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Anatomy

1. Primary Obesity is a chronic condition defined by an excess amount of body fat.

2. (A) Some medicines might affect your body's metabolism. This causes your body to burn calories at a slower rate. A number of drugs that cause less weight gain or even promote weight loss are becoming available to treat these conditions. Awareness of the effects of these agents on body weight may allow modification of a patient's regimen to avoid excessive weight gain. Among the antipsychotics, risperidone, sertindole, olanzapine, and clozapine were found to cause weight gains ranging from 2.1–4.5 kg over the course of 10 wk of treatment.Certain drugs either promote anorexia by potentiating the CNS effect of norepinephrine or both norepinephrine and serotonin or decrease absorption of fat from the gastrointestinal tract. Selective noradrenergic drugs generally produce a 3–8% weight loss compared with placebo and may cause palpitations, tachycardia, insomnia, hypertension, and dry mouth. These drugs have not been independently associated with pulmonary hypertension or valvular heart disease.

(B) The negative impact of obesity can be greater in children with congenital heart disease (CHD), as these patients have underlying myocardial abnormalities on which the cardiovascular risk factors associated to obesity can be superimposed.

3. Cancer is the uncontrolled growth of abnormal cells anywhere in a body. These abnormal cells are termed cancer cells, malignant cells, or tumor cells. These cells can infiltrate normal body tissues. Many cancers and the abnormal cells that compose the cancer tissue are further identified by the name of the tissue that the abnormal cells originated from (for example, breast cancer, lung cancer, colorectal cancer). Cancer is not confined to humans; animals and other living organisms can get cancer.
   Anything that may cause a normal body cell to develop abnormally potentially can cause cancer. Many things can cause cell abnormalities and have been linked to cancer development. Some cancer causes remain unknown while other cancers have environmental or lifestyle triggers or may develop from more than one known cause. Some may be developmentally influenced by a person's genetic makeup. Many patients develop cancer due to a combination of these factors. Although it is often difficult or impossible to determine the initiating event(s) that cause a cancer to develop in a specific person, research has provided clinicians with a number of likely causes that alone or in concert with other causes, are the likely candidates for initiating cancer. The following is a listing of major causes:

   Chemical or toxic compound exposures: Benzene, asbestos, nickel, cadmium, vinyl chloride, benzidine, N-nitrosamines, tobacco or cigarette smoke (contains at least 66 known potential carcinogenic chemicals and toxins), asbestos, and aflatoxin

   Ionizing radiation: Uranium, radon, ultraviolet rays from sunlight, radiation from alpha, beta, gamma, and X-ray-emitting sources

   Pathogens: Human papillomavirus (HPV), EBV or Epstein-Barr virus, hepatitis viruses B and C, Kaposi's sarcoma-associated herpes virus (KSHV), Merkel cell polyomavirus, Schistosoma spp., and other bacteria are being researched as possible agents.

Genetics: A number of specific cancers have been linked to human genes and are as follows: breast, ovarian, colorectal, prostate, skin and melanoma.

(B)Most forms of cancer arise through a Darwinian evolutionary process. The natural selection that ultimately leads to cancer takes place in somatic tissues although it may be triggered by inherited mutations in a small but significant minority. It favors the growth of clones and subclones that are less and less responsive to normal intra- and extracellular growth control mechanisms. The development of molecular biology has led to the identification of many genes that participate in this somatic evolution. They belong to the following groups: Oncogenes, constitutively activated by structural and/or regulatory changes that drive the cell to continuous proliferation; Tumor suppressor genes, that can inhibit the illegitimately activated cell cycle. They contribute to tumor development by loss mutations or permanent down-regulation, e.g. by methylation; Apoptosis inhibitory genes that can contribute to tumor development by raising the apoptotic threshold, and apoptosis promoting genes that can favor the growth of apoptosis prone tumor cells by their loss or inactivation; DNA repair genes whose inactivation can counteract the normal elimination of cells that carry potentially cancer promoting mutations. Inherited mutations in DNA repair genes can lead to familial cancer syndromes. Immortalizing genes that counteract cellular senescence; Angiogenesis promoting genes whose products may stimulate the vascular supply of tumors; Genes whose structural or functional changes may facilitate the escape of tumor cells from immune rejection; The multi-step development of individual tumors can encompass changes in most or all of these genes. They occur independently of each other and without any fixed order or timing. Tumor emancipation from growth control can therefore proceed along various pathways.