Environmental pathology

هذه ملزمة قعدت يومين اكتب فيها

ان شاالله تنال اعجابكم و رضاكم

باذن الله ملزمة كاملة و متكاملة في كل ما يخص الباب

بس هادا مو يعني انو تعتمدو عليها اعتماد كلي
لحد يحط المسؤلية فوق راسي

"اللهم اني بلغت اللهم فاشهد"

ترى لا تفجعكم انو ورق كتيرو لا كلام صغير ترى سههههههللللل و حلو

يمكن العك شوويا في النهاية

للامانة اخر جزيئتين نسختهم من سلايدات الدكتور (حسيتهم) كافية باذن الله

اذا عجبتكم ادعولي

اذا ما اقنعتكم شقوها و ارموهاو لا تدعو عليا

بعد هادي الملزمة في سلايدات الدكتورة نفسها يمكن تعجبكم اكتر من دي

الله يسهل و يعين و يوفق الجميع

و اذا في اخطاء او نقص ارجو ابلاغ ليدراتكم الكرام حتى يوصل الخبر للجميع

☺ **Susiiii** ☺

Introduction:

Many diseases can be caused or influenced by environmental factors.

The term "environment" :encompasses the outdoor, indoor and occupational environments; in each of these the air we breathe, the food and water we consume and direct exposure to toxins are major determinants to health in a population.

The' personal environment': which is greatly influenced by tobacco, alcohol, diet and drugs.

N.B:(factors of the personal environment have greater effect in human health than does the ambient environment).

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Environmental diseases: refers to lesions and disease caused by exposure to chemical or physical agents from both types of environments.

FIRST: INJURY BY PHYSICAL AGENTS

1- Injury by mechanical trauma.

2- Thermal injuries.

3- Electrical injuries.

4-Injury produced by ionizing radiation.

A) MECHANICAL TRAUMA:

The type of injury depends on: -the shape of the colliding object.

-the amount of energy discharged at impact.

- the tissue or organ that received the impact.

Patterns of injuries are:

1) *Abrasions*: a wound produced by scarping or rubbing resulting in removal of superficial layer. (Skin abrasion remove only the epidermal layer. **N.B**: the redness seen in skin is the blood vessels)

2) *Contusions*: (bruises) wound produced by a blunt object and characterized by damage to blood vessels and extravasations of blood into tissue.

(**N.B:** -accompanied by bleeding and formation of hematoma)

3) *Laceration:* tear or disruptive stretching of tissue caused by a blunt object.(The width is larger than the depth.....seen in internal viscera as well), they have intact bridging blood vessels and irregular edges.

4) *Incised wound:* caused by a sharp instrument, bridging blood vessels are severed.(The depth is larger than the width.

5) *Puncture wound:* caused by a long and narrow instrument and could be penetrating if the instrument pierced deep, and perforating when it traverses a tissue to create an exit wound.

Lacerating wounds	**Incised wounds**
Width larger than depth	Depth larger than width
Have intact bridging blood vessels with jagging irregular edges.	The bridging blood vessels are no longer intact.

Penetrating puncture wounds	**Perforating puncture wounds**
Pierces deep to reach the internal viscera.	It's so deep that it passes through the viscera to exit from the other side.

- *Motor vehicle accidents* is the leading cause of death in children an young adultd in most countries.
    - But affects all ages here in the middle east.

Injuries are made by:

- hitting a part of the interior of the vehicle or being hit by something entering the passenger compartment.

-being thrown from the vehicle.

-being trapped in a burning vehicle.

Types of injuries:

- In a wind-shield impact: lacerations, abrasions and contusions of the brain.
- In steering column impact: in the chest fracture of ribs that will lead to pneumothorax(puncture of the lung) in addition to contusion of the heart and laceration of the aorta.
- In a dash-board impact: fractures of the hip, thigh and knee.

Causes of death in mechanical injuries:

- Hemorrhage.
- Head injury.
- Fat embolism.
- Rapture of viscera (depending on the type of viscera).
- Secondary infections.
- Renal shot down: electrolyte imbalance but not immediate death; patients enter shock which is hard to retrieve to normal state.

Hemorrhage: in lacerating wounds: no immediate death.

We need 1/3 of blood to be lost instatntly (not gradually) to enter a hemorrhagic shock.

And loss of 1/2 of the blood to die.

Head injuries:-increase in intracranial pressure

- Compression of vital structure. → DEATH.
- Herniation.
- Skull fractures don't cause death. But if associated with hemorrhage could be fatal.
- Fractures in the temporal and parital area → cutting of middle meningeal artery → epidural hemorrhage.
- Subdural hemorrhage is caused by accelerating or decelerating injuries which will lead to tear of vessels.
- Subarachnoid hemorrhage is due to fracture of skull, laceration of brain.

Cerebral contusions: bruising within the brain.

-by direct impact.

- Usually no skull fracture.

-coup: impact in the same side.

-countercoup: opposite side impact(seen in sphenoid and temporal bones)

e.g.: a person falls on the back of his head and contusions appear in the temporal bones due to accelerating and then a sudden stop.

يعني بالعربي الواحد طاح على قفا راسه تلاقي الرضة امامية
و هاذا للتسارع الذي يلحق الوقوف الفجائي : فيؤدي ان المخ يتحرك لورى بسرعة اثناء السقوط و لما يصقع الانسان في الارض يرتد المخ فيخبط من قدام بسرعة فائقة

Fat embolism: due to fracture of long bones.

Gunshot injury: depends on the firearm used and range of fire.

Entrance wounds : smaller than exit wounds due to the recoiling of elastic tissue of the skin.

Exit wounds: larger than the bullet because the bullet pushes other components from the body with it.

1)contact injury	-Burn:grey-black discoloration. -Abrasions: due to movement of bullet. -Gases may enter subcutaneous and cause a star-shaped laceration. -muzzle print.
2)intermediate range injuries	-Powder tattooing around skin. -irregular wound shape.
3)distant range	-Lacks powder stippling. - The hole is equal to the size of the bullet.

B) THERMAL INJURIES:

- THERMAL BURNS.
- HYPERTHERMIA.
- HYPOTHERMIA.

1) *Burns:*
Outcomes of burns depend on:
- Depth
-percentage of body surface affected.
-internal injury due to fume inhalation.

a)full thickness burns(3rd &4th)	-Total destruction of dermis and epidermis and loss of their regeneration. - They are white or charred, dry and anesthetic.
b)**partial thickness burns(1st&2nd degree)**	-There is the capability for regeneration. -They are pink with blisters and painful.

Pathological effects:

- Burns exceeding 50% of total body surface is fatal wither it was superficial or deep.
- Inhalation of smoky gases may form acids or alkaline in the upper airways and produce inflammatory responses: which may lead to airway obstruction.

- Burned patients are susceptible for bacterial infections which may lead to organ system failure. E.g.: pneumonia, septic shock with acute renal failure or acute respiratory distress syndrome.
- Development of hypermetabolic state which may increase twice as much as normal.

2) *Hyperthermia: prolonged exposure to elevated* ambient temperatures can result in:
-heat cramps: results from loss of electrolytes by sweating. *Cramping of voluntary muscles with vigorous exercise.*
Heat-dissipating mechanisms are able to maintain normal core body temperature.
-**heat exhaustion:** the most common hyperthermia syndrome. Its onset is sudden with prostration and collapse. It's a result of failure of cardiovascular system to compensate for hypovolemia.
After the period of collapse which is brief..... equilibrium is re-established.
-**heat stroke:** associated with high ambient temperature and high humidity. Thermoregulatory mechanisms fail and sweating ceases and core body temperature rises. Necrosis of myocardial muscle can occur. Arrhythmia, disseminated intravascular coagulation and other systemic effects.

3)*hypothermia:* prolonged exposure to low ambient temperature. Associated with high humidity, wet clothing and dilation of superficial blood vessels. It may lead to loss of consciousness followed by bradycardia and atrial fibrillation at lower core temperature.

Local reactions: chilling or freezing cause's injury by two ways:

a)direct effects:	-mediated by physical disruptions within the cell and high salt concentration to the crystallization of the intra-&extracellular water.
b)**indirect effects:**	-results of a circulatory change. -depends on the rate&duration of temperature drop. -slow drop⟶vasoconstriction and increase permeability⟶ edema.

C) RADIATION INJURIES:

Radiation is energy that travels in the form of waves.

Two types of radiation:

1) *Ionizing:* is incapable of displacing the electron from its atom. E.g:UV, infrared light..
2) *Non*-ionizing: has sufficient energy to remove tightly bound electrons.e.g:x-rays and gama rays(with electromagnetic waves with high frequency). Or alpha, beta particles(with high energy neutrons).

The physical properties of radiatants:

- radiation is used in treatment of cancer and diagnostic purposes but its also mutagenic, carcinogenic and teratogenic.
- The radiation is measured by one of the following: roentgen, gray.sievert or curie.

The biological effects of radiation:

- Because ionizing radiation damages DNA, rapidly dividing cells are more vulnerable than quiescent(stable) cells.(tissues with high rate of cell turnover such as bone marrow,gonads,lymphoid tissue and mucosa of the GIT are extremely vulnerable to radiation, and the injury is manifested early after exposure.) while tissues of non-dividing cells don't suffer cell death except in really high doses.
- Vascular damage: narrowing or occlusion of blood vessels leading to impaired healing, fibrosis and chronic ischemic atrophy. This may appear years after exposure. This vascular damage may reach the brain despite its low sensitivity.
- Rate of delivery: although the effect of radiant energy is cumulative, delivery in divided doses may allow cells to repair some of the damages in the intervals.
- Hypoxia: radiant produce free radicals thus indirectly damaging DNA.(hypoxic tissues are resistant to radiation injuries therefore affecting the treatment of neoplasm proliferation.)
- **N.B:**DNA damage and carcinogenesis and fibrosis are common abnormalities that occurs due to prolonged exposure to radiant. High doses may lead to lymphopenia(shrinkage of lymph nodes and spleen and destroying lymphocytes) It may cause anemia by affecting the sensitive hematopoietic cells in the bone marrow; where erythrocytes are radio resistant but anemia can still appear 2-3 weeks after exposure.

Morphology:

Chromosomal changes.

Nuclear swelling, condensation of chromatin and breakage of nuclear membrane.

Apoptosis.

Giant cells with pleomorphic nuclei or more than one nucleus may appear and persist.

Cytoplasm changes: swelling, mitochondrial distortion and degeneration of ER and breakage of membrane.

Tissue susceptibility:

a)**most sensitive**	Lymphoid tissue, bone marrow and GIT
b)**least sensitive**	Bone, brain, muscle and skin

Radiation effect in different tissues:

a)hematopoietic cells	Lymphopenia, thrombocytopenia and bone marrow hypoplasia.
b)vascular	Early thrombosis, late fibrosis and ischemia
c)epidermal	Acute: Erythma chronic: radiodermatitits… potential for epidermal carcinoma
d)GIT	Acute: diarrhea Chronic: adhesion with potential for bowel obstruction.

Cancers caused by radiation: the most common is acute leukemia.

-papillary carcinoma of the thyroid gland.

- osteogenic sarcoma.

Total body irradiation: -atomic bomb, nuclear power plant accident

- Even in low doses will cause devastating effects.
- Effects are mainly on hematopoietic system, GIT and brain.

D) CHEMICAL INJURIES:

- By therapeutic agents
- Alcohol.
- Tobacco.
- Drug abuse.
- Exposure to toxic chemicals.(CO,CCL_4,cyanide,lead,mercuric chloride)

1) *Therapeutic drugs:*
   Adverse drug reactions refer to untoward effects of a drug given in a certain treatment.
   Most frequent drugs that cause these types or reactions are: antibiotics, immunosuppressive and antineoplastic drugs.

   Adverse reaction: could be either:1)predictable:(dose dependent)
   2)unpredictable:idiosyncracy or hypersensitivity.

للأمانة:الجدول الجاي ما اعرف اذا مهم و لا
لأ اخدته من سلايدات الدكتورة فأتأكدو
و صراحة في جدول و معلومات فاضية على
قياسات الاشعة حتلاقوها نهاية الملزمة انشالله
للي مو متطمن. ☺

Adverse effects	Drugs
agranulocytosis, pancytopenia aplastic anemia	chloramphenicolAntineoplastic agents, immunosuppressives
urticaria, expholiative dermatitis	Antineoplastic agents, sulfonamides, hydantoins, some antibiotics, and many other agents
acute tubular necrosis, Tubulointerstitial disease necrosis of papillae,	acute tubular necrosis, Tubulointerstitial disease necrosis of papillae,
lung fibrosis, Asthma	bleomycine, busulphan Salicylates
liver steatosis, cholestasis, necrosis of hepatocytes	tetracycline, estrogens, halothane, chlorpromazine.

Broad spectrum antibiotic side effect:

- o Diarrhea: pseudomembranous colitis
- o Candidacies :mouth, vagina
- o Skin eruption
- o develop of bacterial resistance
- o Other drug specific

• cancer chemotherapeutic:

- o Nausea and vomiting
- o Susceptibility to infection even minor infection can be fatal
- o Tumor lysis syndrome
- o Malignancy: lymphoma

Acetaminophen: a non-prescription analgesic and antipyretic, its mostly conjugated in liver with glucuronide or sulfate.(فجأة فارما

Its accumulation will lead to hepatic necrosis localized in the centrilobular ares of hepatic lobules.

Mechanism of injury:

-covalent binding to hepatic proteins
-depletion of glutathione(GSH).which makes the heptic cells more susceptible to death by ROS.

N.B: the window is between the therapeutic(0.5gm) and the toxic(15-25gm).

The drug is usually safe except if taken in its large dose.

Accedintal overdose is in :children, suicide attempts.

Toxicity begins with: nausea, vomiting, diarrhea and sometimes shock followed by jaundice.

Serious over-dose will lead to liver failure and renal failure.

Treatment: N-acetycysteine.

Aspirin:

-overdose may result could occur accidentally in children or in suicide attempts,

-The major untoward consequences are metabolic with few morphologic.

- at first, respiratory alkalosis develops, followed by metabolic acidosis>>>fatal.

-chronic aspirin toxicity (salicylism) is manifested with: headaches, dizziness, tinnitus (ringing in ear), difficulty in hearing, mental confusion, drowsiness, nausea, vomiting and diarrhea.

-The morphological consequences are: acute erosive gastritis(GIT bleeding and ulceration.

Q: what is analgesic nephropathy?

Mixing of aspirin with acetaminophen, when taken over for several years can cause tubulointerstitial nephritis with renal papillary necrosis.

2) NON-THERAPUETIC DRUGS(drug abuse):

Involves the use of mind-altering substances beyond therapeutic use.

a-cocaine: One of the most addictive of all drugs.

-acute overdose produce seizures, cardiac arrhythmias and respiratory arrest.

Manifestations of cocaine toxicity:

- Cardiovascular affects: tachycardia, hypertension and peripheral vasoconstriction. And myocardial ischemia. And coronary artery vasoconstriction and promotes thrombosis by facilitating platelet aggregation.(cocaine produces an increase demand in O_2 and decreasing the blood flow therefore leading to ischemia and myocardial infarction)
- It can also produce lethal arrhythmias.
- CNS effects: hyperpyrexia and seizures.

- Effects on fetus: decreased blood flow to the placenta resulting in fetal hypoxia and spontaneous abortion.(neurological development maybe impaired in a chronic drug user mom)
- Chronic use may cause:1)perforation of the nasal septum
  2)decrease in lung diffusing capacity.
  3)development of dilated cardiomyopathy.

b-heroin: an addictive opium which is closely related to morphine.

-more harmful than cocaine.

-effects are euphoria, hallucinations, somnolence and sedation.

The physical effects are related to :1)pharmacological action of the drug

2) reactions to the agents or contaminants.

3) hypersensitivity reaction to the drug.

4) diseases contracted incident to the use of needles.

The adverse effects of heroin:

1) *Sudden death*. Related to overdose. Or tolerance of drug is lost.

Mechanism of death is by respiratory depression, arrhythmias, cardiac arrest and pulmonary edema.

1) *Pulmonary problems.* Include edema, septic embolism, lung abscess....
2) *Infections.* Most commonly sites affected are : skin, heart valves, liver and lungs.(viral infections, infections caused by staphylococcus aureus)
3) S*kin.* Formation of cutaneous lesions, abscess, celluitis and ulcerations.
4) *Renal problems.* Formation of amyloidosis and focal glomerulosclerosis; which both induce proteinuria and nephritic syndrome.

E) TOBACCO SMOKING:

- The most common exogenous cause of cancer.
- 1st hand smokes (people who smoke). 2nd hand smokes (people who are near smokers).
- It's the most preventable cause of human death(80% are non-smoker live to the age of 70 while only 50% of smokers survive to that age).
- Cessation of smoking greatly reduces the risk of death from lung cancer.
- Nicotine is addictive.

- *The casual mechanism* is relative to: increased platelet aggregation, decreased myocardial oxygen supply (due to hypoxia due to lung disease), and an increase demand in oxygen and finally a decreased threshold for ventricular fibrillation.

The adverse effect of:

1) Chronic bronchitis and emphysema..
2) Cancer of oral cavity and larynx and esophagus and pancreas and bladder.
3) Cancer of lung
4) Myocardial infarction which follows an atherosclerosis.
5) Peptic ulcer.
6) Systemic atherosclerosis.
7) Maternal smoking leads to increase risk of spontaneous abortion and preterm birth.

F) ALCOHOL:

- It has a mood altering process.
- It may cause drowsiness, coma and respiratory distress.
  Its metabolized in the liver by three enzymes:(alcohol dehyrdrogenase in the cytosol, cytochromes p-450 in microsomes, catalase in perixosomes.)
  Alcohol ⟶ acetaldehyde.
  The main enzyme in alcohol metabolism is: alcohol dehydrogenase.

- Alcohol oxidation by ADH causes a decrease in NAD^+ and increase in NADH.
- NAD^+ is required for fat metabolism; its deficiency will lead to fat accumulation. NAD^+ also needed in conversion of lactate to pyruvate. And an increase in $NADH/NAD^+$ will result in metabolic acidosis.
- Acetaldehyde is toxic and may cause hyperventilation, tachycardia and flushing.
- Metabolism of alcohol by cytochromes will produce ROS and cause lipid peroxidation of cell membrane.
- Induce the release of endotoxins(lipopolysaccharide) which will lead to the release of mediators of inflammation and therefore causing cell injury.

The adverse effects of ethanol:

1) *Acute alcoholism* affects mainly CNS but may induce reversible hepatic and gastric changes.
2) *Chronic alcoholism* does morphological changes in all organs but mainly stomach and liver.

The liver: - main site of chronic injury

- Fatty change (hepatic steatosis).
- Alcoholic hepatitis and cirrhosis(associated with hypertension nad increased risk of hepatocellular carcinoma)

GIT tract: - massive bleeding

- Gastric ulcer or esophageal varcies(associated with cirrhosis)

Thiamine deficeny:lesion resulting from it (peripheral neuropathy, wernickekorsakoff syndrome, cerebral atrophy, cerebeller degeneration, optic neuropathy.

On CVS: congestive cardiomyopathy (alcoholic cardiomyopathy)

On pancreas: acute and chronic pancreatitis.

on fetus: fetal alcohol syndrome; consists of microcephaly, growth retardation, facial abnormalities in new born and mental retardation in older children.

Carcinogenic: to esophagus, oral cavity, liver and breasts (in females).

On nutrition: ethanol leads to malnutrition and VIT B deficiency.

G) TOXIC CHEMICAL SUBSTANCES:

- ***Chloroform and carbon tetrachloride***
  - Cleaning fluids and solvents
  - Exert anesthetic (depressant) effects on the CNS, and on the heart and blood vessels
  - Hepatotoxins: acute hepatic necrosis (centrilobular fatty changes or necrosis), fatty liver, and liver failure
  - Renal tubular necrosis :Oliguria
  - Toxicity may persist for long periods

- ***CO (carbon monoxide):***

- Nonirritating, colorless, tasteless, odorless gas.
-it is an air pollutant but also cause of accidental and sudden death.
- in a small polluted garage …..Lethal coma occurs after 5min.
-CO is a systemic asphxiant that kills by inducing CNS depression.
- CO binds to hemoglobin instead of O_2 and produce hypoxia.

Morphology of CO poisoning:

In acute poising its characterized by **generalized cherry color of the skin and mucos membrane.**

Depending on the onset of poisoning if death occurs then no morphological changes.

With longer survival: the brain is edamateous , with systemic hypoxia.

If exposure was not prolonged, then complete recovery is possible **but** sometimes with these complications: impaired memory, vision, hearing and speech.

- ***Cyanide***
  - Very toxic kills in minutes
  - classic murderer's tool
  - smell of bitter almonds
  - Cyanide blocks cellular respiration by binding to mitochondrial cytochrome oxidase,
  - Generalized petechial hemorrhage

- ***Lead:***

  -lead exposure is through contaminated air and food.

  - absorbed lead is taken by bone and developing teeth(competing with Ca^{+2} and binds with phosphate and its t1/2=20-30 years)

  -5-10% remains in blood and the reminder distributed through tissues.

  -excess lead cause neurological effects in both adults and children (peripheral neuropathy in adults and central effects on children).

  - chronic exposure to lead in children include: low IQ, behavioral problems(hyperactivity) and poor organizational skills.

  -lead peripheral neuropathies in adults are usually reversible.

  - impaired remodeling of calcified cartilage in children which is detected radio graphically as:"lead lines".

  - lead has affinity for SH and interrupt the heme synthesis; leading to microcytic anemia (hypochromic anemia).

  -it also increases the erythrocyte fragility leading to hemolytic anemia.

  Morphology:

  Major target sites: CNS, blood, GIT and kidney.

  Blood changes: *disrupting normal heme synthesis* → *increase levels of zinc-protoprophrin* → *microcytic anemia or basophilic stippling or erythrocyte.*

  brain damage: in children *impairments, blindness, low IQ, and in sever cases psychoses and coma.*

  In adults *peripheral demylinating neuropathy paralysis (wrist drop or foot drop)*

GIT:*lead colic*
Kidneys:*proximal tubular damage, interstitial fibrosis, renal failure and gout.*

- ***Mercury:*** ***-*** *poisoning with mercury is associated with tremors, gingivitis and bizarre behavior such as "mad hatter" of Alice of wonderland*" **(**فاكرين ابو طاقية حق حفلات الشاهي" اليس في بلاد العجائب"؟؟هوا دا المثال)

-*main sources are: contaminated fish.*
-"Minamata disease": include: cerebral palsy, deafness, blindness, and CNS defects in children exposed in uterus.
-developing brain is extremely sensitive to methyl-mercury.

H) MALNUTRITION:

Main causes: poverty, ignorance, chronic alcoholism, acute and chronic illnesses, self-imposed dietry restriction……

1) ***protein-energy malnutrition(PEM):***

-in severe cases its lethal.

-in the body there are two protein compartments: 1) somatic: in skeletal muscles. 2)visceral: in liver.

-two syndromes:"marasmus" : where somatic compartment is severely affected. And "kwashiorkor "where visceral compartment is severely affected.

- common victims are children.

- A child considered to have marasmus have fallen weight to 60% of normal; suffers from growth retardation and loss of muscle (due to catabolism of somatic compartment). And visceral compartment are depleted **partially** therefore normal levels of serum albumin. Its accompanied by anemia and immune deficiency.

- a child with kwashiorkor syndrome have fallen weight to about 60-80% than normal and is due to protein deprivation. (Due to inability of protein absorbance or chronic protein loss). There is a sever loss from the viscera compartment which result in (hyperalbuinemia) which gives rise to generalized or dependent edema. The disease is characterized by loss of appetite. Skin lesion, both hyper-and hypo-pigmentation, fatty liver and defects in immune system.

Secondary PEM: *is in the form of cachexia, commonly developed in patients with cancer.*

Morphology:

1)growth failure. 2)peripheral edema in kwashiorkor.

3)loss of body fat and ms atrophy in marasmus.

- **Fatty liver in kwashiorkor.**
- **Hypoplastic bone marrow in both (marasmus and kwashiorkor) due to little red cell precursors>>>microcytic anemia but if associated with folate deficiency>>>micro-macrocytic anemia.**
- **The brain of infants with malnutritioned mothers show cerebral atrophy reduced number of neurons and impaired mylenization of white matter.**
- **Lymphoid and thymus atrophy in kwashiorkor more than marasmus.**
- **Anatomic alterations due to repeated infections.**
- **Vitamins and iodine deficiency.**

- **2) Eating disorder: *Anorexia Nervosa and Bulimia***
  - *Anorexia nervosa* is self-induced starvation, resulting in marked weight loss;
  - *Bulimia* is a condition in which the patient binges on food and then induces vomiting.
  - Bulimia is more common than anorexia nervosa and generally has a better prognosis.
  - It is estimated to occur in 1% to 2% of women and 0.1% of men, with an average onset at 20 years of age.
  - These eating disorders occur primarily in previously healthy young women who have developed an obsession with attaining thinness.
  - Clinical findings in *Anorexia nervosa*
    - Secondary amenorrhea: Decreased gonadotropin-releasing hormone Caused by loss of body fat and weight produces hypoestroginism.
    - Osteoporosis :Caused by hypoestroginism
    - decreased thyroid hormone release, include cold intolerance, bradycardia, constipation,
    - dehydration and electrolyte abnormalities are frequently present.
    - Increased lanugo (fine, downy hair)
    - Increased hormones associated with stress (e.g., cortisol, growth hormone)
  - Clinical findings in Bulimia due to continual induced vomiting and chronic use of laxatives and diuretics.
    - electrolyte imbalances (hypokalemia), which predispose the patient to cardiac arrhythmias;
    - pulmonary aspiration of gastric contents;

- Esophageal and stomach rupture.
- Acid injury to tooth enamel

2)*Obesity:*

- Obesity is a global epidemic resulting from sedentary lifestyles, improved socioeconomic conditions, and availability of processed, high calorie foods and soft drinks in industrialized societies.
- Obesity is a disorder of energy balance. When food-derived energy chronically exceeds energy expenditure.
- Obesity is a disorder with a multifactorial complex etiology
    - environmental,
    - Genetic
    - Psychological
- Obesity:Body mass index (BMI) ≥ 30kg/m^2 (normal: 18.5-24.9kg/m^2) :BMI = weight (kg)/height (m^2)
- Other factors than body weight
    - Excess fat in the waist and flanks is more important than an excess in the thighs and buttocks.
    - Excess visceral fat in the abdominal cavity has greater significance than excess subcutaneous fat.
- **Pathogenesis**
    - Genetic factors
        - Examples-defects in the leptin gene, syndrome X (obesity, hypertension, diabetes)
    - Acquired causes
        - Endocrine disorders-hypothyroidism, Cushing syndrome
        - Hypothalamic lesions, menopause
- Leptin : It is now established that adipocytes communicate with the hypothalamic centers that control appetite and energy expenditure by secreting leptin (a member of the cytokine family).
    - Hormone is secreted by adipose tissue that maintains energy balance.
    - Leptin increases when adipose stores are adequate.
        - Decreases food intake
        - Increases energy expenditure (stimulates β-oxidation of fatty acids)
    - Leptin decreases when adipose stores are inadequate.
        - Increases food intake
        - Decreases energy expenditure (inhibits β-oxidation of fatty acids)
- Behavioral and dietary changes are the initial therapeutic strategies; weight-loss drugs should be used with caution
- Associated disorders
    - Atherosclerosis
    - Hypertension
    - Diabetes-II
    - Gallbladder disease
    - Some Ca-Colon, breast