**Course Title: General Pathology (MLT 2nd Sec A and B)**

**Mid term assignment**

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**Note:**

* **Write in your own words, do not copy paste.**
* **Use only MS word to attempt questions.**

1. Define the following terms with 2 physiological and pathological examples each.
2. Atrophy

Ans: Q.1:: Atropy:: decrease in size or wasting away of a body part or tissue atrophy of muscles also : arrested development or loss of a part or organ incidental to the normal development or life of an animal or plant.

Examples of atrophy as part of normal development include shrinking and the involution of the thymus in early childhood, and the tonsils in adolescence.

Pathological Example..Pathologic atrophy of muscles can occur with diseases of the motor nerves or diseases of the muscle tissue itself. Examples of atrophying nerve diseases include Charcot-Marie-Tooth disease, poliomyelitis, amyotrophic lateral sclerosis (ALS or Lou Gehrig's disease), and Guillain–Barré syndrome.

B: Hypertrophy

B.. Hyperthropy:Hypertrophy from Greek hyper "excess" and thropy "nourishment") is the increase in the volume of an organ or tissue due to the enlargement of its component cells. It is distinguished from hyperplasia, in which the cells remain approximately the same size but increase in number.Although hypertrophy and hyperplasia are two distinct processes, they frequently occur together, such as in the case of the hormonally-induced proliferation and enlargement of the cells of the uterus during pregnancy..

Example. .. An example of physiologic hypertrophy is in skeletal muscle with sustained weight bearing exercise. An example of pathologic hypertrophy is in cardiac muscle as a result of hypertension.

C:Hyperplasia C::Hyperplasia (from ancient Greek huper, "over" + plasis, "formation"), or hypergenesis, is an increase in the amount of organic tissue that results from cell proliferation.It may lead to the gross enlargement of an organ, and the term is sometimes confused with benign neoplasia or benign tumor.Hyperplasia is a common preneoplastic response to stimulus.Microscopically, cells resemble normal cells but are increased in numbers. Sometimes cells may also be increased in size (hypertrophy).Hyperplasia is different from hypertrophy in that the adaptive cell change in hypertrophy is an increase in the size of cells, whereas hyperplasia involves an increase in the number of cells.

Example::Physiologic hyperplasia: Occurs due to a normal stressor. For example, increase in the size of the breasts during pregnancy, increase in thickness of endometrium during menstrual cycle, and liver growth after partial resection.

Pathologic hyperplasia: Occurs due to an abnormal stressor.

D: MetaplasiaD: Metaplasia (Greek: "change in form") is the transformation of one differentiated cell type to another differentiated cell type. The change from one type of cell to another may be part of a normal maturation process, or caused by some sort of abnormal stimulus. In simplistic terms, it is as if the original cells are not robust enough to withstand their environment, so they transform into another cell type better suited to their environment. If the stimulus causing metaplasia is removed or ceases, tissues return to their normal pattern of differentiation. Metaplasia is not synonymous with dysplasia, and is not considered to be an actual cancer.It is also contrasted with heteroplasia, which is the spontaneous abnormal growth of cytologic and histologic elements. Today, metaplastic changes are usually considered to be an early phase of carcinogenesis, specifically for those with a history of cancers or who are known to be susceptible to carcinogenic changes. Metaplastic change is often viewed as a premalignant condition that requires immediate intervention, either surgical or medical, because metaplasia is associated with cancer.

Example: An example of physiologic metaplasia is the squamous metaplasia that occurs in the uterine cervix during the menstrual cycle as the squamocolumnar junction migrates across the transformation zone.

Pathological Example::

One example of pathological irritation is cigarette smoke, which causes the mucus-secreting ciliated pseudostratified columnar respiratory epithelial cells that line the airways to be replaced by stratified squamous epithelium, or a stone in the bile duct that causes the replacement of the secretory columnar epithelium

2:How does the calcium ions influx affects the cell?write it in your own words.

Ans: Effect of calcium ion influx in the cell.

Intracellular calcium regulates a number of membrane functions in the erythrocyte, including control of shape, membrane lipid composition and cation permeability. Measurement of total red cell calcium has yielded values between 5 and 15 nmol/ml cells, and these low values in part reflect the absence of Ca2+ -containing organelles. Most intracellular Ca2+ is bound and the low cell ionized Ca2+ concentration (approximately 0.2 microM) is maintained by a combination of low membrane permeability and a powerful Ca2+ -pump. This pump has been identified with a (Ca2+ + Mg2+)-stimulated ATPase, and both Ca2+ transport and ATP splitting are stimulated by calmodulin, a low molecular weight protein which binds Ca2+ avidly and activates many Ca2+ -dependent enzymes. Both high and low affinity kinetics for Ca2+ pumping have been demonstrated, depending on the extent of binding of calmodulin to the pump. A stoichiometry of either 1 or 2 Ca2+ ions pumped per ATP molecule split has been shown, and the value may vary with the level of intracellular Ca2+. Phenothiazines, such as chlorpromazine inhibit the Ca2+ -pump by antagonizing the increment in activity produced by calmodulin. The passive inward leak of Ca2+ into erythrocytes can be quantitated by 45Ca2+ uptake into red cells whose Ca2+ -pump has been inhibited. Estimates of the Ca2+ permeability, based on unidirectional influx, yield values many orders of magnitude lower than for nucleated cells. Influx of Ca2+ into human erythrocytes occurs by a facilitated diffusion process, which can be inhibited by phenothiazines and the cinchona alkaloids. Calcium affects many membrane functions including cation permeability, lipid composition and some cytoskeletal interactions which may determine cell shape. Any rise in intracellular Ca2+ activates a specific K+ channel which normally makes little contribution to K+ fluxes. Kinetic studies of this process demonstrate either high or low affinity Ca2+ -activation of K+ efflux, with low affinity of the channel to Ca2+ being the probable state in vivo. Propranolol is the best known activator of Ca2+ -stimulated K+ efflux, although the mechanism of stimulation is unclear. Like other tissues, red cells possess a Ca2+ -activated phosphoinositol phosphodiesterase. Although it has been suggested that the echinocytic shape change induced by Ca2+ is due to the hydrolysis of polyphosphoinositides, it seems more likely that this shape change results from an effect of Ca2+ on the macromolecular interactions of the cytoskeleton. Abnormal Ca2+ permeability may contribute to red cell destruction in a variety of diseases. For example, in sickle cell anemia a large Ca2+ influx occurs when cells are sickled under deoxy conditions, and moreover, the ability of the Ca2+ -pump to extrude the increment of cell Ca2+ is impaired. Thus, red cell Ca2+ is increased 3-7-fold above normal and this may contribute to the short survival of sickle red cells...

3:What is free radical?What is the effect of Reactive Oxygen Specie(ROS) on the cell?

Ans: 3. 3..Free Radical::

In chemistry, a radical is an atom, molecule, or ion that has an unpaired valence electron.With some exceptions, these unpaired electrons make radicals highly chemically reactive. Many radicals spontaneously dimerize. Most organic radicals have short lifetimes.

Effect of Reactive oxygen species on cell::

According to the free radical theory of aging, oxidative damage initiated by reactive oxygen species is a major contributor to the functional decline that is characteristic of aging. While studies in invertebrate models indicate that animals genetically engineered to lack specific antioxidant enzymes (such as SOD), in general, show a shortened lifespan (as one would expect from the theory), the converse manipulation, increasing the levels of antioxidant enzymes, has yielded inconsistent effects on lifespan (though some studies in Drosophila do show that lifespan can be increased by the overexpression of MnSOD or glutathione biosynthesizing enzymes). Also contrary to this theory, deletion of mitochondrial SOD2 can extend lifespan in Caenorhabditis elegans.

In mice, the story is somewhat similar. Deleting antioxidant enzymes, in general, yields shorter lifespan, though overexpression studies have not (with some recent exceptions) consistently extended lifespan. Study of a rat model of premature aging found increased oxidative stress, reduced antioxidant enzyme activity and substantially greater DNA damage in the brain neocortex and hippocampus of the prematurely aged rats than in normally aging control rats.[31] The DNA damage 8-OHdG is a product of ROS interaction with DNA. Numerous studies have shown that 8-OHdG increases in different mammalian organs with age..

Q:4 ANS: 4:

Apoptosis, or programmed cell death, is a form of cell death that is generally triggered by normal, healthy processes in the body.

Necrosis is the premature death of cells and living tissue. ... Caused by factors external to the cell or tissue, such as infection, toxins, or trauma.

Differences::

Apoptosis:

Introduction Apoptosis, or programmed cell death, is a form of cell death that is generally triggered by normal, healthy processes in the body.

Necrosis is the premature death of cells and living tissue. Though necrosis is being researched as a possible form of programmed cell death, it is considered an "unprogrammed" cell death process at this time.

Natural Caused by factors external to the cell or tissue, such as infection, toxins, or trauma.

Effects Usually beneficial. Only abnormal when cellular processes that keep the body in balance cause too many cell deaths or too few. Always detrimental

Process Membrane blebbing, shrinkage of cell, nuclear collapse (nuclear fragmentation, chromatin condensation, chromosomal DNA fragmentation), apoptopic body formation. Then, engulf by white blood cells. Membrane disruption, respiratory poisons and hypoxia which cause ATP depletion, metabolic collapse, cell swelling and rupture leading to inflammation.

Symptoms Usually no noticeable symptoms related to the process. Inflammation, decreasing blood flow at affected site, tissue death (gangrene).

Causes Self-generated signals in a cell. Generally natural part of life, the continuation of the cellular cycle initiated by mitosis. Bacterial or fungal infections, denatured proteins that impede circulation, fungal and mycobacterial infections, pancreatitis, deposits of antigens and antibodies combined with fibrin.

Medical Treatment Very rarely needs treatment. Always requires medical treatment. Untreated necrosis is dangerous and can lead to death

Q:55::Air Embolism::

An air embolism, also known as a gas embolism, is a blood vessel blockage caused by one or more bubbles of air or other gas in the circulatory system. Air embolisms may also occur in the xylem of vascular plants, especially when suffering from water stress. Air can be introduced into the circulation during surgical procedures, lung over-expansion injury, decompression, and a few other causes.

Explanation::

Divers can suffer from arterial gas embolisms as a consequence of lung over-expansion injury. Breathing gas introduced into the venous system of the lungs due to pulmonary barotrauma will not be trapped in the alveolar capillaries, and will consequently be circulated to the rest of the body through the systemic arteries, with a high risk of embolism. Inert gas bubbles arising from decompression are generally formed in the venous side of the systemic circulation, where inert gas concentrations are highest, these bubbles are generally trapped in the capillaries of the lungs where they will usually be eliminated without causing symptoms. If they are shunted to the systemic circulation through a patent foramen ovale they can travel to and lodge in the brain where they can cause stroke, the coronary capillaries where they can cause myocardial ischaemia or other tissues, where the consequences are usually less critical. The first aid treatment is to administer oxygen at the highest practicable concentration, treat for shock and transport to a hospital where therapeutic recompression and hyperbaric oxygen therapy are the definitive treatment..

Sign and Symptoms:

Symptoms include:

Hypotension

Shortness of breath

In divers Edit

Symptoms of arterial gas embolism include

Loss of consciousness

Cessation of breathing

Vertigo

Convulsions

Tremors

Loss of coordination

Loss of control of bodily functions

Numbness

Paralysis

Extreme fatigue

Weakness in the extremities

Areas of abnormal sensation