamin; riboflavin; niacin; vitamin C; vitamin D; gamma-tocopherol; vitamin supplements; multivitamins; iron; phosphorus; zinc; other carotenoids; physical activity; energy expenditure; vegetarian diets; Seventh-day Adventist diets; body fatness; abdominal fatness; birth weight; energy intake	
Substantial effect on risk unlikely	Beta-carotene[1,9]	

3.15 Lung Cancer

This cancer is one of the most common cancers in Indonesia. Globally, lung cancer is the first cause of cancer death in men and the second cause of cancer death in women. Normal bronchial cells become cancer cells because carcinogenic

chemicals in tobacco smoke trigger genetic changes (de Groot & Munden, 2012).

Table 3.24 Characteristics of Lung Cancers

TUMOR TYPE	GROWTH RATE	METASTASIS	MEANS OF DIAGNOSIS	CLINICAL MANIFESTATIONS AND TREATMENT
Squamous cell carcinoma	Slow	Late; mostly to hilar lymph nodes	Biopsy, sputum analysis, bronchoscopy, electron microscopy, immunohistochemistry	Cough, sputum production, airway obstruction; treated surgically, chemotherapy adjunctive
Adenocarcinoma	Moderate	Early	Radiography, fiberoptic bronchoscopy, electron microscopy	Pleural effusion; treated surgically, chemotherapy adjunctive
Large cell carcinoma	Rapid	Early and widespread	Sputum analysis, bronchoscopy, electron microscopy (by exclusion of other cell types)	Chest wall pain, pleural effusion, cough, sputum production, hemoptysis, airway obstruction resulting in pneumonia (if airways involved); treated surgically
Small cell (oat cell) carcinoma	Very rapid	Very early; to mediastinum or distally in lung	Radiography, sputum analysis, bronchoscopy, electron microscopy, immunohistochemistry, and clinical manifestations (cough, chest pain, dyspnea, hemoptysis, localized wheezing)	Airway obstruction, signs and symptoms of excessive hormone secretion; treated by chemotherapy and ionizing radiation to thorax and central nervous system

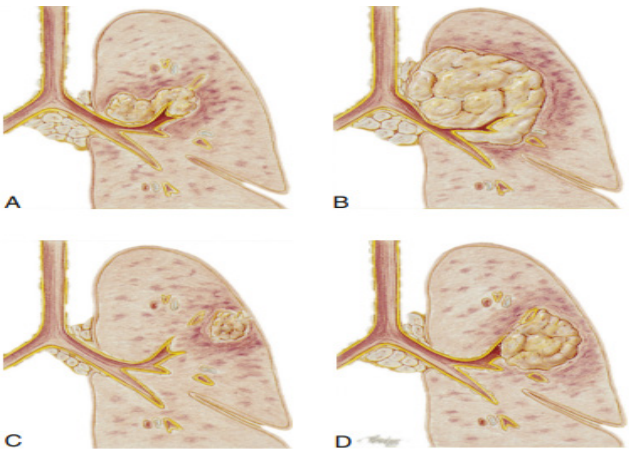

Figure 3.11 Cancer of the Lung. A, Squamous (epidermoid) cell carcinoma. B, Small cell (oat cell) carcinoma. C, Adenocarcinoma. D, Large cell carcinoma. (From Des Jardins T, Burton GG: Clinical manifestations and assessment of respiratory disease, ed 3, St Louis, 1995, Mosby.)

Manifestations Divided into three categories based on: (1) involvement of the lung and adjacent structures; (2) local spread and metastatic effects; and (3) non-metastatic paraneoplastic manifestations. Chronic cough, shortness of breath, and wheezing and hemoptysis (S. Wang, 2013).

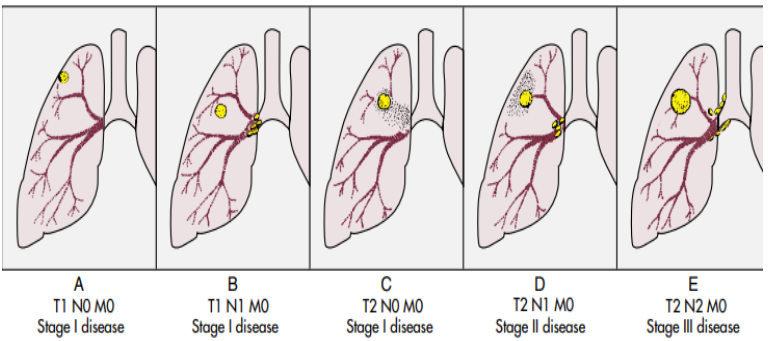

Figure 3.12 The TNM Classification System for Lung Cancer

Diagnosis and Treatment

Physical examination, chest x-ray, bronchoscopy, sputum cytology, percutaneous needle biopsy of lung tissue, and irregular lymph node biopsy, MRI, and ultrasonography are used to locate the lesion and evaluate the extent of disease (Wender et al., 2013).

Table 3.25 Food, Nutrition, Physical Activity, and Cancer of The Lung.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE LUNG

In the judgement of the Panel, the factors listed below modify the risk of cancer of the lung. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		**Arsenic in drinking water[1]** **Beta-carotene supplements[2]**
Probable	**Fruits[3]** **Foods containing carotenoids[4]**	
Limited — suggestive	Non-starchy vegetables[3] Foods containing selenium[4] Foods containing quercetin[4] Selenium[5] Physical activity[6]	Red meat[7] Processed meat[8] Total fat Butter Retinol supplements[2] Low body fatness
Limited — no conclusion	Cereals (grains) and their products; starchy tubers; dietary fibre; pulses (legumes); poultry; fish; eggs; milk and dairy products; total fat; animal fats; plant oils; soft drinks; coffee; tea; alcohol; preservation, processing, and preparation; carbohydrate; protein vitamin A; thiamin; riboflavin; niacin; vitamin B6; folate; vitamin C; vitamin E; multivitamins; calcium; copper; iron; zinc; pro-vitamin A carotenoids; lycopene; flavonoids; culturally-defined diets; body size, shape, and composition (except low body fatness); energy intake	
Substantial effect on risk unlikely	None identified	

3.16 Brain Tumors

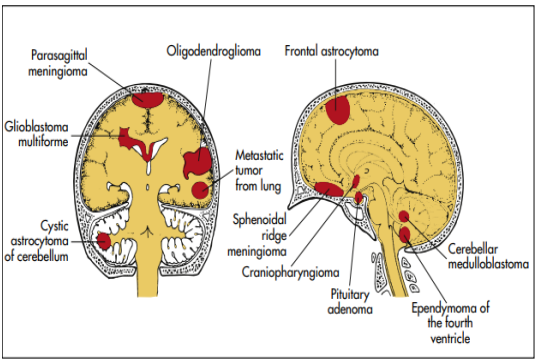

Figure 3.13 Common Sites of Intracranial Tumors

The brain tumor mortality rate is 4.4 per 100,000 in men and women per year. The death rate is based on cases of death due to brain tumors during the 2016–2020 period. The estimated mortality rate for brain tumors in 2022 globally is estimated at 18,250 cases of death. (American Society of Clinical Oncology, 2021).

Cranial tumors cause local and generalized clinical manifestations. The local effects are caused by the destructive action of the tumor itself on a particular site in the brain and compression causing decreased cerebral blood flow. The effects arevaried and include seizures, visual disturbances, unstable gait, and cranial nerve dysfunction. The generalized effects result from increased ICP (Figure 3-15). Localized intracranial lesions that occupy space within the skull are brain tumors. Exposure to ionizing radiation as a cause of primary brain tumors (American Brain Tumor Association, 2023).Secondary tumor or metastasis to the brain from systemic primary cancer.

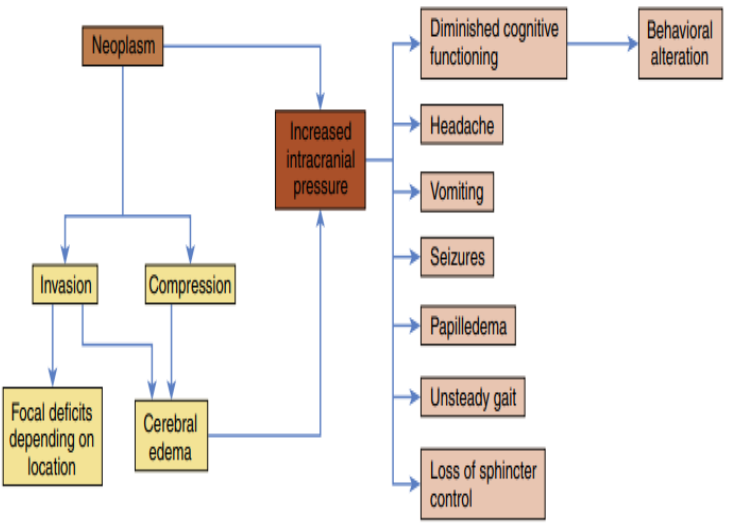

Figure 3.14 Origin of Clinical Manifestations Associated with an Intracranial Neoplasm

Manifestations

Headache, nausea, vomiting, mental changes, papilledema, visual disturbances (eg, diplopia), changes in sensory and motor function, and seizures (Hickey, 2009; Kelly, 2013).

Diagnosis and Treatment

Physical and neurological examination, visual field examination and fundoscopy, electroencephalography (EEG), MRI, cerebral angiography, magnetic resonance angiography and CT angiography. Surgery, irradiation, and chemotherapy are common methods of treating brain tumors.

Table 3.26 Classification of Brain Tumors in Adults

I. Intracerebral Tumors
- A. Gliomas—infiltrate any portion of the brain; most common type of brain tumor
  1. Astrocytomas (grades I and II)
  2. Glioblastoma multiforme (astrocytoma grades III and IV)
  3. Oligodendrocytoma (low and high grades)
  4. Ependymoma (grades I to IV)
  5. Medulloblastoma

II. Tumors Arising From Supporting Structures
- A. Meningiomas
- B. Neuromas (acoustic neuroma, schwannoma)
- C. Pituitary adenomas

III. Developmental Tumors
- A. Angiomas
- B. Dermoid, epidermoid, teroma, craniopharyngioma

IV. Metastatic Lesions

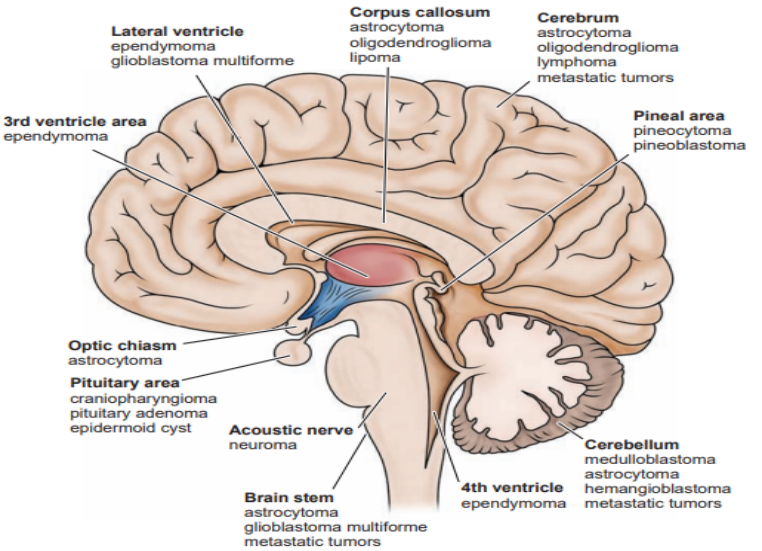

Figure 3.15 Common Brain Tumor Sites

Nursing Management

The patient with a brain tumor may be at increased risk for aspiration as a result of cranial nerve dysfunction. Preoperatively, the gag reflex and ability to swallow are evaluated. In patients with diminished gag response, care includes teaching the patient to direct food and fluids toward the unaffected side, having the patient sit upright to eat, offering a semisoft diet, and having suction readily available. The effects of increased ICP caused by the tumor mass. perform neurologic examination, prevent rapid rise in ICP, self-care supervision and assistance, and ongoing monitoring and intervention for injury prevention.

3.17 Bladder Tumors

Bladder cancer is the 10th most common cancer worldwide. It is the 6th most common cancer in men and the 17th most common cancer in women. There were more than 573.000 new cases of bladder cancer in 2020 (Oncology, 2023).

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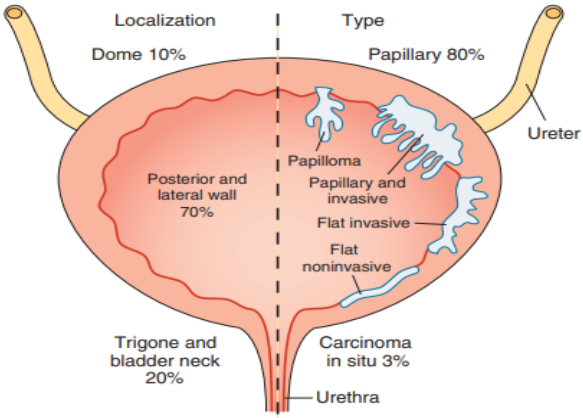


Figure 3-16 Urothelial neoplasms. Most tumors are located on the posterior and lateral walls; trigone and bladder neck are involved less commonly. Malignant tumors may be papillary or flat. Both flat and papillary tumors may be invasive or non-invasive. Benign transitional cell papillomas are rare.

Etiology and Pathogenesis

Bladder cancer is derived from the transitional (urothelial) epithelial cells that line the bladder. These tumors range from benign papillomas and low-grade papillary urothelial carcinoma to invasive urothelial cell carcinoma and highly malignant tumors. Manifestations are Gross hematuria with Frequency, urgency, and dysuria.

Diagnosis and Treatment

Cytology, excretory urography, cystoscopy, and biopsy. Ultrasonography, CT scan, and MRI (Epstein, 2010a). In the surgical treatment of superficial bladder cancer, therapeutic agents directly instilled into the bladder are intravascular chemotherapy.

Table 3.27 Staging of Bladder Carcinoma (TNM System)

STAGE	DESCRIPTION
Primary Tumor	
T0	No primary tumor identified
Ta	Noninvasive papillary carcinoma—not in bladder muscle
Tis	Carcinoma in situ (CIS)
T1	Tumor invades connective tissue
T2	Tumor invades detrusor muscle
T3	Invasion of fatty tissue around bladder
T4	Tumor has invaded adjacent structures
Region of Lymph Nodes	
N0	No lymph node involvement
N1 to N3	Lymph node metastasis to pelvic or adjacent region
Distant Metastasis	
M0	No metastasis
M1	Distant metastasis

Table 3.28 Food, Nutrition, Physical Activity, and Cancer of The Bladder.

FOOD, NUTRITION, PHYSICAL ACTIVITY, AND CANCER OF THE BLADDER

In the judgement of the Panel, the factors listed below modify the risk of cancer of the bladder. Judgements are graded according to the strength of the evidence.

	DECREASES RISK	INCREASES RISK
Convincing		
Probable		
Limited — suggestive	Milk[1]	Arsenic in drinking water[2]
Limited — no conclusion	Cereals (grains) and their products; vegetables; fruits; pulses (legumes); meat; poultry; fish; eggs; total fat; butter; dietetic foods; soft drinks; diet drinks; fruit juices; coffee; tea; caffeine; alcohol; chlorinated surface water; total fluid intake; sweeteners; frying; carbohydrate; protein; vitamin A; folate; vitamin C; vitamin E; multivitamin supplements; selenium; beta-carotene; alpha-carotene; lycopene; beta-cryptoxanthin; lutein; zeaxanthin; flavonoids; physical activity; body fatness; energy intake	
Substantial effect on risk unlikely	None identified	

3.18 Thyroid Cancer

Medical Management

The treatment of choice for thyroid carcinoma is surgical removal. Total or near-total thyroidectomy is performed if possible. Modified neck dissection or more extensive radical neck dissection is performed if there is lymph node involvement. Efforts are made to spare parathyroid tissue to reduce the risk of postoperative hypocalcemia and tetany. After surgery, ablation procedures are carried out with radioactive iodine to eradicate residual thyroid tissue if the tumor is radiosensitive. Radioactive iodine also maximizes the chance of discovering thyroid metastasis at a later date if total-body scans are carried out.

Nursing Management

Important preoperative goals are to gain the patient's confidence and reduce anxiety. Often, the patient's home life has become tense because of his or her restlessness, irritability, and nervousness secondary to hyperthyroidism. Efforts are necessary to protect the patient from such tension and stress to avoid precipitating thyroid storm. If the patient reports increased stress when with family or friends, suggestions are made to limit contact with them. Quiet and relaxing forms of recreation or occupational therapy may be helpful.

Table 3.29 Types of Thyroid Cancer

Type of Thyroid Cancer	Incidence (%)	Characteristics
Papillary adenocarcinoma	70	Most common and least aggressive Asymptomatic nodule in a normal gland Starts in childhood or early adult life, remains localized Metastasizes along the lymphatics if untreated More aggressive in the elderly
Follicular adenocarcinoma	15	Appears after 40 y of age Encapsulated; feels elastic or rubbery on palpation Spreads through the bloodstream to bone, liver, and lung Prognosis is not as favorable as for papillary adenocarcinoma
Medullary	5	Appears after 50 y of age Occurs as part of multiple endocrine neoplasia (MEN) Hormone-producing tumor causing endocrine dysfunction symptoms Metastasizes by lymphatics and bloodstream Moderate survival rate
Anaplastic	5	50% of anaplastic thyroid carcinomas occur in patients older than 60 y Hard, irregular mass that grows quickly and spreads by direct invasion to adjacent tissues May be painful and tender Survival for patients with anaplastic cancer is usually less than 6 mo
Thyroid lymphoma	5	Appears after age 40 y May have history of goiter, hoarseness, dyspnea, pain, and pressure Good prognosis

3.19 Testicular cancer

Most commonly diagnosed cancer in men between the ages of 20 and 34 years. In 2020, there are an estimated 3,000 new cases of this disease in the United States in 2020.It is estimated that 470 deaths from this disease will occur in the United States in 2023. These deaths are either from cancer that spread from the testicles to other parts of the body and could not be effectively treated with chemotherapy, radiation therapy, and/or surgery or from complications from treatment. In 2020, an estimated 9,334 people worldwide died from testicular cancer (American Society of Clinical Oncology, 2023j).

Classification of Testicular Tumors

1. Germinal Tumors

   Germinal tumors make up approximately 90% of all cancers of the testis; germinal tumors are further classified as seminomas or non-seminomas. These cancers grow from the germ cells that produce sperm, thus the name germinal tumors. Seminomas are slow-growing forms of testicular cancer that are usually found in men in their 30s and 40s. Although seminomas can spread to the lymph nodes, the cancer is usually localized in the testes. Non-seminomas are more common and tend to grow more quickly than seminomas. Non-seminomas are often made up of different cell types and are identified according to the cells in which they start to grow.
2. Nongerminal Tumors
3. Secondary Testicular

   Tumors Secondary testicular tumors are those that have metastasized to the testicle from other organs. Lymphoma is the most common cause of secondary testicular cancer. Cancers may also spread to the testicles from the prostate gland, lung, skin (melanoma), kidney, and other organs. The prognosis with these cancers is usually poor because they typically also spread to other organs.

Clinical Manifestations. Back pain, abdominal pain, weight loss, and general weakness may result from metastases. Painless enlargement of the testicle is a significant diagnostic finding, with a feeling of heaviness in the scrotum and groin area.

An evaluation of the mass or unexplained testicular enlargement or pain is the key to early detection (Gilligan, 2007). Teaches TSE, and encourages men to seek medical attention if a testicle becomes hard, enlarged, atrophied, nodular, or painful. Removal of the affected testis by orchiectomy through an inguinal incision with high spermatic cord ligation as well as the option of implanting a testicular prosthesis during orchiectomy (Tanagho & McAninch, 2008).

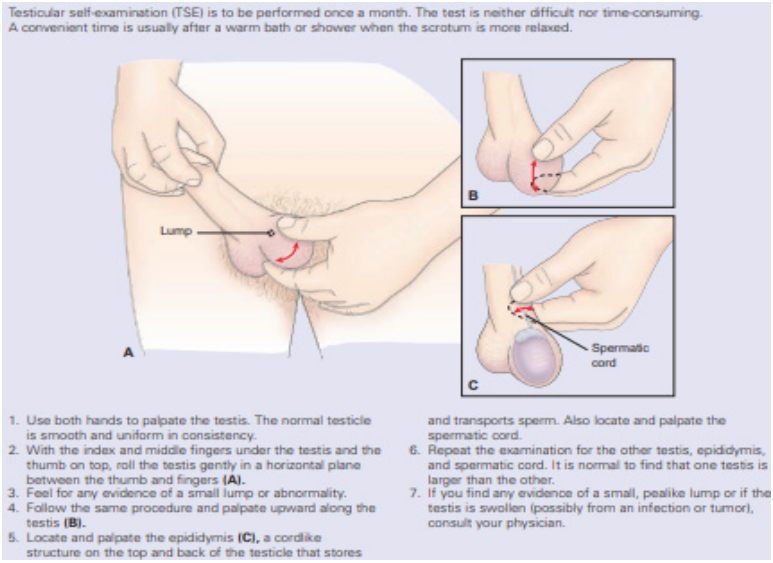


Figure 3.17 Testicular Self-Examination

CHAPTER 4
ONCOLOGIC NURSING

Cancerous cells are described as malignant neoplasms. They demonstrate uncontrolled cell growth that follows no physiologic demand. Benign and malignant growths are classified and named by tissue of origin (Nugent et al., 2014).

Benign and malignant cells differ in many cellular growth characteristics, including the method and rate of growth, ability to metastasize or spread, general effects, destruction of tissue, and ability to cause death. The degree of anaplasia (lack of differentiation of cells) ultimately determines the malignant potential (Nugent et al., 2014).

4.1 Characteristic of Malignant Cells

Cell membranes, specialized proteins, nuclei, chromosomal abnormalities, and the rate of mitosis and growth are the cellular characteristics of cancer cells.

Table 4.1 Tumors and Tissue Types

TISSUE TYPE	BENIGN TUMORS	MALIGNANT TUMORS
Epithelial Surface Glandular	Papilloma Adenoma	Squamous cell carcinoma Adenocarcinoma

Connective Fibrous Adipose Cartilage Bone Blood vessels Lymph vessels Lymph tissue	Fibroma Lipoma Chondroma Osteoma Hemangioma Lymphangioma	Fibrosarcoma Liposarcoma Chondrosarcoma Osteosarcoma Hemangiosarcoma Lymphangiosarcoma Lymphosarcoma
Muscle Smooth Striated	Leiomyoma Rhabdomyoma	Leiomyosarcoma Rhabdomyosarcoma
Neural Tissue Nerve cell Glial tissue Nerve sheaths Meninges	Neuroma Glioma (benign) Neurilemmoma Meningioma	Neuroblastoma Glioblastoma, astrocytoma, medulloblastoma, oligodendroglioma Neurilemmal sarcoma Meningeal sarcoma
Hematologic Granulocytic Erythrocytic Plasma cells Lymphocytic Monocytic		Myelocytic leukemia Erythrocytic leukemia Multiple myeloma Lymphocytic leukemia or lymphoma Monocytic leukemia
Endothelial Tissue Blood vessels Lymph vessels Endothelial lining	Hemangioma Lymphangioma	Hemangiosarcoma Lymphangiosarcoma Ewing's sarcoma

a. **The cell membranes** are altered in cancer cells, which affects fluid movement in and out of the cell. The cell membrane of malignant cells also contains…

b. **Proteins** called tumor-specific antigens (for example, carcinoembryonic antigen and prostate-specific antigen), which develop as they become **less differentiated** (mature) over time. These proteins distinguish the malignant cell from a benign cell of the same tissue type. They may be useful in measuring the extent of disease in a person and in tracking the course of illness during treatment or relapse. Malignant cellular membranes also contain less fibronectin, a cellular cement. They are therefore less cohesive and do not adhere to adjacent cells readily.

c. Typically, **nuclei** of cancer cells are large and irregularly shaped (pleomorphism). Nucleoli, structures within the nucleus that house ribonucleic acid (RNA), are larger and more numerous in malignant cells, perhaps because of increased RNA synthesis.

d. **Chromosomal abnormalities** (translocations, deletions, additions) and fragility of chromosomes are commonly found when cancer cells are analyzed.

e. **Mitosis** (cell division) occurs more frequently in malignant cells than in normal cells.

f. As the cells **grow** and divide, more glucose and oxygen are needed. If glucose and oxygen are unavailable, malignant cells use anaerobic metabolic channels to produce energy, which makes the cells less dependent on the availability of a constant oxygen supply.

g. Specific affinity for certain malignant cells to bind to molecules in specific body tissue.

h. Metastasis is the dissemination or spread of malignant cells from the primary tumor to distant sites by direct spread of tumor cells to body cavities or through lymphatic and blood circulation. Tumors growing in or penetrating body

cavities may shed cells or emboli that travel within the body cavity and seed the surfaces of other organs. This can occur in ovarian cancer when malignant cells enter the peritoneal cavity and seed the peritoneal surfaces of such abdominal organs as the liver or pancreas.

i. Invasion, which refers to the growth of the primary tumor into the surrounding host tissues, occurs in several ways. Mechanical pressure exerted by rapidly proliferating neoplasms may force fingerlike projections of tumor cells into surrounding tissue and interstitial spaces. Malignant cells are less adherent and may break off from the primary tumor and invade adjacent structures. Malignant cells are thought to possess or produce specific destructive enzymes (proteinases), such as collagenases (specific to collagen), plasminogen activators (specific to plasma), and lysosomal hydrolyses. These enzymes are thought to destroy surrounding tissue, including the structural tissues of the vascular basement membrane, facilitating invasion of malignant cells. The mechanical pressure of a rapidly growing tumor may enhance this process.

Metastatic Mechanisms

Lymph and blood are key mechanisms by which cancer cells spread. Angiogenesis, a mechanism by which the tumor cells are ensured a blood supply, is another important process.

a. Lymphatic Spread The most common mechanism of metastasis is lymphatic spread, which is transport of tumor cells through the lymphatic circulation. Tumor emboli enter the lymph channels by way of the interstitial fluid that communicates with lymphatic fluid. Malignant cells also may penetrate lymphatic vessels by invasion. After entering the lymphatic circulation, malignant cells either lodge in the lymph nodes or pass between lymphatic and venous circulation. Tumors arising in areas of the body with rapid and extensive lymphatic circulation are at high risk for metastasis through lymphatic channels. Breast

tumors frequently metastasize in this manner through axillary, clavicular, and thoracic lymph channels.

b. **Hematogenous Spread** Another metastatic mechanism is hematogenous spread, by which malignant cells are disseminated through the bloodstream. Hematogenous spread is directly related to the vascularity of the tumor. Few malignant cells can survive the turbulence of arterial circulation, insufficient oxygenation, or destruction by the body's immune system. In addition, the structure of most arteries and arterioles is far too secure to permit malignant invasion. Those malignant cells that do survive this hostile environment are able to attach to endothelium and attract fibrin, platelets, and clotting factors to seal themselves from immune system surveillance. The endothelium retracts, allowing the malignant cells to enter the basement membrane and secrete lysosomal enzymes. These enzymes then destroy surrounding body tissues and thereby allow implantation.

c. **Angiogenesis** Malignant cells also have the ability to induce the growth of new capillaries from the host tissue to meet their needs for nutrients and oxygen. This process is referred to as angiogenesis. It is through this vascular network that tumor emboli can enter the systemic circulation and travel to distant sites. Large tumor emboli that become trapped in the microcirculation of distant sites may further metastasize to other sites. Research into ways to prevent angiogenesis is ongoing.

Carcinogenesis

Malignant transformation, or carcinogenesis, is thought to be at least a three-step cellular process: initiation, promotion, and progression.

a. **Initiation**, the first step, initiators (carcinogens), such as chemicals, physical factors, and biologic agents, escape normal enzymatic mechanisms and alter the genetic structure of the cellular DNA. Normally, these alterations

are reversed by DNA repair mechanisms, or the changes initiate programmed cellular suicide (apoptosis). Occasionally, cells escape these protective mechanisms, and permanent cellular mutations occur. These mutations usually are not significant to cells until the second step of carcinogenesis.

b. Promotion, repeated exposure to promoting agents (co-carcinogens) causes the expression of abnormal or mutant genetic information even after long latency periods. Latency periods for the promotion of cellular mutations vary with the type of agent and the dosage of the promoter as well as the innate characteristics of the target cell.

Cellular oncogenes, present in all mammalian systems, are responsible for the vital cellular functions of growth and differentiation. Cellular proto-oncogenes are present in cells and act as an "on switch" for cellular growth. Similarly, cancer suppressor genes "turn off" or regulate unneeded cellular proliferation. When the suppressor genes become mutated, rearranged, or amplified or lose their regulatory capabilities, malignant cells are allowed to reproduce. The p53 gene is a tumor suppressor gene that is frequently mutated in many human cancers. This gene regulates whether cells will repair or die after DNA damage. Mutant p53 gene is associated with a poor prognosis and may be associated with determining response to treatment. Once this genetic expression occurs in cells, the cells begin to produce mutant cell populations that are different from their original cellular ancestors.

c. Progression is the third step of cellular carcinogenesis. The cellular changes formed during initiation and promotion now exhibit increased malignant behavior. These cells now show a propensity to invade adjacent tissues and to metastasize. Agents that initiate or promote cellular transformation are referred to as carcinogens.

Etiology

a. **Viruses and Bacteria** Viruses as a cause of human cancers are hard to determine because viruses are difficult to isolate. Infectious causes are considered or suspected, however, when specific cancers appear in clusters. Viruses are thought to incorporate themselves in the genetic structure of cells, thus altering future generations of that cell population— perhaps leading to a cancer. For example, the Epstein-Barr virus is highly suspect as a cause in Burkitt's lymphoma, nasopharyngeal cancers, and some types of non-Hodgkin's lymphoma and Hodgkin's disease.

   Herpes simplex virus type II, cytomegalovirus, and human papillomavirus types 16, 18, 31, and 33 are associated with dysplasia and cancer of the cervix. The hepatitis B virus is implicated in cancer of the liver; the human T-cell lymphotropic virus may be a cause of some lymphocytic leukemias and lymphomas; and the human immunodeficiency virus (HIV) is associated with Kaposi's sarcoma. The bacterium Helicobacter pylori has been associated with an increased incidence of gastric malignancy, perhaps secondary to inflammation and injury of gastric cells.

b. **Physical Agents** Physical factors associated with carcinogenesis include exposure to sunlight or radiation, chronic irritation or inflammation, and tobacco use.
   1. Excessive exposure to the ultraviolet rays of the sun, especially in fair-skinned, blue- or green-eyed people, increases the risk for skin cancers. Factors such as clothing styles (sleeveless shirts or shorts), use of sunscreens, occupation, recreational habits, and environmental variables, including humidity, altitude, and latitude, all play a role in the amount of exposure to ultraviolet light.
   2. Exposure to ionizing radiation can occur with repeated diagnostic x-ray procedures or with radiation therapy

used to treat disease. Fortunately, improved x-ray equipment appropriately minimizes the risk for extensive radiation exposure. Radiation therapy used in disease treatment or exposure to radioactive materials at nuclear weapon manufacturing sites or nuclear power plants is associated with a higher incidence of leukemias, multiple myeloma, and cancers of the lung, bone, breast, thyroid, and other tissues. Background radiation from the natural decay processes that produce radon has also been associated with lung cancer. Homes with high levels of trapped radon should be ventilated to allow the gas to disperse into the atmosphere.

c. **Chemical Agents** About 75% of all cancers are thought to be related to the environment. Tobacco smoke, thought to be the single most lethal chemical carcinogen, accounts for at least 30% of cancer deaths (Heath & Fontham, 2001). Smoking is strongly associated with cancers of the lung, head and neck, esophagus, pancreas, cervix, and bladder. Tobacco may also act synergistically with other substances, such as alcohol, asbestos, uranium, and viruses, to promote cancer development.
   1. Chewing tobacco is associated with cancers of the oral cavity and primarily occurs in men younger than 40 years of age. Many chemical substances found in the workplace have proved to be carcinogens or co-carcinogens. The extensive list of suspected chemical substances continues to grow and includes aromatic amines and aniline dyes; pesticides and formaldehydes; arsenic, soot, and tars; asbestos; benzene; betel nut and lime; cadmium; chromium compounds; nickel and zinc ores; wood dust; beryllium compounds; and polyvinyl chloride.
   2. Most hazardous chemicals produce their toxic effects by altering DNA structure in body sites distant from chemical exposure. The liver, lungs, and kidneys are the organ systems most often affected, presumably because of their roles in detoxifying chemicals.

d. **Genetic and Familial Factors** Almost every cancer type has been shown to run in families. This may be due to genetics, shared environments, cultural or lifestyle factors, or chance alone. Genetic factors play a role in cancer cell development. Abnormal chromosomal patterns and cancer have been associated with extra chromosomes, too few chromosomes, or translocated chromosomes. Specific cancers with underlying genetic abnormalities include Burkitt's lymphoma, chronic myelogenous leukemia, meningiomas, acute leukemias, retinoblastomas, Wilms' tumor, and skin cancers, including malignant melanoma.

   Approximately 5% to 10% of cancers of adulthood and childhood display a familial predisposition. Inherited cancer syndromes, such as premenopausal breast cancer, tend to occur at an early age and at multiple sites in one organ or pair of organs. In cancers with a familial predisposition, individuals may develop multiple cancers; commonly, two or more first-degree relatives share the same cancer type. Cancers associated with familial inheritance include retinoblastomas, nephroblastomas, pheochromocytomas, malignant neurofibromatosis, and breast, ovarian, endometrial, colorectal, stomach, prostate, and lung cancers. In 1994, the BRCA-1 gene was identified; it is linked to breast and ovarian cancer syndrome. The BRCA-2 gene, which has also been identified, is associated with early-onset breast cancer (Nogueira & Appling, 2000). Work continues to identify other specific genes related to cancer incidence (Greco, 2000).

e. **Dietary Factors** Dietary factors are thought to be related to 35% of all environmental cancers (Heath & Fontham, 2001). Dietary substances can be proactive (protective), carcinogenic, or co-carcinogenic. The risk for cancer increases with long-term ingestion of carcinogens or co-carcinogens or chronic absence of proactive substances in the diet.

1. Dietary substances associated with an increased cancer risk include fats, alcohol, salt-cured or smoked meats, and foods containing nitrates and nitrites, and a high caloric dietary intake. Food substances that appear to reduce cancer risk include high-fiber foods, cruciferous vegetables (cabbage, broccoli, cauliflower, Brussels sprouts, kohlrabi), carotenoids (carrots, tomatoes, spinach, apricots, peaches, dark-green and deep-yellow vegetables), and possibly vitamins E and C, zinc, and selenium.
2. Obesity is associated with endometrial cancer and possibly postmenopausal breast cancers. Obesity may also increase the risk for cancers of the colon, kidney, and gallbladder.

f. **Hormonal Agents** Tumor growth may be promoted by disturbances in hormonal balance either by the body's own (endogenous) hormone production or by administration of exogenous hormones. Cancers of the breast, prostate, and uterus are thought to depend on endogenous hormonal levels for growth. Diethylstilbestrol (DES) has long been recognized as a cause of vaginal carcinomas. Oral contraceptives and prolonged estrogen replacement therapy are associated with increased incidence of hepatocellular, endometrial, and breast cancers, whereas they appear to decrease the risk for ovarian and endometrial cancers. The combination of estrogen and progesterone appears safest in decreasing the risk for endometrial cancers. Hormonal changes with reproduction are also associated with cancer incidence. Increased numbers of pregnancies are associated with a decreased incidence of breast, endometrial, and ovarian cancers.

Reducing the risk of cancer in healthy people is primary prevention. Detection and screening to achieve early diagnosis and prompt intervention to stop the cancer process is secondary prevention.

Table 4.2 Imaging Tests Used to Detect Cancer

TEST	DESCRIPTION	DIAGNOSTIC USES
Tumor marker identification	Analysis of substances found in blood or other body fluids that are made by the tumor or by the body in response to the tumor.	Breast, colon, lung, ovarian, testicular, prostate cancers
Magnetic resonance imaging (MRI)	Use of magnetic fields and radiofrequency signals to create sectioned images of various body structures.	Neurologic, pelvic, abdominal, thoracic cancers
Computed tomography (CT scan)	Use of narrow beam x-ray to scan successive layers of tissue for a cross-sectional view.	Neurologic, pelvic, skeletal, abdominal, thoracic cancers
Fluoroscopy	Use of x-rays that identify contrasts in body tissue densities; may involve the use of contrast agents.	Skeletal, lung, gastrointestinal cancers

Ultrasonography (ultrasound)	High-frequency sound waves echoing off body tissues are converted electronically into images; used to assess tissues deep within the body.	Abdominal and pelvic cancers
Endoscopy	Direct visualization of a body cavity or passageway by insertion of an endoscope into a body cavity or opening; allows tissue biopsy, fluid aspiration and excision of small tumors; both diagnostic and therapeutic.	Bronchial, gastrointestinal cancers
Nuclear medicine imaging	Uses intravenous injection or ingestion of radioisotope substances followed by imaging of tissues that have concentrated the radioisotopes.	Bone, liver, kidney, spleen, brain, thyroid cancers

Positron emission tomography (PET scan)	Computed cross-sectional images of increased concentration of radioisotopes in malignant cells provide information about biologic activity of malignant cells; help distinguish between benign and malignant processes and responses to treatment.	Lung, colon, liver, pancreatic, breast, esophagus cancers; Hodgkin's and non-Hodgkin's lymphoma and melanoma
Radioimmunoconjugates	Monoclonal antibodies are labeled with a radioisotope and injected intravenously into the patient; the antibodies that aggregate at the tumor site are visualized with scanners.	Colorectal, breast, ovarian, head and neck cancers; lymphoma and melanoma

4.2 Management of Cancer

Surgery therapy, radiation therapy, chemotherapy, and biologic response altering therapy (BRM), may be used during treatment.

a. Surgery

1. Diagnostic surgery

Diagnostic surgery is the definitive method of identifying the cellular characteristics that influence

all treatment decisions. Diagnostic surgery, such as a biopsy, is usually performed to obtain a tissue sample for analysis of cells suspected to be malignant. In most instances, the biopsy is taken from the actual tumor. The three most common biopsy methods are the: Excisional biopsy, Incisional biopsy, and Needle biopsies.

2. **Prophylactic Surgery**
   The removal of nonvital tissues or organs that will develop into cancer is prophylactic surgery.
3. **Palliative Surgery**
   To make the patient as comfortable as possible and to promote a fulfilling and productive life for as long as possible and to promote a high quality of life—a quality determined by the patient and family are the goals of treatment.
4. **Reconstructive Surgery**
   In an effort to improve function or obtain a more desirable cosmetic effect, curative surgery is performed.

b. Radiation Therapy

In radiation therapy, ionizing radiation is used to interrupt cellular growth. More than half of patients with cancer receive a form of radiation therapy at some point during treatment. Radiation may be used to cure the cancer, as in Hodgkin's disease, testicular seminomas, thyroid carcinomas, localized cancers of the head and neck, and cancers of the uterine cervix. Radiation therapy may also be used to control malignant disease when a tumor cannot be removed surgically or when local nodal metastasis is present, or it can be used prophylactically to prevent leukemic infiltration to the brain or spinal cord. Palliative radiation therapy is used to relieve the symptoms of metastatic disease, especially when the cancer has spread to brain, bone, or soft tissue, or to treat oncologic emergencies, such as superior vena cava syndrome or spinal cord compression.

Two types of ionizing radiation—**electromagnetic rays** (x-rays and gamma rays) and **particles** (electrons [beta particles], protons, neutrons, and alpha particles)—can lead to tissue disruption. The most harmful tissue disruption is the alteration of the DNA molecule within the cells of the tissue. Ionizing radiation breaks the strands of the DNA helix, leading to cell death. Ionizing radiation can also ionize constituents of body fluids, especially water, leading to the formation of free radicals and irreversibly damaging DNA. If the DNA is incapable of repair, the cell may die immediately, or it may initiate cellular suicide (apoptosis), a genetically programmed cell death.

Cells are most vulnerable to the disruptive effects of radiation during DNA synthesis and mitosis (early S, G2, and M phases of the cell cycle). Therefore, those body tissues that undergo frequent cell division are most sensitive to radiation therapy. These tissues include bone marrow, lymphatic tissue, and epithelium of the gastrointestinal tract, hair cells, and gonads. Slower-growing tissues or tissues at rest are relatively radio-resistant (less sensitive to the effects of radiation). Such tissues include muscle, cartilage, and connective tissues.

A radiosensitive tumor is one that can be destroyed by a dose of radiation that still allows for cell regeneration in the normal tissue. Tumors that are well oxygenated also appear to be more sensitive to radiation. In theory, therefore, radiation therapy may be enhanced if more oxygen can be delivered to tumors. In addition, if the radiation is delivered when most tumor cells are cycling through the cell cycle, the number of cancer cells destroyed (cell killing) is maximal.

Certain chemicals, including chemotherapy agents, act as radio sensitizers and sensitize more hypoxic (oxygen-poor) tumors to the effects of radiation therapy. Radiation is delivered to tumor sites by external or internal means.

1. **External Radiation**

   External radiation, If external radiation therapy is used, one of several delivery methods may be chosen, depending on the depth of the tumor. Depending on the amount of energy they contain, x-rays can be used to destroy cancerous cells at the skin surface or deeper in the body. The higher the energy, the deeper the penetration into the body. Kilovoltage therapy devices deliver the maximal radiation dose to superficial lesions, such as lesions of the skin and breast, whereas linear accelerators and betatron machines produce higher-energy x-rays and deliver their dosage to deeper structures with less harm to the skin and less scattering of radiation within the body tissues. Gamma rays are another form of energy used in radiation therapy. This energy is produced from the spontaneous decay of naturally occurring radioactive elements such as cobalt 60. The gamma rays also deliver this radiation dose beneath the skin surface, sparing skin tissue from adverse effects.

2. **Internal Radiation**

   Internal radiation implantation, or brachytherapy, delivers a high dose of radiation to a localized area. The specific radioisotope for implantation is selected on the basis of its half-life, which is the time it takes for half of its radioactivity to decay. This internal radiation can be implanted by means of needles, seeds, beads, or catheters into body cavities (vagina, abdomen, pleura) or interstitial compartments (breast). Brachytherapy may also be administered orally as with the isotope I131, used to treat thyroid carcinomas.

Intracavitary radioisotopes are frequently used to treat gynecologic cancers. In these malignancies, the radioisotopes are inserted into specially positioned applicators after the position is verified by x-ray. These radioisotopes remain in place for a prescribed period and then are removed. Patients are maintained on bed rest and log-rolled to prevent displacement of the intracavitary delivery device. An indwelling urinary catheter is inserted to ensure that the bladder remains empty. Low-residue diets and antidiarrheal agents, such as diphenoxylate (Lomotil), are provided to prevent bowel movement during therapy, to prevent the radioisotopes from being displaced.

Interstitial implants, used in treating such malignancies as prostate, pancreatic, or breast cancer, may be temporary or permanent, depending on the radioisotopes used. These implants usually consist of seeds, needles, wires, or small catheters positioned to provide a local radiation source and are less frequently dislodged. With internal radiation therapy, the farther the tissue is from the radiation source, the lower the dosage. This spares the noncancerous tissue from the radiation dose.

Because patients receiving internal radiation emit radiation while the implant is in place, contacts with the health care team are guided by principles of time, distance, and shielding to minimize exposure of personnel to radiation. Safety precautions used in caring for the patient receiving brachytherapy include assigning the person to a private room, posting appropriate notices about radiation safety precautions, having staff members wear dosimeter badges, making sure that pregnant staff members are not assigned to this patient's care, prohibiting visits by children or pregnant visitors, limiting visits from others to 30

minutes daily, and seeing that visitors maintain a 6-foot distance from the radiation source.

3. Chemotherapy

In chemotherapy, antineoplastic agents are used in an attempt to destroy tumor cells by interfering with cellular functions and reproduction. Chemotherapy is used primarily to treat systemic disease rather than lesions that are localized and amenable to surgery or radiation. Chemotherapy may be combined with surgery or radiation therapy, or both, to reduce tumor size preoperatively, to destroy any remaining tumor cells postoperatively, or to treat some forms of leukemia. The goals of chemotherapy (cure, control, palliation) must be realistic because they will define the medications to be used and the aggressiveness of the treatment plan.

Chemotherapeutic agents may be administered in the hospital, clinic, or home setting by topical, oral, intravenous, intramuscular, subcutaneous, arterial, intracavitary, and intrathecal routes. The administration route usually depends on the type of agent, the required dose, and the type, location, and extent of tumor being treated. Guidelines for the administration of chemotherapy have been developed by the Oncology Nursing Society.

4. Bone Marrow Transplantation

Although surgery, radiation therapy, and chemotherapy have resulted in improved survival rates for cancer patients, many cancers that initially respond to therapy recur. This is true of hematologic cancers that affect the bone marrow and solid tumor cancers treated with lower doses of antineoplastics to spare the bone marrow from larger, ablative doses of chemotherapy or radiation therapy. The role of bone marrow transplantation (BMT) for malignant as well as some

non-malignant diseases continues to grow. Types of BMT based on the source of donor cells include:

5. **Allogeneic donors from other parties.**

6. **Autologous**

   Autologous (from patient) - Autologous BMT is considered for patients with disease of the bone marrow who do not have a suitable donor for allogeneic BMT and for patients who have healthy bone marrow but require bone marrow–ablative doses of chemotherapy to cure an aggressive malignancy.

7. **Syngeneic**

   Syngeneic (of identical twins) - requires identical network matching clients.

8. **Biologic Response Modifiers - altering the immunological relationship between the tumor and the cancer patient (host) to provide therapeutic benefits as a method of treatment.**

4.3 Nursing Process: The Patient with Cancer

The outlook for patients with cancer has greatly improved because of scientific and technological advances. As a result of the underlying disease or various treatment modalities, however, the patient with cancer may experience a variety of secondary problems, such as infection, reduced WBC counts, bleeding, skin problems, nutritional problems, pain, fatigue, and psychological stress.

A. Assessment - Regardless of the type of cancer treatment or prognosis, many patients with cancer are susceptible to the following problems and complications. An important role of the nurse on the oncology team is to assess the patient for these problems and complications.

1. **Infection** In all stages of cancer, the nurse assesses factors that can promote infection. Infection is the leading cause of death in cancer patients. The nurse monitors laboratory studies to detect early changes in WBC counts. Common sites of infection, such as the pharynx, skin, perianal area, urinary tract, and respiratory tract, are assessed frequently. The typical signs of infection (swelling, redness, drainage, and pain), however, may not occur in the immunosuppressed patient due to a diminished local inflammatory response. Fever may be the only sign of infection that the patient exhibits. The nurse also monitors the patient for sepsis, particularly if invasive catheters or infusion lines are in place.

2. **Bleeding** The nurse assesses cancer patients for factors that may contribute to bleeding. These include bone marrow suppression from radiation, chemotherapy, and other medications that interfere with coagulation and platelet functioning, such as aspirin, dipyridamole (Persantine), heparin, or warfarin (Coumadin). Common bleeding sites include skin and mucous membranes; the intestinal, urinary, and respiratory tracts; and the brain. Gross hemorrhage, as well as blood in the stools, urine, sputum, or vomitus (melena, hematuria, hemoptysis, and hematemesis), oozing at injection sites, bruising (ecchymosis), petechiae, and changes in mental status, are monitored and reported.

3. **Skin Problems** The integrity of skin and tissue is at risk in cancer patients because of the effects of chemotherapy, radiation therapy, surgery, and invasive procedures carried out for diagnosis and therapy. As part of the assessment, the nurse identifies which of these predisposing factors are present and assesses the patient for other risk factors, including nutritional

deficits, bowel and bladder incontinence, immobility, immunosuppression, multiple skin folds, and changes related to aging. Skin lesions or ulcerations secondary to the tumor are noted. Alterations in tissue integrity throughout the gastrointestinal tract are particularly bothersome to the patient. Any lesions of the oral mucous membranes are noted, as are their effects on the patient's nutritional status and comfort level.

4. **Hair loss** Alopecia (hair loss) is another form of tissue disruption common to cancer patients who receive radiation therapy or chemotherapy. In addition to noting hair loss, the nurse also assesses the psychological impact of this side effect on the patient and the family.

5. **Nutritional Concerns** Assessing the patient's nutritional status is an important nursing role. Impaired nutritional status may contribute to disease progression, immune incompetence, increased incidence of infection, delayed tissue repair, diminished functional ability, and decreased capacity to continue antineoplastic therapy. Altered nutritional status, weight loss, and cachexia (muscle wasting, emaciation) may be secondary to decreased protein and caloric intake, metabolic or mechanical effects of the cancer, systemic disease, side effects of the treatment, or the emotional status of the patient.

   The patient's weight and caloric intake are monitored on a consistent basis. Other information obtained through assessment includes diet history, any episodes of anorexia, changes in appetite, situations and foods that aggravate or relieve anorexia, and medication history. Difficulty in chewing or swallowing is determined and the occurrence of nausea, vomiting, or diarrhea is noted.

Clinical and laboratory data useful in assessing the patient's nutritional status include anthropometric measurements (triceps skin fold and middle-upper arm circumference), serum protein levels (albumin and transferrin), serum electrolytes, lymphocyte count, skin response to intradermal injection of antigens, hemoglobin levels, hematocrit, urinary creatinine levels, and serum iron levels.

6. **Pain** Pain and discomfort in cancer may be related to the underlying disease, pressure exerted by the tumor, diagnostic procedures, or the cancer treatment itself. As in any other situation involving pain, cancer pain is affected by both physical and psychosocial influences. In addition to assessing the source and site of pain, the nurse also assesses those factors that increase the patient's perception of pain, such as fear and apprehension, fatigue, anger, and social isolation.

7. **Fatique** Acute fatigue, which occurs after an energy-demanding experience, serves a protective function; chronic fatigue, however, does not. It is often overwhelming, excessive, and not responsive to rest, and it seriously affects quality of life. Fatigue is the most commonly reported side effect in patients who receive chemotherapy and radiation therapy. The nurse assesses for feelings of weariness, weakness, lack of energy, inability to carry out necessary and valued daily functions, lack of motivation, and inability to concentrate. Patients may become less verbal and appear pallid, with relaxed facial musculature. The nurse assesses physiologic and psychological stressors that can contribute to fatigue, including pain, nausea, dyspnea, constipation, fear, and anxiety.

8. **Psychosocial Status** Nursing assessment also focuses on the patient's psychological and mental status as the patient and the family face this life threatening experience, unpleasant diagnostic tests and treatment modalities, and progression of disease. The patient's mood and emotional reaction to the results of diagnostic testing and prognosis are assessed, along with evidence that the patient is progressing through the stages of grief and can talk about the diagnosis and prognosis with the family.

9. **Body Image** Cancer patients are forced to cope with many assaults to body image throughout the course of disease and treatment. Entry into the health care system is often accompanied by depersonalization. Threats to self-concept are enormous as patients face the realization of illness, possible disability, and death. To accommodate treatments or because of the disease, many cancer patients are forced to alter their lifestyles. Priorities and values change when body image is threatened. Disfiguring surgery, hair loss, cachexia, skin changes, altered communication patterns, and sexual dysfunction are some of the devastating results of cancer and its treatment that threaten the patient's self-esteem and body image. The nurse identifies these potential threats and assesses the patient's ability to cope with these changes.

B. Diagnosis

Based on the assessment data, nursing diagnoses of the patient with cancer may include the following:

1. Impaired oral mucous membrane
2. Impaired tissue integrity
3. Impaired tissue integrity: alopecia

4. Impaired tissue integrity: malignant skin lesions
5. Imbalanced nutrition, less than body requirements
6. Anorexia
7. Malabsorption
8. Cachexia
9. Chronic pain
10. Fatigue
11. Disturbed body image
12. Anticipatory grieving

C. Planning and Goals

The major goals for the patient may include management of stomatitis, maintenance of tissue integrity, maintenance of nutrition, relief of pain, relief of fatigue, improved body image, effective progression through the grieving process, and absence of complications.

D. Nursing Interventions

The patient with cancer is at risk for various adverse effects of therapy and complications. The nurse in all health care settings, including the home, assists the patient and family in managing these problems.

1. Managing Stomatitis

Stomatitis, an inflammatory response of the oral tissues, commonly develops within 5 to 14 days after the patient receives certain chemotherapeutic agents, such as doxorubicin and 5-fluorouracil, and BRMs, such as IL-2 and IFN. As many as 40% of patients receiving chemotherapy experience some degree of stomatitis during treatment. Patients receiving dose-intensive chemotherapy (considerably higher doses than conventional dosing), such as those undergoing BMT, are at increased risk for stomatitis. Stomatitis

may also occur with radiation to the head and neck. Stomatitis is characterized by mild redness (erythema) and edema or, if severe, by painful ulcerations, bleeding, and secondary infection. In severe cases of stomatitis, cancer therapy may be temporarily halted until the inflammation decreases.

Although multiple studies on stomatitis have been published, the optimal prevention and treatment approaches have not been identified. However, most clinicians agree that good oral hygiene that includes brushing, flossing, and rinsing is necessary to minimize the risk for oral complications associated with cancer therapies. Soft-bristled toothbrushes and nonabrasive toothpaste prevent or reduce trauma to the oral mucosa. Oral swabs with sponge-like applicators may be used in place of a toothbrush for painful oral tissues. Flossing may be performed unless it causes pain or unless platelet levels are below 40,000/mm3 (0.04 × 1012/L). Oral rinses with saline solution or tap water may be necessary for patients who cannot tolerate a toothbrush. Products that irritate oral tissues or impair healing, such as alcohol-based mouth rinses, are avoided. Foods that are difficult to chew or are hot or spicy are avoided to minimize further trauma. The patient's lips are lubricated to keep them from becoming dry and cracked. Topical anti-inflammatory and anesthetic agents may be prescribed to promote healing and minimize discomfort. Products that coat or protect oral mucosa are used to promote comfort and prevent further trauma. The patient who experiences severe pain and discomfort with stomatitis requires systemic analgesics.

Adequate fluid and food intake is encouraged. In some instances, parenteral hydration and nutrition are needed. Topical or systemic antifungal and antibiotic

medications are prescribed to treat local or systemic infections.

2. Maintaining Tissue Integrity

Some of the most frequently encountered disturbances of tissue integrity, in addition to stomatitis, include skin and tissue reactions to radiation therapy, alopecia, and metastatic skin lesions.

The patient who is experiencing skin and tissue reactions to radiation therapy requires careful skin care to prevent further skin irritation, drying, and damage. The skin over the affected area is handled gently; rubbing and use of hot or cold water, soaps, powders, lotions, and cosmetics are avoided. The patient may avoid tissue injury by wearing loose-fitting clothes and avoiding clothes that constrict, irritate, or rub the affected area. If blistering occurs, care is taken not to disrupt the blisters, thus reducing the risk of introducing bacteria. Moisture- and vapor-permeable dressings, such as hydrocolloids and hydrogels, are helpful in promoting healing and reducing pain. Aseptic wound care is indicated to minimize the risk for infection and sepsis. Topical antibiotics, such as 1% silver sulfadiazine cream (Silvadene), may be prescribed for use on areas of moist desquamation (painful, red, moist skin).

3. Assisting Patients to Cope with Alopecia

The temporary or permanent thinning or complete loss of hair is a potential adverse effect of various radiation therapies and chemotherapeutic agents. The extent of alopecia depends on the dose and duration of therapy. These treatments cause alopecia by damaging stem cells and hair follicles. As a result, the hair is brittle and may fall out or break off at the surface of the scalp. Loss of other body hair is less frequent. Hair loss

usually begins within 2 to 3 weeks after the initiation of treatment; regrowth begins within 8 weeks after the last treatment. Some patients who undergo radiation to the head may sustain permanent hair loss. Many health care providers view hair loss as a minor problem when compared with the potentially life-threatening consequences of cancer. For many patients, however, hair loss is a major assault on body image, resulting in depression, anxiety, anger, rejection, and isolation. To patients and families, hair loss can serve as a constant reminder of the challenges cancer places on their coping abilities, interpersonal relationships, and sexuality.

The nurse's role is to provide information about alopecia and to support the patient and family in coping with disturbing effects of therapy, such as hair loss and changes in body image. Patients are encouraged to acquire a wig or hairpiece before hair loss occurs so that the replacement matches their own hair. Use of attractive scarves and hats may make the patient feel less conspicuous. Nurses can refer patients to supportive programs, such as "Look Good, Feel Better," offered by the American Cancer Society. Knowledge that hair usually begins to regrow after completing therapy may comfort some patients, although the color and texture of the new hair may be different.

4. Managing Malignant Skin Lesions

Skin lesions may occur with local extension of the tumor or embolization of the tumor into the epithelium and its surrounding lymph and blood vessels. Secondary growth of cancer cells into the skin may result in redness (erythematous areas) or can progress to wounds involving tissue necrosis and infection. The most extensive lesions tend to disintegrate and are purulent and malodorous. In addition, these lesions are a source of considerable pain and discomfort.

Although this type of lesion is most often associated with breast cancer and head and neck cancers, it can also occur with lymphoma, leukemia, melanoma, and cancers of the lung, uterus, kidney, colon, and bladder. The development of severe skin lesions is usually associated with a poor prognosis for extended survival.

Ulcerating skin lesions usually indicate widely disseminated disease unlikely to be eradicated. Managing these lesions becomes a nursing priority. Nursing care includes carefully assessing and cleansing the skin, reducing superficial bacteria, controlling bleeding, reducing odor, and protecting the skin from pain and further trauma. The patient and family require assistance and guidance to care for these skin lesions at home. Referral for home care is indicated.

5. Promoting Nutrition

Most cancer patients experience some weight loss during their illness. Anorexia, malabsorption, and cachexia are examples of nutritional problems that commonly occur in cancer patients; special attention is needed to prevent weight loss and promote nutrition.

6. Anorexia

Among the many causes of anorexia in the cancer patient are alterations in taste, manifested by increased salty, sour, and metallic taste sensations, and altered responses to sweet and bitter flavors, leading to decreased appetite, decreased nutritional intake, and protein-calorie malnutrition. Taste alterations may result from mineral (e.g., zinc) deficiencies, increases in circulating amino acids and cellular metabolites, or the administration of chemotherapeutic agents. Patients undergoing radiation therapy to the head and neck may experience “mouth blindness,” which is a severe impairment of taste.

Alterations in the sense of smell also alter taste; this is a common experience of patients with head and neck cancers. Anorexia may occur because the person feels full after eating only a small amount of food. This sense of fullness occurs secondary to a decrease in digestive enzymes, abnormalities in the metabolism of glucose and triglycerides, and prolonged stimulation of gastric volume receptors, which convey the feeling of being full. Psychological distress, such as fear, pain, depression, and isolation, throughout illness may also have a negative impact on appetite. The person may develop an aversion to food because of nausea and vomiting after treatment.

7. Malabsorption

Many cancer patients are unable to absorb nutrients from the gastrointestinal system as a result of tumor activity and cancer treatment. Tumors can affect the gastrointestinal activity in several ways. They may impair enzyme production or produce fistulas. They secrete hormones and enzymes, such as gastrin; this leads to increased gastrointestinal irritation, peptic ulcer disease, and decreased fat digestion. They also interfere with protein digestion.

Chemotherapy and radiation can irritate and damage mucosal cells of the bowel, inhibiting absorption. Radiation therapy can cause sclerosis of the blood vessels in the bowel and fibrotic changes in the gastrointestinal tissue. Surgical intervention may change peristaltic patterns, alter gastrointestinal secretions, and reduce the absorptive surfaces of the gastrointestinal mucosa, all leading to malabsorption.

8. Cachexia

Cachexia is common in patients with cancer, especially in advanced disease. Cancer cachexia is related to inadequate nutritional intake along with increasing metabolic demand, increased energy expenditure due to anaerobic metabolism of the tumor, impaired glucose metabolism, competition of the tumor cells for nutrients, altered lipid metabolism, and a suppressed appetite. It is characterized by loss of body weight, adipose tissue, visceral protein, and skeletal muscle. Patients who are cachectic complain of loss of appetite, early satiety, and fatigue. As a result of protein losses they are often anemic and have peripheral edema.

9. General Nutritional Considerations

Whenever possible, every effort is used to maintain adequate nutrition through the oral route. Food should be prepared in ways that make it appealing. Unpleasant smells and unappetizing looking foods are avoided. Family members are included in the plan of care to encourage adequate food intake. The patient's preferences, as well as physiologic and metabolic requirements, are considered when selecting foods. Small, frequent meals are provided, with supplements between meals. Patients often tolerate larger amounts of food earlier in the day rather than later, so meals can be planned accordingly. Patients should avoid drinking fluids while eating, to avoid early satiety. Oral hygiene before mealtime often makes meals more pleasant. Pain, nausea, and other symptoms that may interfere with nutrition are assessed and managed. Medications such as corticosteroids or progestational agents such as megestrol acetate have been used successfully as appetite stimulants.

If adequate nutrition cannot be maintained by oral intake, nutritional support via the enteral route may be necessary. Shortterm nutritional supplementation may

be provided through a nasogastric tube. However, if nutritional support is needed beyond several weeks, a gastrostomy or jejunostomy tube may be inserted. Patients and families are taught to administer enteral nutrition in the home setting.

If malabsorption is a problem, enzyme and vitamin replacement may be instituted. Additional strategies include changing the feeding schedule, using simple diets, and relieving diarrhea. If malabsorption is severe, parenteral nutrition (PN) may be necessary. PN can be administered in several ways: by a long-term venous access device, such as a right atrial catheter, an implanted venous port, or a peripherally inserted central catheter (Fig. 16-6). The nurse teaches the patient and family to care for venous access devices and to administer PN. Home care nurses may assist with or supervise PN in the home.

Interventions to reduce cachexia usually do not prolong survival but may improve the patient's quality of life. Before invasive nutritional strategies are instituted, the nurse should assess the patient carefully and discuss the options with the patient and family. Creative dietary therapies, enteral (tube) feedings, or PN may be necessary to ensure adequate nutrition. Nursing care is also directed toward preventing trauma, infection, and other complications that increase metabolic demands.

10. Relieving Pain

Of all patients with progressive cancer, more than 75% experience pain (Yarbro, Hansen-Frogge & Goodman, 1999). Although patients with cancer may have acute pain, their pain is more frequently characterized as chronic. (For more information on cancer-related pain, see Chap. 13.) As in other situations involving pain, the experience of cancer pain is influenced by both

physical and psychosocial factors.

Cancer can cause pain in various ways (Table 16-9). Pain is also associated with various cancer treatments. Acute pain is linked with trauma from surgery. Occasionally, chronic pain syndromes, such as postsurgical neuropathies (pain related to nerve tissue injury), occur. Some chemotherapeutic agents cause tissue necrosis, peripheral neuropathies, and stomatitis—all potential sources of pain—whereas radiation therapy can cause pain secondary to skin or organ inflammation. Cancer patients may have other sources of pain, such as arthritis or migraine headaches, that are unrelated to the underlying cancer or its treatment.

For many patients, pain is a signal that the tumor is growing and that death is approaching. As the patient anticipates the pain and anxiety increases, pain perception heightens, producing fear and further pain. Chronic cancer pain, then, can be best described as a cycle progressing from pain to anxiety to fear and back to pain again.

Pain tolerance, the point past which pain can no longer be tolerated, varies among people. Pain tolerance is decreased by fatigue, anxiety, fear of death, anger, powerlessness, social isolation, changes in role identity, loss of independence, and past experiences. Adequate rest and sleep, diversion, mood elevation, empathy, and medications such as antidepressants, antianxiety agents, and analgesics enhance tolerance to pain.

Inadequate pain management is most often the result of misconceptions and insufficient knowledge about pain assessment and pharmacologic interventions on the part of patients, families, and health care providers.

Successful management of cancer pain is based on thorough and objective pain assessment that examines physical, psychosocial, environmental, and spiritual factors. A multidisciplinary team approach is essential to determine optimal management of the patient's pain. Unlike instances of chronic nonmalignant pain, systemic analgesics play a central role in managing cancer pain.

The World Health Organization (Dalton & Youngblood, 2000) advocates a three-step approach to treating cancer pain (see Chap. 13). Analgesics are administered based on the patient's level of pain. Nonopioid analgesics (eg, acetaminophen) are used for mild pain; weak opioid analgesics (eg, codeine) are used for moderate pain; and strong opioid analgesics (eg, morphine) are used for severe pain. If the patient's pain escalates, the strength of the analgesic medication is increased until the pain is controlled. Adjuvant medications are also administered to enhance the effectiveness of analgesics and to manage other symptoms that may contribute to the pain experience. Examples of adjuvant medications include antiemetics, antidepressants, anxiolytics, antiseizure agents, stimulants, local anesthetics, radiopharmaceuticals (radioactive agents that may be used to treat painful bone tumors), and corticosteroids.

Preventing and reducing pain help to decrease anxiety and break the pain cycle. This can be accomplished best by administering analgesics on a regularly scheduled basis as prescribed (the preventive approach to pain management), with additional analgesics administered for breakthrough pain as needed and as prescribed. Various pharmacologic and nonpharmacologic approaches offer the best methods of managing cancer pain. No reasonable approaches, even those that may be invasive, should be over looked because

of a poor or terminal prognosis. Nurses help patients and families to take an active role in managing pain. Nurses provide education and support to correct fears and misconceptions about opioid use. Inadequate pain control leads to suffering, anxiety, fear, immobility, isolation, and depression. Improving a patient's quality of life is as important as preventing a painful death.

11. Decreasing Fatigue

In recent years, fatigue has been recognized as one of the most significant and frequent symptoms experienced by patients receiving cancer therapy. Nurses help the patient and family to understand that fatigue is usually an expected and temporary side effect of the cancer process and of many treatments used. Fatigue also stems from the stress of coping with cancer. It does not always signify that the cancer is advancing or that the treatment is failing.

Nursing strategies are implemented to minimize fatigue or assist the patient to cope with existing fatigue. Helping the patient to identify sources of fatigue aids in selecting appropriate and individualized interventions. Ways to conserve energy are developed to help the patient plan daily activities. Alternating periods of rest and activity are beneficial. Regular, light exercise may decrease fatigue and facilitate coping, whereas lack of physical activity and "too much rest" can actually contribute to deconditioning and associated fatigue.

Patients are encouraged to maintain as normal a lifestyle as possible by continuing with those activities they value and enjoy. Prioritizing necessary and valued activities can assist patients in planning for each day. Both patients and families are encouraged to plan to reallocate responsibilities, such as attending to child care, cleaning, and preparing meals. Patients who are

employed full-time may need to reduce the number of hours worked each week. The nurse assists the patient and family in coping with these changing roles and responsibilities.

Nurses also address factors that contribute to fatigue and implement pharmacologic and nonpharmacologic strategies to manage pain. Nutrition counseling is provided to patients who are not eating enough calories or protein. Small, frequent meals require less energy for digestion. Serum hemoglobin and hematocrit levels are monitored for deficiencies, and blood products or EPO are administered as prescribed. Patients are monitored for alterations in oxygenation and electrolyte balances. Physical therapy and assistive devices are beneficial for patients with impaired mobility.

12. Improving Body Image and Self-Esteem

A positive approach is essential when caring for the patient with an altered body image. To help the patient retain control and positive self-esteem, it is important to encourage independence and continued participation in self-care and decision making. The patient should be assisted to assume those tasks and participate in those activities that are personally of most value. Any negative feelings that the patient has or threats to body image should be identified and discussed. The nurse serves as a listener and counselor to both the patient and the family. Referral to a support group can provide the patient with additional assistance in coping with the changes resulting from cancer or its treatment. In many cases, a cosmetologist can provide ideas about hair or wig styling, make-up, and the use of scarves and turbans to help with body image concerns.

Patients who experience alterations in sexuality and sexual function are encouraged to discuss concerns

openly with their partner. Alternative forms of sexual expression are explored with the patient and partner to promote positive self-worth and acceptance. The nurse who identifies serious physiologic, psychological, or communication difficulties related to sexuality or sexual function is in a key position to assist the patient and partner to seek further counseling if necessary.

13. Assisting in the Grieving Process

A cancer diagnosis need not indicate a fatal outcome. Many forms of cancer are curable; others may be cured if treated early. Despite these facts, many patients and their families view cancer as a fatal disease that is inevitably accompanied by pain, suffering, debility, and emaciation. Grieving is a normal response to these fears and to the losses anticipated or experienced by the patient with cancer. These may include loss of health, normal sensations, body image, social interaction, sexuality, and intimacy. The patient, family, and friends may grieve for the loss of quality time to spend with others, the loss of future and unfulfilled plans, and the loss of control over one's own body and emotional reactions.

The patient and family just informed of the cancer diagnosis frequently respond with shock, numbness, and disbelief. It is often during this stage that the patient and family are called on to make important initial decisions about treatment. They require the support of the physician, nurse, and other health care team members to make these decisions. An important role of the nurse is to answer any questions the patient and family have and clarify information provided by the physician.

In addition to assessing the response of the patient and family to the diagnosis and planned treatment, the nurse

assists them in framing their questions and concerns, identifying resources and support people (e.g., spiritual advisor, counselor), and communicating their concerns with each other. Support groups for patients and families are available through hospitals and various community organizations. These groups provide direct assistance, advice, and emotional support.

As the patient and family progress through the grieving process, they may express anger, frustration, and depression. During this time, the nurse encourages the patient and family to verbalize their feelings in an atmosphere of trust and support. The nurse continues to assess their reactions and provides assistance and support as they confront and learn to deal with new problems. If the patient enters the terminal phase of disease, the nurse may realize that the patient and family members are at different stages of grief. In such cases, the nurse assists the patient and family to ac knowledge and cope with their reactions and feelings. Nurses also assist patients and families to explore preferences for issues related to end-of-life care such as withdrawal of active disease treatment, desire for the use of life support measures, and symptom management. Support, which can be as simple as holding the patient's hand or just being with the patient at home or at the bedside, often contributes to peace of mind. Maintaining contact with the surviving family members after the death of the cancer patient may help them to work through their feelings of loss and grief.

14. Monitoring and Managing Potential Complications

Despite advances in cancer care, infection remains the leading cause of death. In the cancer patient, defense against infection is compromised in many different ways. The integrity of the skin and mucous membrane, the body's first line of defense, is challenged by multiple

invasive diagnostic and therapeutic procedures, by adverse effects of radiation and chemotherapy, and by the detrimental effects of immobility.

Impaired nutrition resulting from anorexia, nausea, vomiting, diarrhea, and the underlying disease alters the body's ability to combat invading organisms. Medications such as antibiotics disturb the balance of normal flora, allowing the overgrowth of pathogenic organisms.

Cancer itself may be immunosuppressive. Cancers such as leukemia and lymphoma are often associated with defects in cellular and humoral immunity. Advanced cancer can lead to obstruction by the tumor of the hollow viscera (such as the intestines), blood vessels, and lymphatic vessels, creating a favorable environment for proliferation of pathogenic organisms. In some patients, tumor cells infiltrate bone marrow and prevent normal production of WBCs. Most often, however, a decrease in WBCs is a result of bone marrow suppression after chemotherapy or radiation therapy.

a. Infection

Strict asepsis is essential when handling intravenous lines, catheters, and other invasive equipment. Exposure of the patient to others with an active infection and to crowds is avoided. Patients with profound immunosuppression, such as BMT recipients, may need to be placed in a protective environment where the room and its contents are sterilized and the air is filtered. These patients may also receive low-bacteria diets, avoiding fresh fruits and vegetables. Hand hygiene and appropriate general hygiene are necessary to reduce exposure to potentially harmful bacteria and to eliminate environmental contaminants.

Invasive procedures, such as injections, vaginal or rectal examinations, rectal temperatures, and surgery, are avoided. The patient is encouraged to cough and perform deep-breathing exercises frequently to prevent atelectasis and other respiratory problems. Prophylactic antimicrobial therapy may be used for patients who are expected to be profoundly immunosuppressed and at risk for certain infections. The nurse teaches the patient and family to recognize signs and symptoms of infection to report, perform effective hand hygiene, use antipyretics, maintain skin integrity, and administer hematopoietic growth factors when indicated.

b. Septic Shock

The nurse assesses the patient frequently for infection and inflammation throughout the course of the disease. Septicemia and septic shock are life-threatening complications that must be prevented or detected and treated promptly. Patients with signs and symptoms of impending sepsis and septic shock require immediate hospitalization and aggressive treatment.

Signs and symptoms of septic shock include altered mental status, either subnormal or elevated temperature, cool and clammy skin, decreased urine output, hypotension, dysrhythmias, electrolyte imbalances, and abnormal arterial blood gas values. The patient and family members are instructed about signs of septicemia, methods for preventing infection, and actions to take if infection or septicemia occurs.

The nurse monitors the blood pressure, pulse rate, respirations, and temperature of the patient with shock every 15 to 30 minutes. Neurologic assessments are carried out to detect changes in

orientation and responsiveness. Fluid and electrolyte status is monitored by measuring fluid intake and output and serum electrolytes. Arterial blood gas values and pulse oximetry are monitored to determine tissue oxygenation. The nurse administers intravenous fluids, blood products, and vasopressors as prescribed to maintain the patient's blood pressure and tissue perfusion. Supplemental oxygen is often necessary. Broad-spectrum antibiotics are administered as prescribed to combat the underlying infection.

c. Bleeding and Hemorrhage

Thrombocytopenia, a decrease in the circulating platelet count, is the most common cause of bleeding in cancer patients and is usually defined as a count of less than 100,000/mm3 (0.1 × 1012/L). When the count falls between 20,000 and 50,000/mm3 (0.02 to 0.05 × 1012/L), the risk for bleeding increases. Counts under 20,000/mm3 (0.02 × 1012/L) are associated with an increased risk for spontaneous bleeding, for which the patient requires a platelet transfusion. Platelets are essential for normal blood clotting and coagulation (hemostasis).

In addition to monitoring laboratory values, the nurse continues to assess the patient for bleeding. The nurse also takes steps to prevent trauma and minimize the risk for bleeding by encouraging the patient to use a soft, not stiff, toothbrush and an electric, not straight-edged, razor. Additionally, the nurse avoids unnecessary invasive procedures (e.g., rectal temperatures, intramuscular injections, and catheterization) and assists the patient and family to identify and remove environmental hazards that may lead to falls or other trauma.

Soft foods, increased fluid intake, and stool softeners, if prescribed, may be indicated to reduce trauma to the gastrointestinal tract. The joints and extremities are handled and moved gently to minimize the risk for spontaneous bleeding.

The nurse may administer IL-11, which has been approved by the FDA (Rust, Wood & Battiato, 1999) to prevent severe thrombocytopenia and to reduce the need for platelet transfusions following myelosuppressive chemotherapy in patients with nonmyeloid malignancies. In some instances, the nurse teaches the patient or family member to administer IL-11 in the home.

Blood pressure and pulse and respiratory rates are monitored every 15 to 30 minutes when hospitalized patients experience bleeding.Serum hemoglobin and hematocrit are monitored carefully for changes indicating blood loss. The nurse tests all urine, stool, and emesis for occult blood. Neurologic assessments are performed to detect changes in orientation and behavior. The nurse administers fluids and blood products as prescribed to replace any losses. Vasopressor agents are administered as prescribed to maintain blood pressure and ensure tissue oxygenation. Supplemental oxygen is used as necessary.

d. Evaluation

1. Maintains integrity of oral mucous membranes
2. Maintains adequate tissue integrity
3. Maintains adequate nutritional status
4. Achieves relief of pain and discomfort

5. Demonstrates increased activity tolerance and decreased fatigue
6. Exhibits improved body image and self-esteem
7. Progresses through the grieving process
8. Experiences no complications, such as infection, or sepsis, and no episodes of bleeding or hemorrhage

QUIZ

WHITE BLOOD CELL DISORDERS

I. LEUKEMIA

Cause: idiopathic Increase in immature WBC	Classification: a. Stem cell line involved • Myeloid or Lymphoid b. Time in which symptoms evolve • Acute or Chronic

Classifications of Leukemia

Criteria	AML	CML	ALL	CLL
Age Group	All, incidence rises w/ age, peak 60 y/o	Incidence rises w/ age; median = 40-50 y/o	**Young children;** Boy > girl; peak 4 y/o, > 15 y/o; uncommon	Older adults, > 60 y/o
CBC RBC WBC Platelet	 Decreased Low (normal WBC) Decreased	 Varies Increased (>100,000) Varies	 Decreased Immature Lymphocytes Decreased	 Varies Increased lymphocytes Varies
Clinical manifest-ations	Insufficient production of normal blood cells	Asymptomatic SOB Splenomegaly Hepatomegaly	Immature Lymphocytes Proliferate CNS involvement Splenomegaly hepatomegaly	Lymphadeno-pathy Splenomegaly Hepatomegaly "B symptoms" Anergy
Survival	< 1 year	3 – 5 years	5 years	14 years (early stage) 2,5 years (late stage)
Common cause of death	Infection and hemorrhage	Infection and hemorrhage	Infection, esp.viral	Infection Hemorrhage (late)

"B SYMPTOMS" – PRESENT IN DISORDERS LYMPHOID IN ORIGIN (CLL, multiple myeloma and malignant lymphoma) **H –** High grade fever **U** – uncontrolled weight loss **N** – Night sweat

ACUTE LYMPHOCYTIC LEUKEMIA
Childhood cancer

DIAGNOSTIC TESTS:	**TREATMENT** **Chemotherapeutic Agents**
1. CBC	**1. Vincristine (Oncovin)** N and V IV Extravasation Constipation Optic Neuritis
2. BMA	**2. Doxorubucin (Adriamycin)** Diarrhea Alopecia N and V
3. BMB	**3. Cyclophosphamide** Hematuria and Cystitis Alopecia N and V

II. MULTIPLE MYELOMA

<table>
<tr>
<td>Proliferation of B cells (plasma cells) and tumors within the bones

Risk Factors: Elderly, African American</td>
<td></td>
</tr>
<tr>
<td>Metastasis: in BONE
↓
Bone loss (Ca leak)
↓ ↓
Brittle Bones
↓
Pathologic

Hypercalcemia
↓
Renal calculi
↓
Renal failure

Assessment: Weakness
B
➢ Bone pain
➢ Bone pathologic fracture</td>
<td>Diagnosis:

1. Serum protein electrophoresis

2. Urine protein electrophoresis

3. CBC

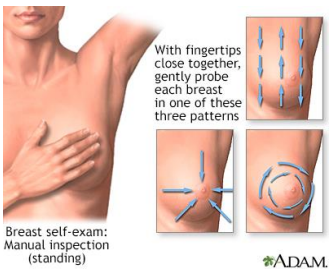
4. Bone marrow biopsy

Management:

1. Increase oral fluids to prevent renal calculi
2. Pain management</td>
</tr>
</table>

SCREENING TESTS

1. Breast self – exam (BSE)



Timing:

- Pre-menopausal
- Menopause
- Palpation: _____
- Inspection: asymmetry, painless, lump, discharge

1. Radical mastectomy → breast tissue + lymph nodes + chest muscles
2. Modified Radical mastectomy → breast tissue + lymph nodes
3. Simple mastectomy → breast tissue
4. Lumpectomy → tumor + surrounding tissue

The best time to do a monthly breast self-exam is about 3 to 5 days after your period starts. Do it at the same time every month Your breasts are not as tender or lumpy at this time in your monthly cycle. If you have gone through menopause, do your exam on the same day every month.

Begin by lying on your back. It is easier to examine all breast tissue if you are lying down.

- Place your right hand behind your head. With the middle fingers of your left hand, gently yet firmly press down using small motions to examine the entire right breast.
- Next, sit or stand. Feel your armpit, because breast tissue goes into that area.
- Gently squeeze the nipple, checking for discharge. Repeat the process on the left breast.
- Use one of the patterns shown in the diagram to

make sure that you are covering all of the breast tissue.

Next, stand in front of a mirror with your arms by your side.

- Look at your breasts directly and in the mirror. Look for changes in skin texture, such as dimpling, puckering, indentations, or skin that looks like an orange peel.
- Also note the shape and outline of each breast.
- Check to see if the nipple turns inward.

Do the same with your arms raised above your head. **https://www.mountsinai.org/health-library/special-topic/breast-self-exam**

2. Mammography

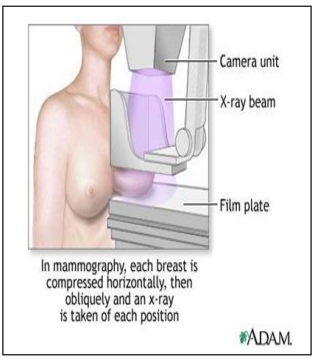
Mammography is a radiographic technique used to detect breast cysts or tumors, especially those not palpable on physical examination.

- Timing: _____
- Pre: Avoiding using – cream, powder, lotion, deodorant
    - Expect: Discomfort (15 – 30 minutes)
- Radiation exposure: _____

Purpose of Mammography

- To screen for malignant breast tumors.
- To investigate breast masses, breast pain, or nipple discharge.
- To differentiate between benign breast disease and malignant tumors.

- To monitor patients with breast cancer who are treated with breast-conserving surgery and radiation.



Mammography Procedure

Patient Preparation

1. Instruct the patient to avoid using underarm deodorant or powder the day of the exam.
2. Explain that the test takes about 15 minutes.
3. Explain to the patient that she may be asked to wait while the films are checked.
4. When scheduling the test, inform the staff if patient has breast implants.
5. Make sure the patient has signed an appropriate consent form.
6. Note and report all allergies.

Implementation

1. The patient rests one breast on a table above the X-ray cassette.
2. The compressor is placed on the breast.
3. The patient holds her breath until the X-ray is taken and she's told to breathe again.
4. An X-ray of the cranicaudal view is taken.
5. The machine is rotated, and the breast is compressed again.
6. An X-ray of the lateral view is taken.

7. The procedure is repeated for the other breast.
8. The film is developed and checked for quality.

Nursing Intervention

1. Answer the patient's questions about the test.
2. Encourage the patient to deep breathe to alleviate fear and anxiety.
3. Make the patient feel comfortable after the procedure.
4. Prepare to educate the patient about her diagnosis.
5. Prepare the patient for further testing or surgery, as indicated.

Interpretation

Normal Results

- The test reveals normal ducts, glandular tissue, and fat architecture.
- No abnormal masses or calcifications are present.

Abnormal Results

- Irregular, poorly outlined, opaque areas suggest malignant tumors, especially if solitary and unilateral.
- Well-outlined, regular, clear spots may be benign, especially if bilateral.

Interfering Factors

- Powders, deodorants, or salves on the breast and axilla that may cause false positive results.
- Failure to remove jewelry and clothing (possible false-positive results or poor imaging).
- Glandural breasts that are common in patients younger than age 30, active lactation and previous breast surgery (possible poor imaging).

- Breast implants (possible hindrance in detecting masses).

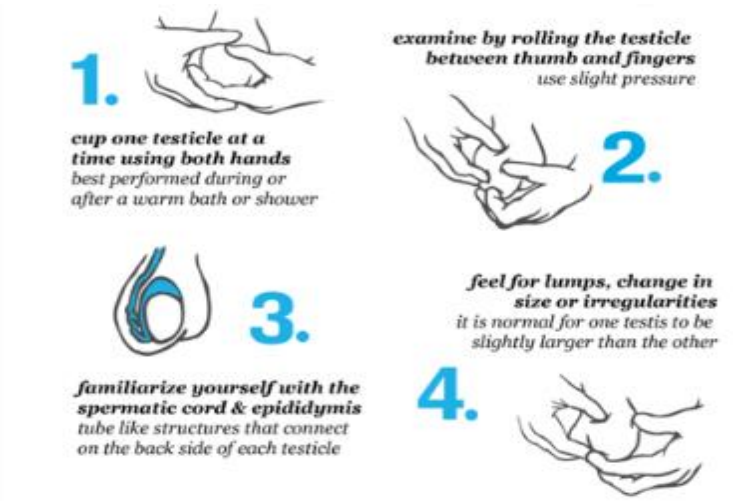
Comlications

- Vasovagal reaction during compression. https://nursingcrib.com/medical-laboratory-diagnostic-test/mammography/

3. Testicular self-exam (TSE)

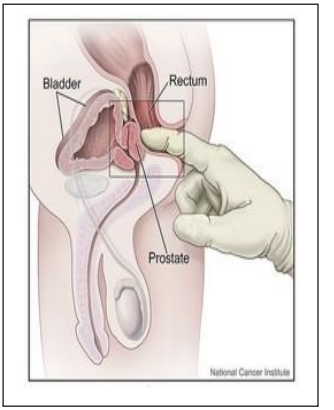
Self-examination of the testes is important for early detection of testicular cancer. The most common method of early detection is performing a monthly exam

- Timing: _____
- Warning sign: _____, pea-sized lump
- Testicular CA high risk: Cryptorchdism
- Management: Orchiopexy – to bring the testicle in the scrotum



4. Digital rectal exam

- Timing: _____ y/o
- Warning sign: Stony-hard prostate
- Normal: boggy, tender (feels like the tip of the nose)



During the digital rectal exam portion, your healthcare provider
carefully inserts their gloved finger (digit) into your rectum.
This allows them to feel the edges and surface of your prostate gland to detect any potential abnormalities.

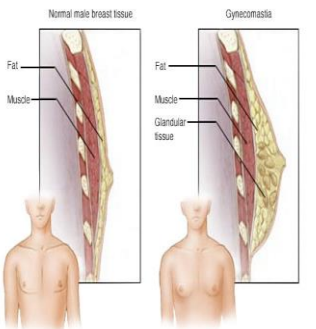
CANCERS (RISK FACTORS)
LUNG CANCER

- Radon gas exposure (cement)
- Smoking
- Pollutants

BREAST CANCER

- Prolonged exposure to hormone (Estrogen)
- Early menarche
- Late menopause
- Null parity – also a risk factor for Ovarian CA
- First child post 35 y/o
- Genetic mutations – BRCA 1 and BRCA 2
- High fat diet
- Obesity
- Caucasian (American women)

Enlarged breasts in men (gynecomastia)



CERVICAL CANCER

- Sexually active below the age of 18
- Infected with human papilloma virus
- Multiple sex partners
- Oral contraceptive usage
- Heavy smoker
- African American

TESTICULAR CANCER

- 15 – 40 y/o

S/Sx

- Painless testicular swelling
- Dragging and pulling sensation

- Palpable lymphadenopathy, abdominal masses
- Gynecomastia

URINE COLLECTION (INFANT)

- Wash the genital
- Open the urine collection bag (wee bag) and place it on the infant
- Diaper the infant
- Check the baby frequently and remove the bag after the infant has urinated

BLADDER CANCER

- Cigarette smoking
- Industrial chemicals
- Exposure to radiation

PROSTATE CANCER

- ABNORMAL: Hard prostate, localized and diffused
- NORMAL: tender and boggy prostate

PANCREATIC CANCER

- Male, African American
- Smoking, toxins
- Diet: High fat, Red meat
- History: Pancreatitis, DM

COLARECTAL CANCER

- Male, African American
- Increasing age, Obese
- Alcohol, Smoking
- Diet: High fat and Protein, Low fiber
- History: CA, Polyps, Inflammatory Bowel Syndrome, gastrectomy

MALIGNANT MELANOMA

- Most common cause of skin cancer-may lead to death
- Poor prognosis even with treatment

Risk factors:

- Exposure to UV light, Family history, Elderly
- Caucasian
- Chronic friction to skin

- Exposure to irritating chemical

NI: Avoid _____
SPF of at least: 15 – 50
Wide brimmed hats

The first five letters of the alphabet are a guide to help you recognize the warning signs of melanoma.
A is for Asymmetry. Most melanomas are asymmetrical. If you draw a line through the middle of the lesion, the two halves don't match, so it looks different from a round to oval and symmetrical common mole.

B is for Border. Melanoma borders tend to be uneven and may have scalloped or notched edges. Common moles tend to have smoother, more even borders.

C is for Color. Multiple colors are a warning sign. While benign moles are usually a single shade of brown, a melanoma may have different shades of brown, tan or black. As it grows, the colors red, white or blue may also appear.

D is for Diameter or Dark. While it's ideal to detect a melanoma when it is small, it's a warning sign if a lesion is the size of a pencil eraser (about 6 mm, or ¼ inch in diameter) or larger. Some experts say it is important to look for any lesion, no matter what size, that is darker than others.

E is for Evolving. Any change in size, shape, color or elevation of a spot on your skin, or any new symptom in it, such as bleeding, itching or crusting, may be a warning sign of melanoma. https://www.skincancer.org/skin-cancer-information/melanoma/melanoma-warning-signs-and-images/

MALIGNANT LYMPHOMAS

	Hodgkin's Lymphoma	**Non-Hodgkin's Lymphoma**
Cause - UNKNOWN	Epstein Barr Virus	Immunosuppression (HIV/AIDS)
Tumor Cells	Reed Sternberg cells	Malignant B lymphocytes or metastatic B cells
Onset of symptoms	Early (stage 1): PAINLESS enlarged cervical lymph node Late: "B symptoms"	Late (stage 3): PAINLESS enlargement of 1 or more lymph nodes "B symptoms" (high grade fever, night sweat, uncontrolled weight loss)
Prognosis	Good	Poor
Diagnosis Test	FNAB – Fine needle aspiration biopsy	
CHEMOTHERAPY / RADIATION THERAPY REVERSE ISOLATION		

Characteristics of Cancer Cells:

Cancer:

1. Uncontrolled cell growth "immature"
2. Capacity to invade and infiltrate/destroy normal body tissue
3. Uncontrolled proliferation
4. Poorly differentiated
5. Altered biochemical properties
6. Chromosomal instability
7. Capacity to metastasize or spread

GRADING AND STAGING

GRADING	STAGING
Grade I: well differentiated (mild dysplasia) Grade II: moderately differentiated (moderate dysplasia) Grade III: poorly differentiated (severe dysplasia) Grade IV: undifferentiated	Stage 0: carcinoma in _____ Stage I: Tumor limited to the _____ Stage II: _____ Stage III: Extensive _____ spread Stage IV: _____

Physiologic Responses to Oncologic and Hematologic Disorders:

1. Pain
2. Cachexia
3. Anemia
4. Thrombocytopenia
5. Leukopenia
6. Infection
7. Neurologic s/sx
8. Altered abdominal function
9. Respiratory depression
10. Paraneoplastic Syndrome

Seven Warning Signs of Cancer

C – hange in _____ and _____ habits

A - _____ that never heals

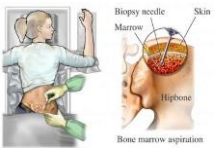
U – nusual _____ or discharge

T – hickening or a _____

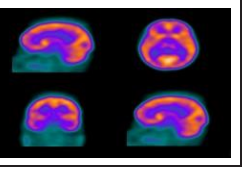
I – ndigestion or difficulty _____ O – bvious change in _____ or _____ N – aging _____ or hoarseness

EARLY DETECTION AND SCREENING

1. Breast self-examination Monthly _____ Days after menses (regular) _____ (Irregular) Menopause: _____ Start with: _____	6. **Sigmoidoscopy/Colonoscopy** Starting: _____ years old Every 5 years: _____ Every 10 years: _____ Preparation: consent, NPO, enema 1 hour before _____ During: monitor heart and respiratory function due to vagal stimulation Post: **WOF**: perforation
2. Mammography Yearly starting: Preparation: do not apply deodorant, power and creams Radiation: _____ hour sun exposure	**7.CT Scan** X-ray beams With or without contrast Keep still for: atleast ____ minutes Contraindication: _____ and patient with _____
3. Pap Smear 4. Routine: 21-29: every _____ years 30-65: every _____	8. **MRI** Magnetic: Field Remove: _____ If claustrophobic: _____

years NO: douching, vaginal medication	Contraindication: _____
5. Testicular Self-Exam _____ after a warm shower 1. Stand in front of the mirror 2. Support testicle with 1 hand and feel each with other Normal: _____ Abnormal: _____	9. **Bone Marrow Exam** Site: Pedia: _____ Adult: _____  Preparation: _____ anasthesia **WOF**: Bleeding/Infection
6. Digital Rectal Examination _____: > 50 years Check: _____ Position: _____ Sign of prostate cancer:stony hard prostate	10. **Oncofetal Antigen** Normal during: _____ Present in adult in certain _____ Example: AFP CEA

A SPECT SCAN is a type of nuclear imaging test, which means
it uses a radioactive substance and a special camera to create
3-D pictures. While imaging test such as X-rays can show what
the structures inside your body look like, a **SPECT SCAN** produces
images that show how your organs work.



TREATMENT MODALITIES

1. Surgery

Types:

1. Diagnostic
2. Prophylactic
3. Curative
4. Control
5. Palliative
6. Reconstructive/Rehabilitative

Chemotherapy

Alkylating Agent -Cell-cyle-nonspecific, toxic to hematologic cells	**Antimeta-bolites** -cell-cyle specific, toxic to hematologic cells	**Plant alkaloids** -cell-cyle specific, inhibit mitosis	**Hormones**	**Anti tumor Antibiotic**
a. Cyclophospha-mide (Cytovan)	a. Methotrexate (Rheumatrex) S.E: Alopecia	a. Vinca alkaloids: Vinblastine (Velban) Vincristine (Oncovin)	a. Tamoxifene (Nolvadex) S.E: Edema Hypercalcemia Increase risk in	a. Doxorubicin (Adriamycin)/ Daunaru-bicin (Dauno-

	Stomatitis Hyperurecemia Hepatotoxity Antidote: Folinic Acid (Leucovorin Rescue)	S.E: Neuropathy Neurotoxic Numbness/ paresthesia Constipation Phlebitis at IV site	uterine cancer	mycin) S.E: Irreversible Cardiomyopathy
b. Cisplatin (Platinol) S.E: Alopecia/ gonadal Suppression/Nephrotoxi-city	b.Cytarabine (Ara-C) S.E: Conjunctivitis with high doses		b. Diethystilbestrol (DES) (Stilphostrol) S.E: Edema Hyperclcemia Impotence Gynecomastia in males	b. dactinomycin (Actinomycin D) * extensively used for pediatric carcinoma
c. Busulfan (Myleran) S.E: Pulmonary Fibrosis	c. Mercaptopurine (6-MP) S.E: hyperuricemia Hepatotoxic d. 5FU (fluorouracil) S.E: alopecia Stomatitis Diarrhea Photosensitivity		c. Testosterone (Depotestosterone) S.E: same with DES d. Prednisone (Deltasone) S.E: Edema Impotence	c.Bleomycin S.E:Pulmonary Fibrosis

A patient is diagnosed with acute lymphocytic leukemia. He is being advised to undergo chemotherapy. They are different chemotherapeutic agents/drugs. Identify which side effect or adverse affect it is from the drug being given.

1. Vincristine (Oncovin)
2. Doxorubucin (Adriamycin)
3. Cyclophosphamide (Cytoxan)

NEUROTOXIC

CARDIOTOXIC

BLADDER TOXIC

OPTIC NEURITIS

CYCTITIS

IV EXTRAVASATION

7. Bone Marrow Transplant

Donor: _____ : compatible to family member

Complications:

_____ : Twin

Failure to engraft

_____ : Self

Graft vs Host Disease

Venous Occlusive Disease

1. HARVEST: _____ ML OF Bone marrow
2. Conditioning : chemotherapy / radiation
3. TRANSPLANT : IV AT bedside
4. ENGRAFTMENT : patient accepts donated bone marrow in _____ weeks

Radiation Therapy

Reduces _____
Anorexia
D _____ mouth
Irritation of mucosa
Alopecia
T _____ effects
Impaired _____ integrity
Over _____
Nausea/Vomiting

<table>
<tr><td>EXTERNAL (TELETHERAPY)</td><td colspan="2">INTERNAL</td></tr>
<tr><td rowspan="2">Do's Don'ts

>keep skin dry
>remove ink marks
>clean skin with
>apply lotion,cream, oil,
mild soap and
>exposed to sunlight water
>wear tigh belt/clothes
>report moist desquamation/
Blister</td><td>Unsealed</td><td>Sealed</td></tr>
<tr><td>Patient and excrete are biohazard up to _____ hours of administration

Flush toilet _____ times

Disinfect toilet with _____

Wash soiled linen _____</td><td>Priority:

CBR without BRP Flat on bed up to 30 degrees if not tolerated enema prior to insertion</td></tr>
</table>

	Don't share bathroom with _____ and _____	

Summary of Radioactivity:

- Put a check mark or x mark:

Radiation Therapy	Radioactivity Patient	Excretions
External		
Internal (sealed)		
Internal (unsealed)		

PRINCIPLES OF BRACHYTHERAPY

A.What should be at bedside in case of dislodgement

1.
2.
3.

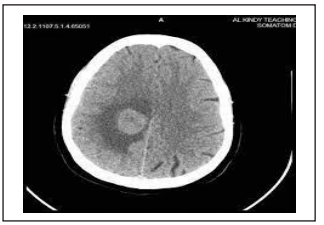
B. Time allowed to be in contact with the patient for the whole shift

_____

C. Distance _____ feet away the patient

D. Type of room _____

BRAIN CANCER



Risk Factors:

1. **B**oys
2. **R**adiation
3. **A**IDS
4. **I**nherited
5. **N**eoplasm of other organs

S/Sx:

1. Cerebral hypoxia

Nursing Diagnosis

2. Increase _____; decrease _____
3. Poor _____ performance

Management:

4. Frontal: _____
5. Parietal: _____
6. Occipital : _____
7. Cerebellar: _____

1. Surgery
2. Radiation
3. Chemotherapy

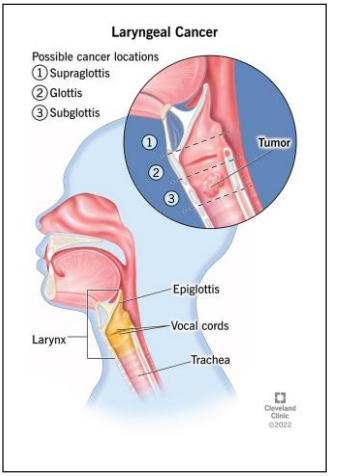
LARYNGEAL CANCER

Risk Factors:

1. Father: _____ men
2. Family history
   Exam
3. F ____ and above (age)
4. Frequent _____
5. Frequent _____

Dx:
1. Laryngoscopic

2. Biopsy



Assessment:

1. Hoarseness
2. Foul breath
3. Weight loss
4. Dysphagia/Dyspnea
5. Painless neck mass

Management: Laryngectomy
Partial/Complete
Change of airway
Post-op mgt: -position:
Provide
Stoma care

Speech therapy

1. Esophageal speech: Instructed:
   1 hour after:
   Hypernasal voice
2. Electro larynx: battery operated apparatus held under lower jaw ____ voice
3. TEP: Fistula between: _____
   Disadvantage: _____
   Normal Voice

LUNG CANCER

Causes: Cigarette smoking
Pollutants

Assessment:

- Cough
- Wheezing
- SOB
- Hemoptysis
- Chest pain
- Hoarseness
- Dysphagia
- Weight loss

Management:

1. Radiation
2. Chemotherapy
3. Surgery
4. Water seal drainage

THYROID CANCER

Risk Factor:

- F _____ y/o
- Female
- F _____ history

Management:

1. Surgery: _____

Hypocalcemia: WOF laryngospasm
Hemorrhage
Edema
Laryngeal never damage
NX: Trache Set

Assessment:

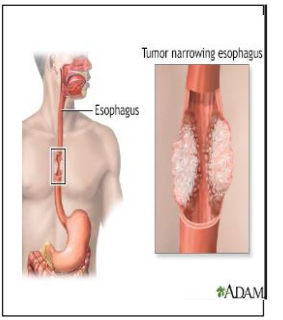
- Painless _____
- Pain in _____
- Pain in _____

2. Radiation: Systemic-RAI-SI/1311
CI: pregnancy, lactation, children
3. Thvroid Replacement: lifetime

ESOPHAGEAL CANCER

Risk Faktor:

- Esophageal _____
- Smoking
- Older than _____ y.o
- Population of African American/poor oral hygiene
- Hereditary
- Alcohol/Achalasia



Assessment:

- Dysphagia-early sign
- Dyspnea-late sign
- Wt, _____ and _____

Management:

1. Surgery: _____

Teaching: stop _____

Diet: CHON and CHO (increase or decrease)

2. Radiation
3. Chemotherapy

GASTRIC CANCER

Risk factors:

- Alcohol
- Born type ____ personality
- C _____ food
- D _____ dietary fiber

Dx: Gastric Analysis

Gastroscopy and Biopsy

Management:

- E _____ race
- Family history
- H _____ infection

1. Surgery
   a. Billroth I: also known as _____
   b. Billroth II: also knwn as _____

Assessment:

- Anorexia
- B _____ in stool
- C _____ emesis
- D _____ in weight
- E _____ pain
- Fatique

Complications: _____

c. Total Gastrectomy

2. Chemotherapy
3. Radiation

LIVER CANCER

Risk Factors:

- Hepatitis ____ and ____
- E ____ pills (hepatotoxic)
- People in Africa and Asia
- Alfatoxins, Alcohol

Dx: Liver Biopsy

Pre: _____ position

Instruct patient to inhale, exhale and hold breath

5 – 10 seconds

Post:_____ position

Assessment:

- Loss of
- Indigestion
- Vomiting
- Enlarged liver
- R _____ pain
- Jaundice
- Ascites
- Hepatomegaly

Management:

1. Surgery-resection
2. Chemotherapy and Radiation
3. Liver transplant

PANCREATIC CANCER

Risk Factors:

- Alcohol/ _____ men
- Cigarette Smoking
- Pancreatitis
- High _____ diet
- Chemotherapy
- Assessment:
- Anorexia
- Abdominal bloating
- Abdominal pain at
- Joundice

Management:

1. Surgery: _____
   Cx: Hypovolemic shock
2. Radiation
4. Drugs necessary after surgery: (pancreatin/ pancrealipase), bile salt, insulin

Dx: Increase amylase, lipase, tripsin, and bilirubin

COLON CANCER

Cause:

- Poor diet: decreased fiber, increase fat and CHON
- Hereditary; History of IBD

Management:

Bowel resection and color of stoma (normal): _____
Opening of pouch: ____ inch larger than the stoma
Liquid stool initially
Observe for leakage
Skin Care: _____
The consistency depends on the location
Once 1/3 or ½ full-empty
Mucus is expected
You avoid gas forming foods

Assessment:

- Blood in stool
- Anorexia
- Abdominal

Abnormal Stools:

- Ascending colon: _____
- Descending colon: constipation
- Rectal: alternating diarrhea and constipation

BREAST CANCER

Risk Factors:

- Advancing maternal age/American
- Breast Cancer history
- Cigarette Smoking
- Diet: increase _____
- Early _____, late _____
- First child after _____ y/o
- Gravida 0 or nulliparity

Assessment:

- **B**leeding/nipple discharges
- **R** _____ or nipple
- **E** _____ of one breast
- **A**symmetry
- **S**kin _____ - orange peel
- **T**hickening/lump

DX:

- **M**ammography
- **A**spiration Biopsy
- **S**taging via
- **S**erum Tumor marker

Management:
Surgery:
_____ tomy: lump + tissue + nipple + lymp node
_____ Mastectomy: MRM + muscle

Nursing Care

Bleeding/discharge monitoring
Recollapse JP drain (-) pressure
E _____ affected arm on pillow, exercise (stress ball, combing)
Assess ____ on the unaffected side
Support group
Turn on _____ side

OVARIAN CANCER

- Grows rapidly, spreads fast and is often bilateral
- Metastasis: _____
- Prognosis:

Risk factors:

- **O**besity
- **V**aginal use of
- **A**ge: >
- **R**ace:
- **I**nfertility
- **A**buse of ____ drug
- **N**ulliparty

Assessment:
A _____ enlargement
A _____
A _____
A _____ disturbances

Management:

1. Surgery: Laparotomy, bilateral salphingo-oophorectomy, TAH_BSO
2. Chemotherapy: taxol (paclitaxel)
3. Radiation
4. Immunotherapy
5. Hormonal Agents: Tamoxifen

ENDOMETRIAL CANCER

Risk factors:

- Family history
- Infertility
- Habitual abortion
- Old age
- Prolonged use of _____ pills
- Endometrial polyps
- Increased Estrogen

Assessment:

- Post-menopausal bleeding;_____
  - vaginal bleeding
- Watery, _____ discharge
- Low back pain, pelvic and abdominal pain
- Enlarge uterus in advance stages

Dx: Endometrial Biopsy
Fractional Curettage

Management:

1. Surgery: Total Hysterectomy and Bilaateral Salphingo-oophorectomy
2. Radiation
3. Hormonal Agents
   Progestational Therapy: Depo-provera (medroxy progesterone) or Megestrol Acetate (Megace)
4. Chemotherapy

CERVICAL CANCER

Risk factors:

- African American woman
- Behavior: Multiple partners
- Chronic instrumentation of the cervix
- Disease: HPV
- Early age of sexual intercouse/pregnancy

Assessment:

- Post ____ bleeding
- Painful ____
- Period irregularities
- Progress: foul smelling discharge
- Pelvic pain

Dx: Pap smear and Schiller test – cervical biopsy

Management:

Surgery – Hysterectomy
Conization

Radiation: Intra cavity Cessium

PROSTATE CANCER

- Slow growing: usually androgen dependent

Risk Factors:

- Family history
- Age
- More common : Obese
- African-american race
- STD/smoking

Assessment: Fequency, urgency,Nocturia
Small urinary stream

Dx: Tumor markers:

1. PSA
   Normal: _____
   BPH: _____
   Prostate Ca: > 10
2. Elevated acid and alkaline
   Phosphatase

Management:

Hormonal manipulation – limit the amount of circulating androgens

- Diethylestilbestrol (DES)
- Leuprolide acetate (Lupron)
- Flutamide (Eulexin)
- Goserelin acetate (Zoladex)

Orchiectomy-limit production of testosterone (palliative)

Transurethral resection of the prostate (TURP)

- Insertion of a scope into the urethra to excise prostatic tissue

TESTICULAR CANCER

Risk factors:

- Male: 15-40 yo
- Race: Caucasian
- Linked to cryptorchidism

Assessment:

- Lump
- Large
- Loaded
- Lymphadenopathy
- Leg pain

Dx: TSE, ↑ AFP, HCG

Treatment:

1. Surgery-unilateral Orchiectomy
2. Radiotion to lympatic
3. Chemotherapy-Cisplatin (Platinol)

Nursing Care:

- Can resume activities after 1 week
- No lifting > 20 lbs
- No stair climbing
- Monthly TSE

Sutures removed after ___ days

BLADDER CANCER

Risk factors:

- Common in male
- Chronic bladder infection
- Chemicals
- Contrast medium
- Chronic use of analgesic

Assessment:

- Frequent urination
- Painless hematuria – earliest
- Dysuria

Dx: Cytoscopy; Biopsy

Management:

1. Surgery
    a. Cystectomy
    b. Ileal Conduit (urinary diversions)
2. Continent Diversion – kock pouch
    - Clean intermittent self-catheterization every ___ hours and before bedtime

RENAL CANCER

Risk Factors:

- Renal stones
- Exposure to ____ and ____
- Nephrotoxic
- Asbestos
- Link to hereditary
-

Classic triad: (Late)

Palpable A _____

Palpable N _____

Painless H _____

Dx:

- IVP ; CT Scan

Treatment:

1. Surgery: Nephrectomy
2. Radiation
3. Chemotherapy
4. Immunotherapy – Intravenous Interleukin

Identify which risk factors are associated with the following type of cancers mentioned: Match Column A to Column B

COLUMN A	COLUMN B
1. Early menarche and late menopause 2. Sexually active below 18 years old 3. Painless testicular swelling 4. Radon gas exposure 5. HPV 6. Genetic mutations- BRCA1 and BRCA2 7. Null parity 8. Pollutants 9. Oral contraceptive usage 10. Smoking	**A. LUNG CANCER** **B. BREAST CANCER** **C. CERVICAL CANCER** **D. TESTICULAR CANCER** **E. BLADDER CANCER**

1. **When reviewing the chart for a patient with cervical, the nurse notes that the cancer is staged as Tis, NO, MO. The nurse will teach the patient that**:

1. The cancer cell are well-differentiated.
2. It is difficult to determine the original site of the cervical cancer
3. Further testing is needed to determine the spread of the cancer
4. The cancer is localized to the cervix

2. **Which of the following interventions is the key to increasing the survival rates of clients with lung cancer?**

1. Early bronchoscopy
2. Early detection
3. High-dose chemotherapy
4. Smoking cessation

3. **The patient is diagnosed with breast cancer. The doctor advised her to undergo a procedure and remove her breast to prevent worsening of the condition. Highlight your nursing interventions and discharge teaching post-mastectomy:**

Elevate affected arm	Hair combing	Assess BP on the affected arm
Cleaning the house	Assess BP on the unaffectes arm	Carrying heavy boxes
Monitor drainage	Dangle the affected arm	Carrying Bag on the affected arm

ONCOLOGIC EMERGENCIES

Emergency	Symptoms	Management
Septic Shock	Put an arrow on the blanks provided, arrow up if increase, down if decrease: • ___ BP • ___ HR • ___ RR • Fever • Chills	• IV Antibiotics • Management of hydration status
Disseminated Intravascular Coagulation (DIC)	• Severe bleeding • ____ Hgb and Hct	• FFP • Cryoprecipitate
Pericardial Temponade	• JVD • ___ SBP • ___ HR • ___ RR • ___ CO • ___ CVP	• Pericardiocentesis • Oxygen • Vasopressor
Superior Vena cava Syndrome	• Altered LOC • JVD • Periorbital/Facial Edema • DOB • Chest Pain • Non-productive cough • Edema and Flushing of arms and shoulders	• Diuretics • Corticosteroids • _____ position • NO BP and venipuncture on UE
Hypercalcemia	• Weakness • Constipation • Dehydration • Dysrhythmia	• Monitor Serum ___ • ECG • hydration
Spinal Cord Compression	• Paresthesia • Pain • Altered reflexes	• Corticosteroids • radiation

	• Cervical: • Thoracic: • Lumbosacral:	
Tumor Lysis Syndrome	• ___ K • ___ Phosphorus • ___ Uric Acid • ___ Calcium • Acute Renal Failure	• Prevention: Adequate hydration 48 hours before and after cytoxic therapy • Diuretics: (Acetazolamides)

REFERENCES

Al-Sukhni, W., Aronson, M., & Gallinger, S. (2008). Hereditary Colorectal Cancer Syndromes: Familial Adenomatous Polyposis and Lynch Syndrome. In *Surgical Clinics of North America* (Vol. 88, Issue 4). https://doi.org/10.1016/j.suc.2008.04.012

America Cancer Society. (2012). *Cancer Facts & Figures 2012*. America Cancer Society.

American Brain Tumor Association. (2023). *A Primer of brain tumor: a patient's reference manual, 8th ed*. ABTA. www.abta.org

American Cancer Society. (2009). *Cancer Facts & Figures 2008*. ACS.

American Cancer Society. (2019). *Prostate Cancer Early Detection, Diagnosis, adn Staging*. Cancer.Org. https://www.cancer.org/content/dam/CRC/PDF/Public/8795.00.pdf

American Cancer Society. (2021). *About Stomach Cancer*. https://www.cancer.org/cancer/stomach-cancer/causes-risks-prevention/risk-factors.html

American Institute for Cancer Research. (2007). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective*.

American Society of Clinical Oncology. (2023a). *Brain Tumor: Statistics*. Cancer.Net. https://www.cancer.net/cancer-types/brain-tumor/statistics#:~:text=Brain tumors account for 85,spinal cord tumor in 2020.

American Society of Clinical Oncology. (2023b). *Breast Cancer: Statistics*. Cancer.Net.

American Society of Clinical Oncology. (2023c). *Cervical Cancer: Statistic*. Cancer.Net.

American Society of Clinical Oncology. (2023d). *Gallbladder Cancer: Statistics*. Cancer.Net. https://www.cancer.net/

cancer-types/gallbladder-cancer/statistics
American Society of Clinical Oncology. (2023e). *Kidney Cancer: Statistic*. Cancer.Net. https://www.cancer.net/cancer-types/kidney-cancer/statistics
American Society of Clinical Oncology. (2023g). *Ovarian, Fallopian Tube, and Peritoneal Cancer: Statistic*. Cancer.Net.
American Society of Clinical Oncology. (2023h). *Pancreatic Cancer: Statistic*. Cancer.Net.
American Society of Clinical Oncology. (2023i). *Prostate Cancer: Statistics*. Cancer.Net. https://www.cancer.net/cancer-types/prostate-cancer/statistics
American Society of Clinical Oncology. (2023j). *Testicular Cancer: Statistics*. Cancer.Net.
American Society of Clinical Oncology. (2023k). *Thyroid Cancer: Statistics*. Cancer.Net.
Andrén-Sandberg, Å. (2012). Molecular biology of Gallbladder cancer: Potential clinical implications. In *North American Journal of Medical Sciences* (Vol. 4, Issue 10). https://doi.org/10.4103/1947-2714.101979
Arzumanyan, A., Reis, H. M. G. P. V., & Feitelson, M. A. (2013). Pathogenic mechanisms in HBV- and HCV-associated hepatocellular carcinoma. In *Nature Reviews Cancer* (Vol. 13, Issue 2). https://doi.org/10.1038/nrc3449
Birnbaum, D. J., Mamessier, E., & Birnbaum, D. (2012). The emerging role of the TGFβ tumor suppressor pathway in pancreatic cancer. In *Cell Cycle* (Vol. 11, Issue 4). https://doi.org/10.4161/cc.11.4.19130
Blonski, W., Kotlyar, D. S., & Forde, K. A. (2010). Non-viral causes of hepatocellular carcinoma. In *World Journal of Gastroenterology* (Vol. 16, Issue 29). https://doi.org/10.3748/wjg.v16.i29.3603
Bornschein, J., Rokkas, T., Selgrad, M., & Malfertheiner, P. (2011). Gastric cancer: clinical aspects, epidemiology and molecular background. *Helicobacter*, *16*(1).
CA, G., N, T., X, L.-B. L. C., FX, B., E, R., D, B.-M. H. P.,

H, B., V, P., C, S., R, T., S, P., J, M., J, D., G, H., L, K., MJ, S., JM, A., A, B., C, N., … E, R. (2011). Dietary factors and in situ and invasive cervical cancer risk in the European prospective investigation into cancer and nutrition study. *International Journal of Cancer*, *129*(2), 449–459.

Cabibbo, G., Maida, M., Genco, C., Antonucci, M., & Cammà, C. (2012). Causes of and prevention strategies for hepatocellular carcinoma. *Seminars in Oncology*, *39*(4). https://doi.org/10.1053/j.seminoncol.2012.05.006

Carcio, H. A. (2011). *Advanced Health Assessment of Women: Skill, Procedures, and Management, Second Edition* (2nd ed.). Springer Publishing.

Carr, L. L., Finigan, J. H., & Kern, J. A. (2011). Evaluation and treatment of patients with non-small cell lung cancer. In *Medical Clinics of North America* (Vol. 95, Issue 6). https://doi.org/10.1016/j.mcna.2011.08.001

Carruba, G., Webber, M. M., Quader, S. T. A., Amoroso, M., Cocciadiferro, L., Saladino, F., Trosko, J. E., & Castagnetta, L. A. M. (2002). Regulation of Cell-to-Cell Communication in Non-Tumorigenic and Malignant Human Prostate Epithelial Cells. *Prostate*, *50*(2).

Casadei, R., Monari, F., Buscemi, S., Laterza, M., Ricci, C., Rega, D., D'Ambra, M., Pezzilli, R., Calculli, L., Santini, D., & Minni, F. (2010). Total pancreatectomy: Indications, operative technique, and results: A single centre experience and review of literature. In *Updates in Surgery* (Vol. 62, Issue 1). https://doi.org/10.1007/s13304-010-0005-z

Center For Disease Control and Prevention. (2006). *Fast Facts and Fact Sheets: Smoking and tobacco uses, fact sheet*. CDC.

Champion, V. L., Monahan, P. O., Springston, J. K., Russell, K., Zollinger, T. W., Saywell, R. M., & Maraj, M. (2008). Measuring mammography and breast cancer beliefs in African American women. *Journal of Health Psychology*, *13*(6). https://doi.org/10.1177/1359105308093867

Chen, S. L., Iddings, D. M., Scheri, R. P., & Bilchik, A. J. (2006). Lymphatic Mapping and Sentinel Node Analysis: Current Concepts and Applications. *A Cancer Journal For Clinicians*, *56*(5), 292–309.

Cogliano, V. J., Baan, R., Straif, K., Grosse, Y., Secretan-Lauby, B., Ghissassi, F. El, Bouvard, V., Benbrahim-Tallaa, L., Guha, N., Freeman, C., Galichet, L., & Wild, C. P. (2011). Preventable exposures associated with human cancers. *Journal of The National Cancer Institute*, *103*(24).

Compare, D., Rocco, A., & Nardone, G. (2010). Risk factors in gastric cancer. *European Review for Medical and Pharmacological Sciences*, *14*(4), 302–308.

Cooperberg, M., Presti, J. J., Shinahara, K., & Carrol, P. (2013). Neoplasm of the Prostate Gland. In *Smith and Tanagho's General Urology* (18th ed., pp. 350–379). Mc Graw Hill.

Corbo, V., Tortora, G., & Scarpa, A. (2012). Molecular Pathology of Pancreatic Cancer: From Bench-to-Bedside Translation. *Current Drug Targets*, *13*(6). https://doi.org/10.2174/138945012800564103

Cornett, P., & Dea, T. (2013). 2009 Current Medical Diagnosis and Treatment. In *Current Medical Diagnosis and Treatment* (52nd ed., pp. 1593–1663). Mc Graw Hill.

Curry, S. J., Krist, A. H., Owens, D. K., Barry, M. J., Caughey, A. B., Davidson, K. W., Doubeni, C. A., Epling, J. W., Kemper, A. R., Kubik, M., Seth Landefeld, C., Mangione, C. M., Phipps, M. G., Silverstein, M., Simon, M. A., Tseng, C. W., & Wong, J. B. (2018). Screening for cervical cancer us preventive services task force recommendation statement. *JAMA - Journal of the American Medical Association*, *320*(7). https://doi.org/10.1001/jama.2018.10897

Daley, W. P., Peters, S. B., & Larsen, M. (2008). Extracellular Matrix Dynamics in Development and Regenerative Medicine. *Journal of Cell Science*, *121*(3), 255–264.

Dalton, J. A., & Youngblood, R. (2000). Clinical application of the World Health Organization analgesic ladder. In *Journal of Intravenous Nursing* (Vol. 23, Issue 2).

de Groot, P., & Munden, R. F. (2012). Lung Cancer Epidemiology, Risk Factors, and Prevention. In *Radiologic Clinics of North America* (Vol. 50, Issue 5). https://doi.org/10.1016/j.rcl.2012.06.006

De Salvo, G. L., Gava, C., Lise, M., & Pucciarelli, S. (2004). Curative surgery for obstruction from primary left colorectal carcinoma: Primary or staged resection? In *Cochrane Database of Systematic Reviews* (Vol. 2004, Issue 2). https://doi.org/10.1002/14651858.CD002101.pub2

Duell, E. J. (2012). Epidemiology and potential mechanisms of tobacco smoking and heavy alcohol consumption in pancreatic cancer. *Molecular Carcinogenesis*, *51*(1). https://doi.org/10.1002/mc.20786

Dutcher, J. P., Mourad, W. F., & Ennis, R. D. (2012). Integrating innovative therapeutic strategies into the management of renal cell carcinoma. In *ONCOLOGY (United States)* (Vol. 26, Issue 6).

Dutta, U. (2012). Gallbladder cancer: Can newer insights improve the outcome? *Journal of Gastroenterology and Hepatology (Australia)*, *27*(4). https://doi.org/10.1111/j.1440-1746.2011.07048.x

El-Serag, H. (2011). Hepatocellular carcinoma. *The New England Journal of Medicine*, *365*(12), 1118–1127.

Fazal, S., & Saif, M. W. (2007). Supportive and palliative care of pancreatic cancer. *Journal of the Pancreas*, *8*(2).

Fisher, G. J., & Voorhees, J. J. (1998). Molecular mechanisms of photoaging and its prevention by retinoic acid: Ultraviolet irradiation induces MAP kinase signal transduction cascades that induce Ap-1-regulated matrix metalloproteinases that degrade human skin in vivo. *Journal of Investigative Dermatology Symposium Proceedings*, *3*(1). https://doi.org/10.1038/jidsymp.1998.15

Fontham, E. T. (1994). Environmental tobacco smoke and lung cancer in nonsmoking women. A multicenter study. *JAMA: The Journal of the American Medical Association*,

271(22). https://doi.org/10.1001/jama.271.22.1752

Forner, A., Reig, M., & Bruix, J. (2018). Hepatocellular carcinoma. In *The Lancet* (Vol. 391, Issue 10127). https://doi.org/10.1016/S0140-6736(18)30010-2

Fuks, D., Regimbeau, J. M., Le Treut, Y. P., Bachellier, P., Raventos, A., Pruvot, F. R., Chiche, L., & Farges, O. (2011). Incidental gallbladder cancer by the AFC-GBC-2009 study group. *World Journal of Surgery*, *35*(8). https://doi.org/10.1007/s00268-011-1134-3

Gaddam, S., & Wani, S. (2013). Endoscopic therapy of Barrett esophagus. *Gastrointestinal Endoscopy Clinics of North America*, *23*(1), 1–16.

Gigek, C. O., Chen, E. S., Calcagno, D. Q., Wisnieski, F., Bubrano, R. R., & Smith, M. A. (2012). Epigenetic mechanisms in gastric cancer. *Epigenomics*, *4*(3), 279–294.

Gilligan, R. (2007). Adversity, resilience and the educational progress of young people in public care. *Emotional and Behavioural Difficulties*, *12*(2). https://doi.org/10.1080/13632750701315631

GL, M., J, P., & DA, S. (2012). The Female Reproductive System. In *Rubin's Pathology: Clinicopathologic Foundations of Medicine* (6th ed.). Wolters Kluwer Health/Lippincott Williams & Wilkins.

Global Headquarters: American Cancer Society. (2020). *Colorectal Cancer: Facts & Figures 2020-2022*. https://www.cancer.org/content/dam/cancer-org/research/cancer-facts-and-statistics/colorectal-cancer-facts-and-figures/colorectal-cancer-facts-and-figures-2020-2022.pdf

Goel, A., & Boland, C. R. (2012). Epigenetics of colorectal cancer. *Gastroenterology*, *143*(16).

Goldberg, S. R., & Diegelmann, R. F. (2010). Wound Healing Primer. *Surgical Clinics of North America*, *90*(6), 1133–1146.

Gonzalez, C. A., & Riboli, E. (2010). Diet and cancer prevention: Contributions from the European Prospective

Investigation into Cancer and Nutrition (EPIC) study. *European Journal of Cancer*, *46*(14), 2555–2562.

Google. (n.d.). *Nursing Process: The Patient with Cancer*. BrainKart.Com. https://www.brainkart.com/article/Nursing-Process-The-Patient-With-Cancer_31754/

Gore, R. M., & Shelhamer, R. P. (2007). Biliary tract neoplasms: Diagnosis and staging. *Cancer Imaging*, *7*(SPEC. ISS. A). https://doi.org/10.1102/1470-7330.2007.9016

Gorin, M. A., Ayyathurai, R., & Soloway, M. S. (2012). Diagnosis and treatment of bladder cancer: How can we improve? *Postgraduate Medicine*, *124*(3). https://doi.org/10.3810/pgm.2012.05.2545

Gralow, J. R., Biermann, J. S., Farooki, A., Fornier, M. N., Gagel, R. F., Kumar, R., Litsas, G., McKay, R., Podoloff, D. A., Srinivas, S., & Van Poznak, C. H. (2013). NCCN task force report: Bone health in cancer care. *JNCCN Journal of the National Comprehensive Cancer Network*, *11*(SUPPL.3). https://doi.org/10.6004/jnccn.2013.0215

Greco, C., Pares, O., Pimentel, N., Louro, V., Santiago, I., Vieira, S., Stroom, J., Mateus, D., Soares, A., Marques, J., Freitas, E., Coelho, G., Seixas, M., Lopez-Beltran, A., & Fuks, Z. (2021). Safety and efficacy of virtual prostatectomy with single-dose radiotherapy in patients with intermediate-risk prostate cancer results from the PROSINT phase 2 randomized clinical trial. *JAMA Oncology*, *7*(5). https://doi.org/10.1001/jamaoncol.2021.0039

Hainsworth, J. D., Lennington, W. J., & Greco, F. A. (2000). Overexpression of Her-2 in patients with poorly differentiated carcinoma or poorly differentiated adenocarcinoma of unknown primary site. *Journal of Clinical Oncology*, *18*(3). https://doi.org/10.1200/jco.2000.18.3.632

Herreros-Villanueva, M., Hijona, E., Cosme, A., & Bujanda, L. (2012). Adjuvant and neoadjuvant treatment in pancreatic cancer. In *World Journal of Gastroenterology* (Vol. 18, Issue 14). https://doi.org/10.3748/wjg.v18.i14.1565

Institute of Medicine. (2001). *Rebuilding the Unity of Health*

and the Environment: A New Vision of Environmental Health for the 21st Century.

Ioannou, G. N., Splan, M. F., Weiss, N. S., McDonald, G. B., Beretta, L., & Lee, S. P. (2007). Incidence and Predictors of Hepatocellular Carcinoma in Patients With Cirrhosis. *Clinical Gastroenterology and Hepatology*, *5*(8). https://doi.org/10.1016/j.cgh.2007.02.039

Islami, F., & Kamangar, F. (2008). Helicobacter pylori and esophageal cancer risk: a meta-analysis. *Cancer Prevention Research*, *1*(5), 329–338.

Jemal, A., Ward, E. M., Johnson, C. J., Cronin, K. A., Ma, J., Ryerson, A. B., Mariotto, A., Lake, A. J., Wilson, R., Sherman, R. L., Anderson, R. N., Henley, S. J., Kohler, B. A., Penberthy, L., Feuer, E. J., & Weir, H. K. (2017). Annual Report to the Nation on the Status of Cancer, 1975-2014, Featuring Survival. In *Journal of the National Cancer Institute* (Vol. 109, Issue 9). https://doi.org/10.1093/jnci/djx030

Kaz, A., Luo, Y., Dzieciatkowski, S., Chak, A., Willis, J., Upton, M., Leidner, R., & Grady, W. (2012). Aberrantly methylated PKP1 in the progression of Barrett's esophagus to esophageal adenocarcinoma. *Genes Chromosomes Cancer*, *51*(4), 384–393.

Kelly, D. (2013). An encephalitis primer. *Advances in Experimental Medicine and Biology*, *764*. https://doi.org/10.1007/978-1-4614-4726-9_10

Kiriluk, K. J., Prasad, S. M., Patel, A. R., Steinberg, G. D., & Smith, N. D. (2012). Bladder cancer risk from occupational and environmental exposures. In *Urologic Oncology: Seminars and Original Investigations* (Vol. 30, Issue 2). https://doi.org/10.1016/j.urolonc.2011.10.010

Klöppel, G., & Lüttges, J. (2004). The pathology of ductal-type pancreatic carcinomas and pancreatic intraepithelial neoplasia: Insights for clinicians. In *Current Gastroenterology Reports* (Vol. 6, Issue 2). https://doi.org/10.1007/s11894-004-0037-y

Koukoura, O., Sifakis, S., & Spandidos, D. A. (2012). DNA

methylation in the human placenta and fetal growth (Review). *Molecular Medicine Reports*, *5*(4), 883–889.

Kudo, Y., Kamisawa, T., Anjiki, H., Takuma, K., & Egawa, N. (2011). Incidence of and risk factors for developing pancreatic cancer in patients with chronic pancreatitis. *Hepato-Gastroenterology*, *58*(106).

Lencioni, R. (2012). Chemoembolization for hepatocellular carcinoma. *Seminar Oncology*, *39*(4), 503–509.

Leonardi, G., Vahter, M., Clemens, F., Goessler, W., Gurzau, E., Hemminki, K., Hough, R., Koppova, K., Kumar, R., Rudnai, P., Surdu, S., & Fletcher, T. (2012). Inorganic arsenic and basal cell carcinoma in areas of Hungary, Romania, and Slovakia: A case-control study. *Environmental Health Perspectives*, *120*(5). https://doi.org/10.1289/ehp.1103534

Logan, R. F. (2012). A single FIT (≥ 50 ng Hb/mL) detected 38% of screen-detected advanced colorectal neoplasia. *Annals of Internal Medicine*, *157*(10). https://doi.org/10.7326/0003-4819-157-10-201211200-02010

Lomberk, G. (2008). Pain Management. *Pancreatology*, *8*(6), 542–543.

Lønning, P. E. (2011). The potency and clinical efficacy of aromatase inhibitors across the breast cancer continuum. In *Annals of Oncology* (Vol. 22, Issue 3). https://doi.org/10.1093/annonc/mdq337

Loveday, C., Turnbull, C., Ramsay, E., Hughes, D., Ruark, E., Frankum, J. R., Bowden, G., Kalmyrzaev, B., Warren-Perry, M., Snape, K., Adlard, J. W., Barwell, J., Berg, J., Brady, A. F., Brewer, C., Brice, G., Chapman, C., Cook, J., Davidson, R., … Rahman, N. (2011). Germline mutations in RAD51D confer susceptibility to ovarian cancer. *Nature Genetics*, *43*(9). https://doi.org/10.1038/ng.893

Lowenfels, A. B., Maisonneuve, P., Boyle, P., & Zatonski, W. A. (1999). Epidemiology of gallbladder cancer. In *Hepato-Gastroenterology* (Vol. 46, Issue 27). https://doi.org/10.1007/978-981-15-5289-2_3

Ma, Y. T., Collins, S. I., Young, L. S., Murray, P. G., & Woodman, C. B. J. (2011). Smoking initiation is followed by the early acquisition of epigenetic change in cervical epithelium: a longitudinal study. *British Journal of Cancer*, *104*(9).

Macgregor-Das, A. M., & Lacobuzio-Donahue, C. A. (2013). Molecular Pathways in Pancreatic Carcinogenesis. *Journal of Surgical Oncology*, *107*(1), 8–14.

Marsh, R. D. W., Alonzo, M., Bajaj, S., Baker, M., Elton, E., Farrell, T. A., Gore, R. M., Hall, C., Nowak, J., Roy, H., Shaikh, A., & Talamonti, M. S. (2012). Comprehensive review of the diagnosis and treatment of biliary tract cancer 2012. PART I: Diagnosis-clinical staging and pathology. In *Journal of Surgical Oncology* (Vol. 106, Issue 3). https://doi.org/10.1002/jso.23028

Marutsuka, T., Shimada, S., Shiomori, K., Hayashi, N., Yagi, Y., Yamamane, T., & Ogawa, M. (2003). Mechanism of Peritoneal Metastasis After Operation for Non-Serosa-Invasive Gastric Carcinoma: An Ultrarapid Detection System for Intraperitoneal Free Cancer Cells and a Prophylactic Strategy for Peritoneal Metastasis. *Clinical Cancer Research*, *9*(2), 678–685.

Masuzaki, R., Karp, S. J., & Omata, M. (2012). New serum markers of hepatocellular carcinoma. *Seminars in Oncology*, *39*(4). https://doi.org/10.1053/j.seminoncol.2012.05.009

Mawhinney, M. R., & Glasgow, R. E. (2012). Current treatment options for the management of esophageal cancer. *Cancer Management and Research*, *4*, 367–377.

McCance, K. L., & Huether, S. E. (2019). *Pathophysiology: The Biologic Basis for Disease in Adults and Children* (8th ed.). Elsevier.

McGlynn, K. A., & London, W. T. (2011). The Global Epidemiology of Hepatocellular Carcinoma: Present and Future. In *Clinics in Liver Disease* (Vol. 15, Issue 2). https://doi.org/10.1016/j.cld.2011.03.006

Mehnert, J. M., & Kluger, H. M. (2012). Driver mutations

in melanoma: Lessons learned from bench-to-bedside studies. *Current Oncology Reports*, *14*(5). https://doi.org/10.1007/s11912-012-0249-5

Melcalfe, A., & Ferguson, M. (2008). Skin Stem and Progenitor Cells: Using Regeneration as a Tissue Engineering Strategy. *Cellular and Mollecular Life Science*, *65*(1), 24–32.

Morgan, E., Soerjomataram, I., Rumgay, H., Coleman, H. G., Thrift, A. P., Vignat, J., Laversanne, M., Ferlay, J., & Arnold, M. (2022). The Global Landscape of Esophageal Squamous Cell Carcinoma and Esophageal Adenocarcinoma Incidence and Mortality in 2020 and Projections to 2040: New Estimates From GLOBOCAN 2020. *Gastroenterology*, *163*(3).

Narayan, G., & Murty, V. V. (2010). Integrative genomic approaches in cervical cancer: Implications for molecular pathogenesis. *Future Oncology*, *6*(10). https://doi.org/10.2217/fon.10.114

National Cancer Institute. (2010). *Reducing Environtmental Cancer Risk: What We Can do Now: President's Cancer Panel 2008-2009 Annual Report*.

National Cancer Institute. (2018). *Secondhand Smoke and Cancer*. https://www.cancer.gov/about-cancer/causes-prevention/risk/tobacco/second-hand-smoke-fact-sheet

National Cancer Institute. (2019). *SEER Cancer Stat Facts*. National Institutes of Health.

National Cancer Institute. (2023a). *Colon Cancer Treatment (PDQ®)–Patient Version*. NIH. https://www.cancer.gov/types/colorectal/patient/colon-treatment-pdq

National Cancer Institute. (2023b). *Colorectal Cancer Screening (PDQ®)–Health Professional Version*. NIH. https://www.cancer.gov/types/colorectal/hp/colorectal-screening-pdq

National Center for Environmental Health. (2011). *CDC's Tobacco Laboratory: 2008-2009*. Centers for Disease Control and Prevention.

National Toxicology Program. (2011). *NTP 12th Report on*

Carcinogens.

Nelson, W. G., De Marzo, A. M., & Isaacs, W. B. (2003). Prostate Cancer. *The New England Journal of Medicine*, *349*(4), 366–381.

Ness, M. V, Gregg, J., Wang, J., & Chen, M. (2012). Genetics and molecular pathology of gastric malignancy: Development of targeted therapies in the era of personalized medicine. *Journal of Gastrointestinal Oncology*, *3*(3), 243–251.

Nguyen, T. D., & DeAngelis, L. M. (2007). Brain Metastases. In *Neurologic Clinics* (Vol. 25, Issue 4). https://doi.org/10.1016/j.ncl.2007.07.011

Nogueira, S. M., & Appling, S. E. (2000). Breast cancer: genetics, risks, and strategies. In *The Nursing clinics of North America* (Vol. 35, Issue 3).

Nugent, P. M., Green, J. S., & Ann, H. S. M. (2014). *Mosby's comprehensive review of nursing for the NCLEX-RN examination*. Elsevier.

Oncology, A. S. of C. (2023). *Bladder Cancer: Statistics*. Cancer.Net.

Palmer, W. C., & Patel, T. (2012). Are common factors involved in the pathogenesis of primary liver cancers? A meta-analysis of risk factors for intrahepatic cholangiocarcinoma. *Journal of Hepatology*, *57*(1). https://doi.org/10.1016/j.jhep.2012.02.022

Pandey, M. (2003). Risk factors for gallbladder cancer: A reappraisal. In *European Journal of Cancer Prevention* (Vol. 12, Issue 1). https://doi.org/10.1097/00008469-200302000-00004

Pettet, A. G., & Flinton, D. M. (2022). Radiotherapy related fatigue: strategies and knowledge of the UK radiographer and nurse. *Radiography*, *8*(3), 149–158. https://www.sciencedirect.com/science/article/abs/pii/S1078817402903718

Porth, C. M. (2015). *Essentials of Pathophysiology: Concepts of Altered Health States* (4th ed.). Wolters Kluwer Health/Lippincott Williams & Wilkins.

Raimondi, S., Lowenfels, A. B., Morselli-Labate, A. M.,

Maisonneuve, P., & Pezzilli, R. (2010). Pancreatic cancer in chronic pancreatitis; Aetiology, incidence, and early detection. *Best Practice and Research: Clinical Gastroenterology*, *24*(3). https://doi.org/10.1016/j.bpg.2010.02.007

Reid, B. J., Kostadinov, R., & Maley, C. C. (2011). New Strategies in Barrett's Esophagus Integrating clonal evolutionary theory with clinical management. *Clinical Cancer Research*, *17*(11), 3512–3519.

Robbins, S. L., Kumar, V., & Cotran, R. S. (2010). *Robbins and Cotran Pathologic Basis of Disease* (8th ed.). Saunders/Elsevier.

Rowland, L. P. (2005). *Merritt's Neurology* (11th ed.). Lippincott Williams & Wilkins.

Russak, J. E., & Rigel, D. S. (2012). Risk Factors for the Development of Primary Cutaneous Melanoma. In *Dermatologic Clinics* (Vol. 30, Issue 3). https://doi.org/10.1016/j.det.2012.05.002

Sankaranarayanan, R., Nene, B. M., Shastri, S. S., Jayant, K., Muwonge, R., Budukh, A. M., Hingmire, S., Malvi, S. G., Thorat, R., Kothari, A., Chinoy, R., Kelkar, R., Kane, S., Desai, S., Keskar, V. R., Rajeshwarkar, R., Panse, N., & Dinshaw, K. A. (2009). HPV Screening for Cervical Cancer in Rural India. *New England Journal of Medicine*, *360*(14). https://doi.org/10.1056/nejmoa0808516

Schauer, I. G., Sood, A. K., Mok, S., & Liu, J. (2011). Cancer-associated fibroblasts and their putative role in potentiating the initiation and development of epithelial ovarian cancer. In *Neoplasia* (Vol. 13, Issue 5). https://doi.org/10.1593/neo.101720

Scientific Figure on ResearchGate. (n.d.). *Establishment of a 3D tumour model and targeted therapy of BRAF-mutant colorectal cancer*. ResearchGate. https://www.researchgate.net/figure/Stages-of-colorectal-cancer-CRC-The-development-of-CRC-usually-begins-by-a_fig1_337929033

Sgourakis, G., Gockel, I., Karaliotas, C., Moehler, M.,

Schimanski, C. C., Schmidberger, H., & Junginger, T. (2012). Survival after chemotherapy and/or radiotherapy versus self-expanding metal stent insertion in the setting of inoperable esophageal cancer: A case-control study. *BMC Cancer*, *12*. https://doi.org/10.1186/1471-2407-12-70

Smith, R. A., Andrews, K. S., Brooks, D., Fedewa, S. A., Manassaram-Baptiste, D., Saslow, D., Brawley, O. W., & Wender, R. C. (2018). Cancer screening in the United States, 2018: A review of current American Cancer Society guidelines and current issues in cancer screening. *CA: A Cancer Journal for Clinicians*, *68*(4). https://doi.org/10.3322/caac.21446

Society of Gynecologic Oncology. (2014). *Carcinoma of the Cervix*. Staging Handbook. https://staginghandbook.weebly.com/carcinoma-of-the-cervix.html

Stoita, A., Penman, I. D., & Williams, D. B. (2011). Review of screening for pancreatic cancer in high risk individuals. In *World Journal of Gastroenterology* (Vol. 17, Issue 19). https://doi.org/10.3748/wjg.v17.i19.2365

Sun, M., Shariat, S. F., Cheng, C., Ficarra, V., Murai, M., Oudard, S., Pantuck, A. J., Zigeuner, R., & Karakiewicz, P. I. (2011). Prognostic factors and predictive models in renal cell carcinoma: A contemporary review. In *European Urology* (Vol. 60, Issue 4). https://doi.org/10.1016/j.eururo.2011.06.041

Tanagho, E. A., & McAninch, J. W. (2008). *Smith's General Urology* (17th ed.). Mc Graw Hill.

Teller, P., & White, T. K. (2009). The Physiology of Wound Healing: Injury Through Maturation. *Surgical Clinics of North America*, *89*(3), 599–610.

Thomas, M. B. (2008). Targeted therapies for cancer of the gallbladder. In *Current Opinion in Gastroenterology* (Vol. 24, Issue 3). https://doi.org/10.1097/MOG.0b013e3282f6a7df

Tobler, N. E., & Detmar, M. (2006). Tumor and Lymph Node Lymphangiogenesis-Impact on Cancer Metastasis.

Journal of Leukocyte Biology, *80*(4), 691–696.

Trichopoulos, D., Bamia, C., Lagiou, P., Fedirko, V., Trepo, E., Jenab, M., Pischon, T., Nöthlings, U., Overved, K., Tjønneland, A., Outzen, M., Clavel-Chapelon, F., Kaaks, R., Lukanova, A., Boeing, H., Aleksandrova, K., Benetou, V., Zylis, D., Palli, D., … Riboli, E. (2011). Hepatocellular carcinoma risk factors and disease burden in a european cohort: A nested case-control study. *Journal of the National Cancer Institute*, *103*(22). https://doi.org/10.1093/jnci/djr395

Tummala, P., Junaidi, O., & Agarwal, B. (2011). Imaging of pancreatic cancer: An overview. *Journal of Gastrointestinal Oncology*, *2*(3). https://doi.org/10.3978/j.issn.2078-6891.2011.036

Tyson, G. L., & El-Serag, H. B. (2011). Risk factors for cholangiocarcinoma. *Hepatology*, *54*(1). https://doi.org/10.1002/hep.24351

Vats, A., Bielby, R., Tolley, N., Nerem, R., & Polak, J. (2005). Stem Cells. *Lancet*, *366*(9485), 592–602.

Vesco, K. K., Whitlock, E. P., Eder, M., Lin, J., Burda, B., Senger, C. A., Holmes, R. S., Fu, R., & Zuber, S. (2011). *Screening for Cervical Cancer*.

Vincent, A., Herman, J., Schulick, R., Hruban, R. H., & Goggins, M. (2011). Pancreatic Cancer. *Lancet*, *378*(9791), 607–620.

Wang, F., Wang, B., & Qiao, L. (2012). Association between obesity and gallbladder cancer. In *Frontiers in bioscience : a journal and virtual library* (Vol. 17). https://doi.org/10.2741/4070

Wang, L.-H. (2004). Molecular Signaling Regulating Anchorage-Independent Growth of Cancer Cells. *Mt Sinai Journal of Medicine*, *71*(6), 361–367.

Wang, P., Mei, J., Tao, J., Zhang, N., Tian, H., & Fu, G.-H. (2012). Effects of Helicobacter pylori on biological characteristics of gastric epithelial cells. *Histol Histopathology*, *27*(8), 1079–1091.

Wang, S. (2013). Lung Cancer. In *Current Medical Diagnosis*

and Treatment (52nd ed., pp. 1595–1602). Mc Graw Hill.

Wender, R., Fontham, E. T., Jr Barrera, E., Colditz, G. A., Church, T. S., Ettinger, D. S., Etzioni, R., Flowers, C. R., Gazelle, G. S., Kelsey, D. K., LaMonte, S. J., Michaelson, J. S., Oeffinger, K. C., Shih, Y.-C. T., Sullivan, D. C., Travis, W., Walter, L., Wolf, A. M., Brawley, O. W., & Smith, R. A. (2013). American Cancer Society Lung Cancer Screening Guidelines. *CA: A Cancer Journal for Clinicians*, *63*(2), 107–117.

Yahalom, J. (2011). Patients with H pylori-independent MALT lymphoma are curable with radiotherapy. *Oncology (Williston Park)*, *25*(12).

Yee, N. S. (2013). Toward the goal of personalized therapy in pancreatic cancer by targeting the molecular phenotype. In *Advances in Experimental Medicine and Biology* (Vol. 779). https://doi.org/10.1007/978-1-4614-6176-0_5

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