Animal Health Care Service

Level-IV

Based on May 2018, Version 3 Occupational standard

Module Title: Identifying Pathological Lesions, Abnormalities and Conditions

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LG #47	LO #1- Describe pathological terminologies

Instruction sheet
This learning guide is developed to provide you the necessary information regarding the following content coverage and topics: • Describing concepts of pathology. • Identifying an associated technical pathological terminology clearly. This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, you will be able to:- • Describing concepts of pathology. • Identifying an associated technical pathological terminology clearly.
Learning Instructions:
1. Read the specific objectives of this Learning Guide. 2. Follow the instructions described below. 3. Read the information written in the “Information Sheets”. Try to understand what are being discussed. Ask your trainer for assistance if you have hard time understanding them. 4. Accomplish the “Self-checks” which are placed following all information sheets. 5. Ask from your trainer the key to correction (key answers) or you can request your trainer to correct your work. (You are to get the key answer only after you finished answering the Self-checks).

Information Sheet 1- Describing the concepts of pathology

1. Definition of pathology

Pathology is the study of the way diseases and illnesses develop or the study of the causes and effects of disease or injury. The word pathology came from the Latin words **"patho" & "logy".** 'Patho' means disease and 'logy' means study, therefore pathology is a scientific study of disease. It investigates the essential nature of disease and is usually summarized as the study of the functional and morphological changes in the tissue and fluids of the body during disease. Disease is a state in which some part of the body is not functioning properly or it is an altered (deranged) normal physiological activity of the body. Pathology gives explanations of a disease by studying the following four aspects of the disease. This means

- the study of the essential nature of diseases and especially of the structural and functional changes produced by them studied animal pathology
- something abnormal:
  - ✓ The structural and functional deviations from the normal that constitute disease or characterize a particular disease the pathology of pneumonia.
  - ✓ Deviation from propriety or from an assumed normal state of something nonliving or nonmaterial.

Notes: Health, illness and disease differences are:

- **Health** is a state of an individual living in complete harmony, with his environment; surroundings.
- **Disease** is a condition in which an individual shows an anatomical, chemical or physiological deviation from the normal. (Discomfort with environment & body).
- **Illness** is the reaction of an individual to disease in the form of illness.

Self-check 1	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Choose the best answer

1. What is pathology (4 point)?

2. Differentiate health and illness (2pts).

Note: **Satisfactory rating – above 4 points** **Unsatisfactory - below 4 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 2- Identifying pathological terminology

2.1 Branches of pathology

A. **General pathology:** concerns with basic alterations of tissues as a result of disease. E.g. Fatty changes, Thrombosis, Amyloidosis, Embolism, Necrosis.

B. **Systemic pathology**: deals with alterations in tissues/ organs of a particular system. E.g. integument, muscle, haematopoietic system, nervous system and organs of special sense (eye and ear), circulatory, respiratory, digestive, urinary and reproductive systems.

C. **Specific Pathology**: is the application of the basic alterations learned in general pathology to various specific diseases. It involves whole body or a part of body. e.g. Tuberculosis, Rinderpest.

D. **Experimental Pathology**: concerns with the production of lesion through experimental methods. E.g. Rotavirus ~ calves ~ enteritis/ diarrhoea in calves.

E. **Clinical Patholog**y includes certain laboratory methods which help in making the diagnosis using animal excretions/ secretions/ blood/ skin scrapings/ biopsy etc. e.g. Urine examination, Blood examination.

F. **Post-mortem Pathology:** is examination of an animal after death. Also known as Necropsy or Autopsy. It forms the base for study of pathology.

G. **Microscopic Pathology**: deals with examination of cells/ tissues/ organs using microscope. It is also known as histopathology / cellular pathology. E.g. Microscopy, Electron microscopy.

H. **Humoral Pathology:** is the study of alterations in fluids like antibodies in serum.

I. **Chemical Pathology**: is the study of chemical alterations of body fluids/ tissues. E.g. Enzymes in tissue.

J. **Physiological Pathology**: deals with alteration in the functions of organ/ system. It is also known as Pathophysiology. e.g. Indigestion, Diarrhoea, Abortion.

K. **Nutritional Pathology**: is the study of diseases occurred due to deficiency or excess of nutrients. e.g. Vit.-A deficiency induced nutritional roup, rickets due to calcium deficiency.

L. **Comparative Pathology:** is the study of diseases of animals with a comparative study in human beings and other animals. E.g. Zoonotic diseases such as Tuberculosis.

M. ***Oncology*** is the study of cancer/ tumor/ neoplasms.

N. I**mmunopathology** deals with the study of diseases mediated by immune reactions. It includes Immunodeficiency diseases, autoimmunity and hypersensitivity reactions.

O. **Cytopathology** is the study of cells shed off from the lesions for diagnosis.

P. **Anatomical pathology**: is a medical specialty that is concerned with the diagnosis of disease based on the macroscopic, microscopic, biochemical, immunologic and molecular examination of organs and tissues.

Q. **Forensic Pathology** includes careful examination and recording of pathological lesions in case of veterolegal cases.

R. **Toxopathology or Toxic Pathology** deals with the study of tissue/ organ alterations due to toxins/ poisons.

2.2 Necessary pathological terminology

- **Etiology** is the study of causation of disease.
- **Homeostasis** is the mechanism by which body keeps equilibrium between health and disease. E.g. Adaptation to an altered environment.
- **Diagnosis** is an art of precisely knowing the cause of a particular disease (Dia= thorough, gnosis= knowledge).
- **Symptoms:** any subjective evidence of disease of animal characterized by an indication of altered bodily or mental state as told by owner (Complaints of the patients).
- **Signs:** indication of the existence of something, any objective evidence of disease, perceptible to veterinarian (Observations of the clinicians).
- **Syndrome**: a combination of symptoms caused by altered physiological process.
- **Lesion** is a pathological alteration in structure or function that can be detectable.

- **Pathogenesis** is the progressive development of a disease process. It starts with the entry of cause in body and ends either with recovery or death. It is the mechanism by which the lesions are produced in body.
- **Incubation period** is the time elapses between the action of a cause and manifestation of disease.
- **Course of disease** is the duration for which the disease process remains till fate either in the form of recovery or death.
- **Prognosis** is an estimate by a clinician of probable severity/ outcome of disease.
- **Morbidity rate** is the percentage/ proportions of affected animals out of total population in a particular disease outbreak. E.g. out of 100 animals, 20 are suffering from diarrhoea. The morbidity rate of diarrhoea will be 20%.
- **Mortality rate** is the percentage/ proportions of animals out of total population died due to disease in a particular disease outbreak. e.g. In a population of 100 animals, 20 falls sick and 5 died. The mortality rate will be 5%.
- **Case fatality rate** is the percentage/ proportions of animals died among the affected animals. In a population of 100 animals, 20 falls sick and 5 died. The case fatality rate will be 25%.
- **Biopsy** is the examination of tissues received from living animals.
- **Infection** is the invasion of the tissues of the body by pathogenic organisms resulting in the development of a disease process.
- I**nfestation** is the superficial attack of any parasite/ organisms on the surface of body.
- **Pathogenicity** is the capability of an organism for producing a disease.
- **Virulence** is the degree of invasiveness of pathogenic organism.
- **Morphologic changes** refer to the structural alterations in cells or tissues that occur following the pathogenetic mechanisms. The structural changes in the organ can be seen with the naked eye or they may only be seen under the microscope. Those changes that can be seen with the naked eye are called gross morphologic changes & those that are seen under the microscope are called microscopic changes. Some of the morphological changes are listed as follow:

- ✓ **Contusions or bruises** arise from rupture of blood vessel with disintegration of extravassated blood.
- ✓ **Abrasions** are circumscribed areas where epithelium has been removed by injury and it may indicate the direction of force.
- ✓ **Erosions:** a partial loss of surface epithelium on skin or mucosal surface.
- ✓ **Laceration:** severance of tissue by excessive stretching and is common over bony surfaces or are produced by cut through a dull instrument.
- ✓ **Compression injury** is produced as a result of force applied slowly e.g. during parturition.
- ✓ **Hyperthermia** means increased body temperature due to high environmental temperature e.g. Pets in hot environment without water.
- ✓ **Hyperpnoea:** increased respiration
- ✓ **Tachycardia:** rapid heart beat
- ✓ **Eupnea:** true respiration
- ✓ **Eucardia:** true heart beat
- ✓ **Degeneration:** loss of muscle relaxation or decrease work load
- ✓ **Hypothermia:** decreased body temperature
- ✓ **Atrophy:** decrease in muscle mass
- ✓ **Hypertrophy:** increase in muscle mass

2.2 Etiology

Etiology of a disease means the cause of the disease. If the cause of a disease is known it is called primary etiology. If the cause of the disease is unknown it is called idiopathic. Knowledge or discovery of the primary cause remains the backbone on which a diagnosis can be made, a disease understood, & a treatment developed. There are two major classes of etiologic factors: genetic and acquired (infectious, nutritional, chemical, physical, etc.). Detailed discussion will be given in subsequent topics. The etiology is followed by pathogenesis.

2.3 Pathogenesis

Pathogenesis means the mechanism through which the cause operates to produce the pathological and clinical manifestations. The pathogenetic mechanisms could take place in the latent or incubation period. Pathogenesis leads to morphologic changes.

2.3. Morphologic changes

The morphologic changes refer to the structural alterations in cells or tissues that occur following the pathogenetic mechanisms. The structural changes in the organ can be seen with the naked eye or they may only be seen under the microscope. Those changes that can be seen with the naked eye are called gross morphologic changes & those that are seen under the microscope are called microscopic changes. Both the gross & the microscopic morphologic changes may only be seen in that disease, i.e. they may be specific to that disease. Therefore, such morphologic changes can be used by the pathologist to identify (i.e. to diagnose) the disease. In addition, the morphologic changes will lead to functional alteration & to the clinical signs & symptoms of the disease.

2.2. Functional derangements and clinical significance

The morphologic changes in the organ influence the normal function of the organ. By doing so, they determine the clinical features (symptoms and signs), course, and prognosis of the disease. In summary, pathology studies:-

Etiology ➡Pathogenesis ➡ Morphologic changes ➡Clinical features & Prognosis of all diseases.

Understanding of the above core aspects of disease (i.e. understanding pathology) will help one to understand how the clinical features of different diseases occur & how their treatments work. This understanding will, in turn, enable health care workers to handle & help their patients in a better & scientific way. It is for these reasons that the health

science student should study pathology. In addition, the pathologist can use the morphologic changes seen in diseases to diagnose different diseases. There are different diagnostic modalities used in pathology. Most of these diagnostic techniques are based on morphologic changes.

2.3. Pathophysiology: is the study of the disordered physiological processes that cause, result from, or are otherwise associated with a disease or injury. It focused mainly on:

- Physical and clinical manifestations
- Laboratory findings
- Diagnostic tests
- Treatment strategies

Self-Check – 2	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Define and explain the difference between health, injury and illness in relation to their pathological concepts (4pts).

2. What is pathophysiology (2pts).

Note: **Satisfactory rating – above 6 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

LG #48	LO #2- Identify cellular injury

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following **content coverage** and topics:

- Describing lesions; structural and functional changes of cells in response to injurious or harmful stimuli.
- Identifying the principal types of injuries that occur from harmful stimuli to the cells.
- Identifying the changes indicative of cell damage including *degeneration*, *necrosis* and *extracellular accumulations* of substances.

This guide will also assist you to attain the learning outcomes stated in the cover page.

Specifically, upon completion of this learning guide, **you will be able to**:

- Describe lesions; structural and functional changes of cells in response to injurious or harmful stimuli.
- Identify the principal types of injuries that occur from harmful stimuli to the cells.
- Identify the changes indicative of cell damage including degeneration, necrosis and extracellular accumulations of substances.

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the “Information Sheets”. Try to understand what are being discussed. Ask your trainer for assistance if you have hard time understanding them.
4. Accomplish the “Self-checks” which are placed following all information sheets.
5. Ask from your trainer the key to correction (key answers) or you can request your trainer to correct your work. (You are to get the key answer only after you finished answering the Self-checks).

Information Sheet 1- Describe lesions; structural and functional changes of cells in response to injurious or harmful stimuli.

2.1 Definition of lesions

Lesion, in pathology, a structural or biochemical change in an organ or tissue produced by disease processes or wound. Lesion a region in an organ or tissue which has suffered damage through injury or disease, such as a wound, ulcer, abscess, or tumors. The most common causes of skin lesions are injury, aging, infectious diseases, allergies, and small infections of the skin or hair follicles. Lesions may be classified as:

- Anatomic (evident to the unaided senses),
- Histologic (evident only under a microscope), or
- Biochemical (evident only by chemical analysis).

2.2 Types of pathological lesions

I. Primary lesions

Primary skins lesions are either present from birth or develop over your lifetime. They are associated with a specific cause or can be a reaction to either internal or external environments. They tend to be divided into three types of groups:

- Skin lesions formed by fluid within the skin layers, such as vesicles or pustules.
- Skin lesions that are solid, palpable masses, such as nodules or tumors.
- Flat, non-palpable skin lesions like patches and macules

Types of primary lesion include:

- **Bulla:** A vesicle that is greater than 0.5 centimeters (cm) or 1/5 of an inch and filled with fluid
- **Cyst:** A raised, circumscribed area of the skin, filled with fluid or semi-solid fluid
- **Macule:** A non-palpable, flat lesion that is different in color, and less than 0.5cm in size

- **Papule** (also maculopapular): An elevated solid lesion, up to 0.5 cm in size, circumscribed and firm. It can appear in various colors
- **Patch:** A non-palpable, flat lesion that is different in color and greater than 0.5 cm in size
- **Plaque**: Greater than 1-2 cm in diameter, raised like a papule, solid, rough, and flat-topped
- **Vesicle:** A fluid-filled blister less than 0.5 cm in size
- **Pustule:** Similar to a vesicle but filled with pus instead of fluid
- **Nodule**: A circular, elevated, solid bump of greater than 0.5 cm
- **Telangiectasia**: Clusters of 'spider veins' where tiny blood vessels cause red lines on the skin
- **Tumor:** Is larger than 0.5 cm but similar to a nodule in appearance. They can be benign or malignant
- **Wheal:** An irregular- shaped, solid, elevated area that can vary in color and is transient

II. Secondary lesions

Secondary skin lesions are caused when a primary skin lesion is disturbed, irritated, or changes over time. For example, if eczema is scratched and causes a crust to form, the crust is a secondary lesion. Examples of secondary skin lesions include:

- **Atrophy:** Occurs when skin becomes paper-thin, transparent, and wrinkled, usually due to the use of a topic agent like topical steroids
- **Crust:** A rough, elevated area formed from dried fluid (which can be pus, blood, or serum)
- **Erosion:** Loss of the epidermis, moist and glistening in appearance
- **Excoriation:** Linear scratches that result in the loss of epidermis
- **Fissure:** Linear breaks in the skin that go deeper than the epidermis into the dermis. They can be painful and can be caused by excessive dryness.
- **Lichenification:** A rough, thickening of the epidermis

- **Maceration:** This is when skin becomes wet, wrinkly, and lighter in color due to being in contact with water or fluid for too long. This can occur due to leaking wounds due to improper wound care.
- **Phyma:** A thickening of the skin, often seen in advanced rosacea
- **Scale:** A build-up of keratinized cells that form patches and then flake off the skin
- **Ulcer:** A wound deeper than the epidermis, damaging the dermis, concave, variable in size, and graded depending on depth
- **Umbilication:** A dip inside a skin lesion that looks similar to a navel

A typical gross anatomic lesion might be the solid tumour of a carcinoma of the colon, while the corresponding histological lesion would be the atypical cells (dysplasia) that precede or surround the gross tumour; and a biochemical lesion associated with the same disease process would be the abnormal carcinoembryonic antigen found in the blood of some colon cancer patients.

2.3 Introduction to cellular injury

Cell damage (also known as cell injury) is a variety of changes of stress that a cell suffers due to external as well as internal environmental changes. Cell injury underlies all diseases. So to understand diseases one, has to start by knowing what cell injury is. When a cell is exposed to an injurious agent (i.e. the causes of diseases), the possible outcomes are:

- The cell may adapt to the situation or
- They cell may acquire a reversible injury or
- The cell may obtain an irreversible injury & may die. The cell may die via one of two ways: either by necrosis or by apoptosis.
- Which of these outcomes occur depends on both the injurious agent & on cellular factors. In other words, the result depends on the type, severity, & duration of the injury & on the type of the cell.

2.4 Types of cellular adaptation

The types of cellular adaptation include hypertrophy, atrophy, hyperplasia and metaplasia.

A. Hypertrophy

Hypertrophy is increase in the size of cells. Increased workload leads to increased protein synthesis & increased size & number of intracellular organelles which, in turn, leads to increased cell size. The increased cell size leads to increased size of the organ. Examples: the enlargement of the left ventricle in hypertensive heart disease & the increase in skeletal muscle during sternous exercise.

B. Hyperplasia

Hyperplasia is an increase in the number of cells. It can lead to an increase in the size of the organ. It is usually caused by hormonal stimulation. It can be physiological as in enlargement of the breast during pregnancy or it can pathological as in endometrial hyperplasia.

C. Atrophy

Atrophy is a decrease in the size of a cell. This can lead to decreased size of the organ. The atrophic cell shows autophagic vacuoles which contain cellular debris from degraded organelles. Atrophy can be caused by:

i. Disuse
ii. Undernutrition
iii. Decreased endocrine stimulation
iv. Denervation
v. Old age

D. Metaplasia

Metaplasia is the replacement of one differentiated tissue by another differentiated tissue. There are different types of metaplasia. Examples include:

a. **Squamous metaplasia**: this is replacement of another type of epithelium by squamous epithelium. For example, the columnar epithelium of the bronchus can be replaced by squamous epithelium in cigarette smokers

b. **Osseous metaplasia**: this replacement of a connective tissue by bone, for example at sites of injury**.**

Self-Check – 1	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Write the development of organ lesion (2pts)

2. Define hypertrophy, atrophy and hypotrophy neatly (4pts)

Note: **Satisfactory rating – above 4 points** **Unsatisfactory - below 4 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 2- Identify the principal types of injuries

2.1 Principles of cell injury

Cell injury is defined as a variety of stresses a cell encounters as a result of changes in its internal and external environment. The cellular response to stress may vary and depends upon the following:

- The type of cell and tissue involved.
- Extent and type of cell injury.

2.2 Morphology of Cellular Injury: cells react to adverse influences:-

i. **Cellular adaptation**

- Hyperplasia
- Hypertrophy
- Atrophy
- Metaplasia
- Dysplasia

ii. **Reversible injury**

- intracellular edema,
- fatty change,
- hyaline change,
- amyloidosis,
- mucoid degeneration,
- pathologic pigments

iii. Irreversible injury and dying: Necrosis, Gangrene followed by pathological calcification. Cell death is a state of irreversible injury. It may occur in the living body as a local or focal change: autolysis, necrosis and apoptosis. The changes that follow it: gangrene and pathologic calcification.

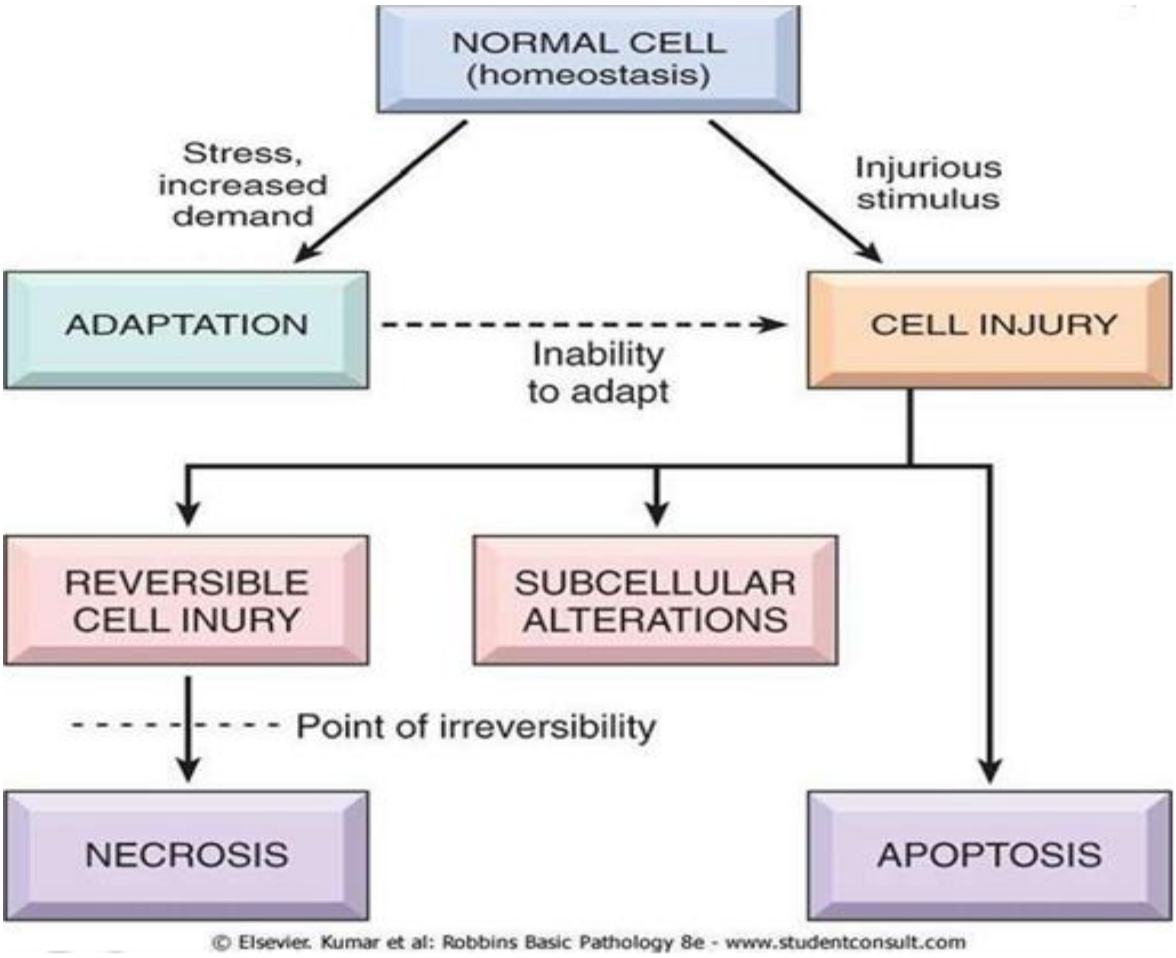

Figure 1: Cellular injury step and its factors

2.3 Cellular Adaptation

- Adaptations are reversible changes in the size, number, phenotype, metabolic activity, or functions of cells in response to changes in their environment
- Cells must constantly adapt, even under normal conditions, to changes in their environment.
- These physiological adaptations usually represent responses of cells to normal stimulation by hormones or endogenous chemical substances.
    - For example, as in the enlargement of the breast and induction of lactation by pregnancy.

Types of Cell Injury: Reversible and Irreversible:

A. **Reversible injury:** If the ischaemia or hypoxia is of short duration, the effects are reversible on rapid restoration of circulation. The sequential changes in reversible cell injury are as under:
   - Intracellular edema
   - Fatty change
   - Hyaline change
   - Amyloidosis
   - Mucoid Degeneration
   - Pathologic Pigments

B. **Irreversible Injury: -** Persistence of ischaemia or hypoxia results in irreversible changes in structure and function of the cell (cell death). Cell death is a state of irreversible injury. It may occur in the living body as a local or focal change: autolysis, necrosis and apoptosis. The changes that follow it: gangrene and pathologic calcification.
   - Increased cell volume.
   - Ruptured lysosome.
   - Damaged cell membrane.
   - Lysed ER
   - Aggregate cystoskeleton.
   - Mitochondrial Swelling and Calcification.

Self-Check – 2	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Explain the term of cell injury (2pts)

2. List and explain the types of cell injury (2pts**)**

Note: **Satisfactory rating – above 4 points** **Unsatisfactory - below 4 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 3- Identify the changes indicative of cells damage

A. DEGERATION

The normal cell is a highly complex unit in which the various organelles and enzyme systems continuously carry out the metabolic activities that maintain cell viability and support its normal functions. Normal function is dependent on:

1. The immediate environment of the cell
2. A continuous supply of nutrients such as oxygen, glucose, and amino acids; and
3. Constant removal of the products of metabolism, including CO_2.

Degeneration refers to the process by which tissue deteriorates and loses its functional ability due to traumatic injury, aging and wear and tear. While degeneration affects all tissues of the body, the natural degenerative process can particularly affect the vertebral discs and joints of the spine, resulting in chronic back pain. Degeneration is reversible but may progress to necrosis if injury persists. When it is associated with abnormal cell function, cell degeneration may also cause clinical disease.

For example, the failure of the blood supply to an organ (ischaemia) due to thrombosis will cause massive cell death due to lack of oxygen. A large area of cell death caused by ischaemia is called an infarction. Another example of cell necrosis is seen in severe viral infections with cytopathic viruses (e.g. **rinderpest**).

Notes

- ***Nonlethal Injury = Degeneration***
- ***Programmed Cell Death = Apoptosis***
- ***Lethal Injury = Necrosis***

Characterizations of Cell degeneration are:

- Abnormality of biochemical function,
- a recognizable structural change, or a combined biochemical and
- Structural abnormality.

B. NECROSIS (Cell Death)

Lethal injuries to the tissues of a living individual cause cell death **(necrosis).** Necrosis is accompanied by biochemical and structural changes and is **irreversible.** The necrotic cells cease to function; if necrosis is sufficiently extensive, clinical disease results.
Notes: Lethal Injury = Necrosis

Cells can die via one of the following two ways:

1. Necrosis
2. Apoptosis

I. Necrosis

In necrosis, excess fluid enters the cell, swells it, & ruptures its membrane which kills it. After the cell has died, intracellular degradative reactions occur within a living organism. Necrosis does not occur in dead organisms. In dead organisms, autolysis & heterolysis take place. Necrosis occurs by the following mechanisms:

a. **Hypoxia:** is decreased oxygen supply to tissues. It can be caused by ischemia, anemia, carbon monoxide poisoning and poor oxygenation of blood due to pulmonary disease.
b. **Free radical-induced cell injury:** Free radical is any molecule with a single unpaired electron in the outer orbital. Examples include superoxide & the hydroxyl radicals. Free radicals are formed by normal metabolism, oxygen toxicity, ionizing radiation, & drugs & chemicals, & reperfusion injury.
c. **Cell membrane damage**
d. **Increased intracellular calcium level**

Types of necrosis

The types of necrosis include:

1. **Coagulative necrosis**

Cogulative necrosis most often results from sudden interruption of blood supply to an organ, especially to the heart. It is, in early stages, characterized by general preservation of tissue architecture. It is marked by the following nuclear changes: Pyknosis (which is chromatin clumping & shrinking with increased basophilia), karyorrhexis (fragmentation of chromatin), & karyolysis (fading of the chromatin material).

2. **Liquefactive necrosis**

Liquefactive necrosis is characterized by digestion of tissue. It shows softening & liquefaction of tissue. It characteristically results from ischemic injury to the CNS. It also occurs in suppurative infections characterized by formation of pus.

3. **Fat necrosis**

Fat necrosis can be caused by trauma to tissue with high fat content, such as the breast or it can also be caused by acute hemorrhagic pancreatitis in which pancreatic enzymes diffuse into the inflamed pancreatic tissue & digest it. The fatty acids released from the digestion form calcium salts (soap formation or dystrophic calcification). In addition, the elastase enzymes digest the blood vessels & cause the hemorrhage inside the pancreas, hence the name hemorrhagic pancreatitis.

4. **Caseous necrosis**

Caseous necrosis has a cheese-like (caseous, white) appearance to the naked eye. And it appears as an amorphous eosinophilic material on microscopic examination. Caseous necrosis is typical of tuberculosis.

5. Gangrenous necrosis

This is due to vascular occlusion & most often affects the lower extremities & the bowel. It is called wet gangrene if it is complicated by bacterial infection which leads to superimposed liquefactive necrosis. Whereas it is called dry gangrene if there is only coagulative necrosis without liquefactive necrosis.

II. Apoptosis

Apoptosis is the death of single cells within clusters of other cells. (Note that necrosis causes the death of clusters of cells.) In apoptosis, the cell shows shrinkage & increased acidophilic staining of the cell. This is followed by fragmentation of the cells. These fragments are called apoptotic bodies. It can also be seen in pathological conditions caused by mild injurious agents. Apoptosis is not followed by inflammation or calcification.

C. Extracellular Accumulation

1. Fatty change

This is accumulation of triglycerides inside parenchymal cells. It is caused by an imbalance between the uptake, utilization, & secretion of fat. Fatty change is usually seen in the liver, heart, or kidney. Fatty liver may be caused by alcohol, diabetes mellitus, malnutrition, obesity, & poisonings. These etiologies cause accumulation of fat in the hepatocytes by the following mechanisms:

- Increased uptake of triglycerides into the parenchymal cells.
- Decreased use of fat by cells.
- Overproduction of fat in cells
- Decreased secretion of fat from the cells.

2. The accumulations of pigments

Pigments can be exogenous or endogenous. Endogenous pigments include melanin, bilirubin, hemosiderin, & lipofuscin. Exogenous pigments include carbon. These pigments can accumulate inside cells in different situations.

2.1. Melanin

Melanin is a brownish-black pigment produced by the melanocytes found in the skin. Increased melanin pigmentation is caused by suntanning & certain diseases e.g. nevus, or malignant melanoma. Decreased melanin pigmentation is seen in albinism & vitiligo.

2.2. Bilirubin

Bilirubin is a yellowish pigment, mainly produced during the degradation of hemoglobin. Excess accumulation of bilirubin causes yellowish discoloration of the sclerae, mucosae, & internal organs. Such a yellowish discoloration is called jaundice.

Jaundice is most often caused by:

a. Hemolytic anemia: is characterized by increased destruction of red blood cells.
b. Biliary obstruction: this is obstruction of intrahepatic or extrahepatic bile ducts. It can be caused by gallstones.
c. Hepatocellular disease: this is associated with failure of conjugation of bilirubin.

2.3. Hemosiderin

Hemosiderin is an iron-containing pigment derived from ferritin. It appears in tissues as golden brown amorphous aggregates & is identified by its staining reaction (bluecolor) with the Prussian blue dye. Hemosiderin exists normally in small amounts within tissue macrophages of the bone marrow, liver, & spleen as physiologic iron stores. It accumulates in tissues in excess amounts in certain diseases.This excess accumulation is divided into 2 types:

I. Hemosiderosis

When accumulation of hemosiderin is primarily within tissue macrophages & is not associated with tissue damage, it is called hemosiderosis.

II. Hemochromatosis

When there is more extensive accumulation of hemosiderin, often within parenchymal cells, which lead to tissue damage, scarring & organ dysfunction, it is called hemochromatosis.

3. Mineralization (Calcification)

Calcification is abnormal deposition of calcium salts in tissue other than bone. Calcium is normally present in blood and deposited in bones. Calcium if deposited in an abnormal tissue with normal or abnormal blood calcium level is considered as pathological condition. Pathological calcifications are:

- Metastatic calcification
- Dystrophic calcification

a. Metastatic calcification

Deposition of calcium occurs in soft tissue following increase in the blood calcium (Hypercalcaemia i.e. 0.12 mg/dL). Hypercalcaemia may arise due to

- Parathyroid tumour in which high levels of parathormone favours phosphate excretion through kidneys (hyperphosphaturia), hypophosphataemia and withdrawal of calcium from bones.
- Primary and secondary bone tumours cause rarefaction of bone.
- Nutritional cause with high vitamin D intake resulting in increased absorption of calcium.
- Renal disease with retention of phosphate, hypophophosphaturia, depression of calcium, parathyroid stimulation and hypercalcaemia.

Notes: Wherever acid is secreted calcium deposition occurs. E.g. Stomach: HCl – Kidneys, Hippuric acid; Lungs - CO2.

b. Dystrophic calcification

Dystrophic calcification is calcification of abnormal tissue with normal blood calcium levels. E.g. Necrotic tissues (Tuberculous lesion, suppurative lesion, renal tubular epithelial cells in mercurial poisoning), scar tissue, dead parasites, atherosclerotic plaques, old thrombi

- Pathogenesis
  - ✓ Deposition of calcium occurs around the nidus. The phosphates from the dead tissues form the nidus. Further, the calcium combines with phosphates to form calcium soaps.
- Gross lesions
  - ✓ Hard, gritty mass and on section gritty sound is heard.

4. Crystals (uretes/ uric acid)

Deposition of different kinds of crystals in tissues like uric acid, sulfonamides and oxalates etc. The uric acid and urates when deposits in tissues it is known as gout. Gout is a disease condition in which urates and uric acid deposited in tissues characterized by intense pain and acute inflammation.

a. Etiology

- Common in poultry due to deficiency of uricase enzyme.
- Deficiency of vitamin-A
- Absence or inadequate amount of uricase.

b. Macroscopic and microscopic features

- White chalky mass of urates and uric acid.
- Deposition of urates/ uric acid on pericardium, kidneys etc.
- Dialation of ureter due to excessive accumulation of urates.

- Presence of sharp crystals in tissue
- Crystals are surrounded by inflammatory cells including macrophages, giant cells and lymphocytes

5. Proteins (Amyloids)

The amyloidosis is heterogeneous group of disorders that are characterized by the deposition of abnormally folded proteins in tissues. Amyloid deposits are formed from globular, soluble proteins, which undergo misfolding and, subsequently, aggregate into insoluble fibrils, leading to progressive organ damage.

Cause of amyloidosis

- Amyloidosis is caused by an abnormality in certain cells found in the bone marrow, called plasma cells.
- The abnormal plasma cells produce abnormal forms of light chain proteins, which enter the bloodstream and can form amyloid deposits.
- Healthy animal have normal light chain proteins in their blood that are part of their natural antibody proteins. These help protect the body from infection.
- The abnormal light chains in patients with amyloidosis clump together into thread-like strings (amyloid fibrils) that the body cannot clear away easily.
- Over time, amyloid fibrils build up as amyloid deposits in tissues and organs. This gradually stops them functioning properly, causing the many symptoms of amyloidosis.

Self-check-3	Written Test

Name ……..……. ID No ……. Signature …….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Choose the best answer

1. Define what cell injury (2pts)

2. Define what is degeneration (2pts)

3. Write the differences of cell degeneration, cell aptosis and cell necrosis (6pts).

Note: **Satisfactory rating – above 4 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

LG #49	LO #3- Identify inflammatory reactions and exudates

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following **content coverage** and topics:

- Describing inflammatory reactions of an animal body.
- Identifying and describing cardinal signs, phases and the inflammatory exudates of acute inflammation.
- Identifying and describing the chronic inflammation and the cells involved in it.
- Identifying and describing clinical important inflammatory lesions including maculae, vesicle, pustule, nodule, erosion, ulcer, scab, and crust.
- Recognizing the process of tissue repair and healing in an animal body.

The need for assistance to improve performance is communicated clearly to the appropriate person. This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, **upon completion of this learning guide, you will be able to**:

- Describe inflammatory reactions of an animal body.
- Identify and describe cardinal signs, phases and the inflammatory exudates of acute inflammation.
- Identify and describe the chronic inflammation and the cells involved in it.
- Identify and describe clinical important inflammatory lesions including maculae, vesicle, pustule, nodule, erosion, ulcer, scab, and crust.
- Recognize the process of tissue repair and healing in an animal body.

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the “Information Sheets”. Try to understand what are being discussed. Ask your trainer for assistance if you have hard time understanding them.
4. Accomplish the “Self-checks” which are placed following all information sheets.
5. Ask from your trainer the key to correction (key answers) or you can request your trainer to correct your work. (You are to get the key answer only after you finished answering the Self-checks).

1.1 Inflammatory response (inflammation)

The inflammatory response (inflammation) occurs when tissues are injured by bacteria, trauma, toxins, heat, or any other cause. The damaged cells release chemicals including histamine, bradykinin, and prostaglandins. These chemicals cause blood vessels to leak fluid into the tissues, causing swelling. Inflammation is a complex process of vascular and cellular alterations occurs in body in response to injury. The term inflammation has been derived from a Latin word inflammare, means to '**set on fire'**.

The inflammation is considered as an important event in body for implementation of existing defense mechanisms in circulating blood to dilute, naturalize or kill the irritant causative agent. Thus, it is said that the immunity is the resistance of body, while inflammation is the implementation of that immunity. It is beneficial to body except chronic persistent and immune origin. Inflammation starts with sub- lethal injury and ends with healing.

1.2 Inflammatory response: is a defense mechanism that evolved in higher organisms to protect them from infection and injury. Its purpose is to localize and eliminate the injurious agent and to remove damaged tissue components so that the body can begin to heal. The response consists of changes in blood flow, an increase in permeability of blood vessels, and the migration of fluid, proteins, and white blood cells (leukocytes) from the circulation to the site of tissue damage.

Etiology of cell injury:

1. **Genetic causes**
    - Developmental defects:

Errors in morphogenesis

- Cytogenetic (Karyotypic) defects: chromosomal abnormalities

- Single-gene defects:

Mendelian disorders

- Multifactorial inheritance disorders.

2. Acquired causes

- Hypoxia and ischemia
- Physical agents
- Chemical agents and drugs
- Microbial agents
- Immunologic agents
- Nutritional derangements
- Aging
- Psychogenic diseases
- Iatrogenic factors
- Idiopathic diseases.

2.1. Oxygen deprivation: Hypoxia

- Ischemia (loss of blood supply).
- Inadequate oxygenation (cardio respiratory failure).
- Loss of oxygen carrying capacity of the blood (anemia or CO poisoning).

2.2. Physical agents:

- Trauma
- Heat
- Cold
- Radiation
- Electric shock

2.3. Chemical agents and drugs:

- Endogenous products: urea, glucose
- Exogenous agents
- Therapeutic drugs: hormones
- Nontherapeutic agents: lead or alcohol

2.4. Infectious agents:

- Viruses
- Rickettsiae
- Bacteria
- Fungi
- Parasites

2.5. Abnormal immunological reactions: The immune process is normally protective but in certain circumstances the reaction may become deranged.

- Hypersensitivity to various substances can lead to anaphylaxis or to more localized lesions such as asthma.

- In other circumstances the immune process may act against the body cells – autoimmunity.

2.6. Nutritional imbalances:

- Protein-calorie deficiencies are the most examples of nutrition deficiencies.
- Vitamins deficiency.
- Excess in nutrition are important causes of morbidity and mortality.
- Excess calories and diet rich in animal fat are now strongly implicated in the development of atherosclerosis.
- Obesity alone leads to an increased vulnerability to certain disorders, such as atherosclerosis, coronary heart disease, diabetes mellitus.

2.7. Aging:

- Programmed aging whereby after a defined number of divisions the cell undergoes terminal differentiation.
- Development of an increasing population of cells irreversibly committed to senescence and death.

Self-Check – 1	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Choose the best answer

1. Define what inflammatory response is or reaction means (4pts)?

2. What is the cause of cells inflammatory reactions are (4pts)

Note: **Satisfactory rating – above 3 points** **Unsatisfactory - below 3 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 2- Identify and describe cardinal signs, phases and the inflammatory exudates of acute inflammation.

2.1 Definition of inflammation

Inflammation, a response triggered by damage to living tissues. Inflammation is a ***local response to cellular injury*** that is marked by capillary dilatation, leukocytic infiltration, redness, heat, and pain and that serves as a mechanism initiating the elimination of noxious agents and of damaged tissue. There are **five cardinal signs** of inflammation, though it may also cause additional symptoms if severe.

1. **Pain:** An unpleasant sensation that can range from mild, localized discomfort to agony. Pain has both physical and emotional components. The physical part of pain results from nerve stimulation. Pain may be contained to a discrete area, as in an injury, or it can be more diffuse, as in disorders like fibromyalgia.
2. **Redness: Erythema** (from the Greek erythros, meaning red) is redness of the skin or mucous membranes, caused by hyperemia (increased blood flow) in superficial capillaries. It occurs with any skin injury, infection, or inflammation.
3. **Swelling:** is an increase in the size or a change in the shape of an area of the body. Swelling can be caused by collection of body fluid, tissue growth, or abnormal movement or position of tissue.
4. **Hotness**: is characterized by high temperature. Being at or exhibiting a temperature that is higher than normal or desirable: a hot forehead.
5. **Loss of function:** A mutation that results in reduced or abolished muscle (protein) function. Muscle function loss is when a muscle does not work or move normally. The medical term for complete loss of muscle function is paralysis.

2.2 Phases of acute inflammation

Acute inflammation is the early (almost immediate) response of a tissue to injury. It is nonspecific and may be evoked by any injury short of one that is immediately lethal. Acute inflammation may be regarded as the first line of defense against injury and is

characterized by changes in the microcirculation: exudation of fluid and emigration of leukocytes from blood vessels to the area of injury. Acute inflammation is typically of short duration, occurring before the immune response becomes established, and it is aimed primarily at removing the injurious agent. Phases of acute inflammation may include

i. Vascular phase

In the vascular phase, small blood vessels adjacent to the injury dilate (vasodilatation) and blood flow to the area increases. The endothelial cells initially swell, then contract to increase the space between them, thereby increasing the permeability of the vascular barrier. This process is regulated by chemical mediators.

Exudation of fluid leads to a net loss of fluid from the vascular space into the interstitial space, resulting in oedema (tumour). The formation of increased tissue fluid acts as a medium for which inflammatory proteins (such as complement and immunoglobulins) can migrate through. It may also help to remove pathogens and cell debris in the area through lymphatic drainage.

ii. Exudative phase

Exudative phase indicates significant alteration in the normal permeability of the small blood vessels in the region of injury. The two components of exudate, fluid and protein, serve good purposes.They are attracted to the site of injury by the presence of chemotaxins, the mediators released into the blood immediately after the insult.

- Margination: cells line up against the endothelium
- Rolling: close contact with and roll along the endothelium
- Adhesion: connecting to the endothelial wall
- Emigration: cells move through the vessel wall to the affected area

iii. Cellular phase

The cellular stage of acute inflammation is marked by changes in the endothelial cells lining the vasculature and movement of phagocytic leukocytes into the area of injury or infection. The most important feature of inflammation is the accumulation of white blood cells at the site of injury. Most of these cells are phagocytes, certain “cell-eating” leukocytes that ingest bacteria and other foreign particles and also clean up cellular debris caused by the injury. The main phagocytes involved in acute inflammation are the neutrophils, a type of white blood cell that contains granules of cell-destroying enzymes and proteins. Outcomes cellular phase results are:

- Complete resolution – with total repair and destruction of the insult
- Fibrosis and scar formation – occurs in cases of significant inflammation
- Chronic inflammation – from a persisting insult
- Formation of an abscess

2.3 Inflammatory exudates of acute inflammation

The main characteristics of acute inflammation are the exudation of fluid and plasma proteins (edema) and the emigration of leukocytes (predominantly neutrophils). Neutrophils and other motile white cells emigrate or move from the blood vessels to the perivascular tissues and the injury (implant) site.

a. “**Exudate”** is fluid buildup caused by tissue leakage due to inflammation or local cellular damage.

b. “**Transudate**” is fluid buildup caused by systemic conditions that alter the pressure in blood vessels, causing fluid to leave the vascular system.

The fluid is exudate if one of the following Light’s criteria is present:

- Effusion protein/serum protein ratio greater than 0.5
- Effusion lactate dehydrogenase (LDH)/serum LDH ratio greater than 0.6
- Effusion LDH level greater than two-thirds the upper limit of the laboratory's reference range of serum LDH.

Lactate dehydrogenase (LDH) is an enzyme normally inside of cells in the body. When there is damage to cells, LDH leaks out and becomes part of the effusion. Measuring a high LDH in the effusion is indicative of cell damage, which typically comes from an exudative process. This is why one of Light's criteria measures LDH in the effusion.

Causes of Transudative cells

- Partially collapsed lung tissue (atelectasis): Due to increased negative pressure inside the lung cavity
- Cerebrospinal fluid (CSF) leak into lung cavity (pleural space): Thoracic spine injury, ventriculoperitoneal (VP) shunt dysfunction
- Heart failure
- Liver dysfunction
- Low blood albumin (hypoalbuminemia)
- Iatrogenic (misplaced catheter into lung)
- Nephrotic syndrome
- Peritoneal dialysis
- Blockage of urinary system causing urine backup in the body (urinothorax due to obstructive uropathy)

Causes of Exudative Effusions

- **Abdominal fluid**: Abscess in tissues near lung, fluid in the abdomen (ascites), Meigs syndrome, pancreatitis
- **Connective tissue disease**: churg-strauss disease, lupus, rheumatoid arthritis, Wegener granulomatosis.
- **Endocrine**: Low thyroid (hypothyroidism), ovarian hyperstimulation.
- **Iatrogenic**: Drug-induced, esophageal perforation, feeding tube in lung
- **Infectious**: Abscess in tissues near lung, bacterial pneumonia, fungal disease, parasites, tuberculosis
- **Inflammatory**: Acute respiratory distress syndrome (ARDS), asbestosis, pancreatitis, radiation, sarcoidosis, high levels of urea in the blood (uremia)
- **Lymphatic abnormalities**: Chylothorax (fluid around the lungs), cancer in the lymph nodes, lymphangiectasia (over-dilation of lymph vessels)
- **Malignancy**: Cancer, lymphoma, leukemia, mesothelioma, paraproteinemia

Self-Check – 2	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. What is inflammation in pathological concepts (4pts)
2. list and explain the 5 cardinal signs of inflammation (2pts)
3. differentiate the term ‘exudate and transudate’ clearly and neatly (4pts)

Note: **Satisfactory rating – above 3 points** **Unsatisfactory - below 3 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 3 - Identify and describe the chronic inflammation and the cells involved in it.

Chronic inflammation

Chronic inflammation is the response of tissue against injurious agent that persists for a long time. The tissue affected by chronic inflammation usually show immune response, phagocytosis, necrosis and repair. Chronic inflammatory process is usually productive or proliferative rather than exudative.

Causes

Chronic inflammation usually develops following acute inflammation due to persistence of infection. Chronic inflammation usually follows repeated acute inflammation Moreover, It may be primary develop due to a low grade of tissue response to persist infection with intracellular microorganisms as Mycobacterium tuberculosis, prolonged exposure to non-degradable but potent toxic substance as silicosis and immune reaction as type IV hypersensitivity.

The affected tissue in chronic inflammation usually shows the following characters, Immune response represented by plasma cells, lymphocytes and macrophages. Moreover, phagocytosis, necrosis and repair, which characterized by presence of newly, formed capillary and fibrous tissue proliferation and collagen fiber deposition.

Microscopic picture

The characteristic feature of chronic inflammation is infiltration of tissue with mononuclear cells specially macrophages, lymphocytes and plasma cells besides newly develop blood vessels, fibroblast proliferation and collagen fibre deposition.

Gross appearance

Chronic inflammation is characterized by absence of cardinal signs of inflammation. The inflammatory area is indurated and grayish white in color. Moreover the inflammation

may be not noticed. Granulomatous inflammation Granulomatous inflammation is a special kind of chronic inflammation, which occurs in the presence of indigestible material and/or cell-mediated immunity. Moreover, granulomatous inflammation is a type of chronic inflammation characterized by presence of granuloma. Granuloma composed of closely packed collection of cells or sheets of predominantly macrophages. Macrophage in granuloma usually activated (epithelioid cells). Besides macrophages, granulomatous inflammation shows lymphocytes, plasma cells, giant cells and sometimes neutrophils. The classic granulomatous diseases include tuberculosis, leprosy, foreign body reactions, including the reactions to everything from sutures to schistosome eggs.

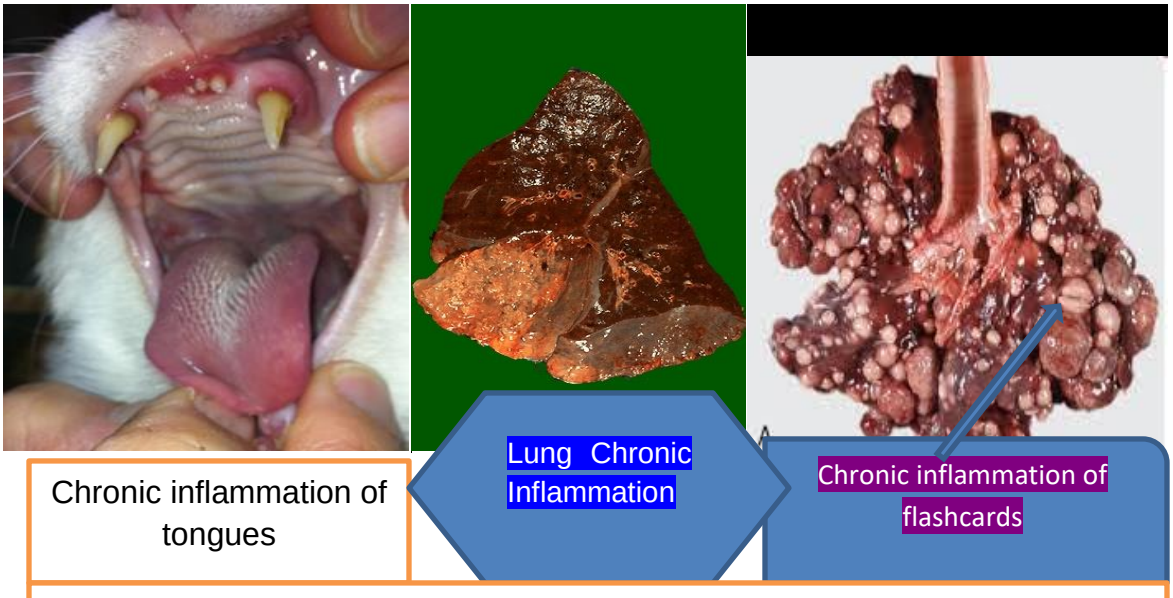

Figure 2: chronic inflammation of different animals

Self-Check – 3	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Define and take an example of chronic inflammation means (4pts)?

2. What is the etiological agent of chronic inflammation means (2pts)?

Note: **Satisfactory rating – above 6 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 4- Identifying and describing clinical inflammatory lesions

4.1 Clinical inflammatory lesions

Inflammatory cells are a source of cytokines and growth factors that may target the endothelial cells and contribute to the development of structural and functional abnormalities of the vessel wall. Inflammatory lesions in acne include small red bumps (papules), pustules, large red bumps (nodules) and pseudocysts (these are fluctuant nodules). Inflammatory acne lesions are often painful.

- **Macule:** Macules are circumscribed alterations in skin color. The skin surface is either elevated or depressed in relation to the surrounding skin. Macules may be of any size or color.
- **Papule:** Papule is a solid, elevated lesion with no visible fluid which may be up to ½ cm. in diameter. The elevation may be accounted for by metabolic deposits, infiltrates, or hyperplasia of cellular elements, etc. A papulosquamous lesion is a papule with desquamation (scaling).
- **Nodules:** Nodules are forms of papules, but are larger and deeper. They may be located in the dermis or subcutaneous tissue, or in the epidermis. Nodules are usually ½ cm. or more in diameter. Ex: Metastatic neoplasm; xanthoma
- **Plaque:** An elevated area of skin 2 cm. or more in diameter. It may be formed by a coalescence of papules or nodules. The surface area is greater than its height. It is a plate-like lesion.
- **Wheal:** A wheal is an evanescent rounded or flat-topped elevation in the skin that is edematous, and often erythematous. They may vary in size from a few mm. to many cm. The shape may change and these lesions are usually pruritic (itchy). These are really variations of papules, nodules or plaques that are evanescent.
- **Vesicles and Bullae:** (Blisters) Vesicles are circumscribed epidermal elevations in the skin containing clear fluid and less than ½ cm. in diameter. If the lesion has a diameter of greater than ½ cm, it is called a bulla. Vesicles and bullae arise from a

cleavage at various levels of the skin. The more superficial the location, the more flaccid the bullous lesion. Vesicles and bullae are commonly called blisters. It is the diameter, not the cleavage plane that differentiates vesicles and bullae.

- **Pustule:** A pustule is a circumscribed elevation of the skin that contains a purulent exudate that may be white, yellow, or greenish-yellow in color.
- **Abscess:** A localized collection of pus in a cavity formed by disintegration or necrosis of tissue.
- **Cyst:** A cyst is a closed sac that contains liquid or semisolid material. On palpation a cyst is usually resilient.
- **Atrophy:** Atrophy of the skin may involve the epidermis, or the dermis, or both. It is the thinning process associated with decreased number of cutaneous cells. Sometimes the normal skin markings may be lost. Dermal atrophy may give rise to a depression in the skin. Stria (plural striae) is linear, atrophic, pink, purple, or white lesions of the skin and is sometimes called “stretch marks”.
- **Sclerosis:** Sclerosis refers to a circumscribed, diffuse hardening or induration in the skin. It is usually produced by induration of the dermis and/or subcutaneous tissue. Palpation is often necessary in diagnosing sclerosis.
- **Erosion:** A loss of epidermis.
- **Ulcer:** A loss of epidermis and dermis (and sometimes deeper tissue). If erosions and/or ulcers are produced by scratching, the term excoriation is used.
- **Scar:** Scars occur whenever ulceration has taken place and they reflect the pattern of healing. They may be hypertrophic, atrophic, or cribriform (perforated with multiple small pits).
- **Crusts (scabs):** Crusts result when serum, blood, or purulent exudate dries and it is a hallmark of pyogenic infection. Crusts are yellow when they have arisen from dried serum; green or yellow-green when formed from purulent exudate; and brown or dark red when formed from blood.
- **Lichenification:** A chronic thickening of the epidermis with exaggeration of its normal markings, often as a result of scratching or rubbing.

- **Acne:** presents primarily as papules but can also cause pustules, nodules, or cysts. It is most common on the face, neck, chest, and upper back and can leave scars if not treated
- **Keratitis:** is caused by exposure to sunlight (ultraviolet radiation) and appears as thick, scaly crusts on the skin.
- **Blisters:** a small bubble on the skin filled with serum and caused by friction, burning, or other damage.
- **Cellulitis:** a lesion caused by agents that affected skin appears swollen and red and may be hot and tender. Without treatment with an antibiotic, cellulitis can be life-threatening.
- **Sore:** physically painful or sensitive, as a wound, hurt, or diseased part: a sore arm. Suffering bodily pain from wounds, bruises, etc., as a person: He is sore because of all that exercise. Suffering mental pain; grieved, distressed, or sorrowful: to be sore at heart.
- **Dermatitis:** is a general term that describes a common skin irritation. It has many causes and forms and usually involves itchy, dry skin or a rash. Or it might cause the skin to blister, ooze, and crust or flake off.
- **Enzema:** a particular type of inflammatory reaction of the skin in which there is erythema (reddening), edema (swelling), papules (bumps), and crusting of the skin followed, finally, by lichenification (thickening) and scaling of the skin.
- **Utricaria:** is a unique dermatologic disorder caused by infiltration of mast cells in the skin and has pathology distinct from common urticaria but can present with urticarial lesions associated with blisters.
- **Scabies:** is an itchy skin condition caused by a tiny burrowing mite called Sarcoptes scabiei. Intense itching occurs in the area where the mite burrows.
- **Cyst:** is caused by an infestation of the skin by the human itch mite (Sarcoptes scabiei var. hominis). The microscopic scabies mite burrows into the upper layer of the skin where it lives and lays its eggs. The most common symptoms of scabies are intense itching and a pimple-like skin rash.

The shape, size, color and texture of the primary lesion as well as any symptoms that may or may not be present are important in describing skin lesions. The arrangements of lesions in relation to one another as well as their distribution over the body are also important in fully describing a dermatosis. The following terms may apply to the shape or arrangement of skin lesions: linear, annular, polycyclic; aciform; serpiginous; grouped (herpetiform and zosteriform); agminate (collected together into clusters or masses); reticular (netlike). The following terms are helpful in describing the distribution of skin lesions: generalized; localized; bilateral; unilateral; symmetrical; asymmetrical; sun-exposed; intertriginous.

Miscellaneous:

- *Pruritus = itching*
- *Pruritic = itchy*
- *Erythema = redness of the skin produced by vascular congestion or increased perfusion.*

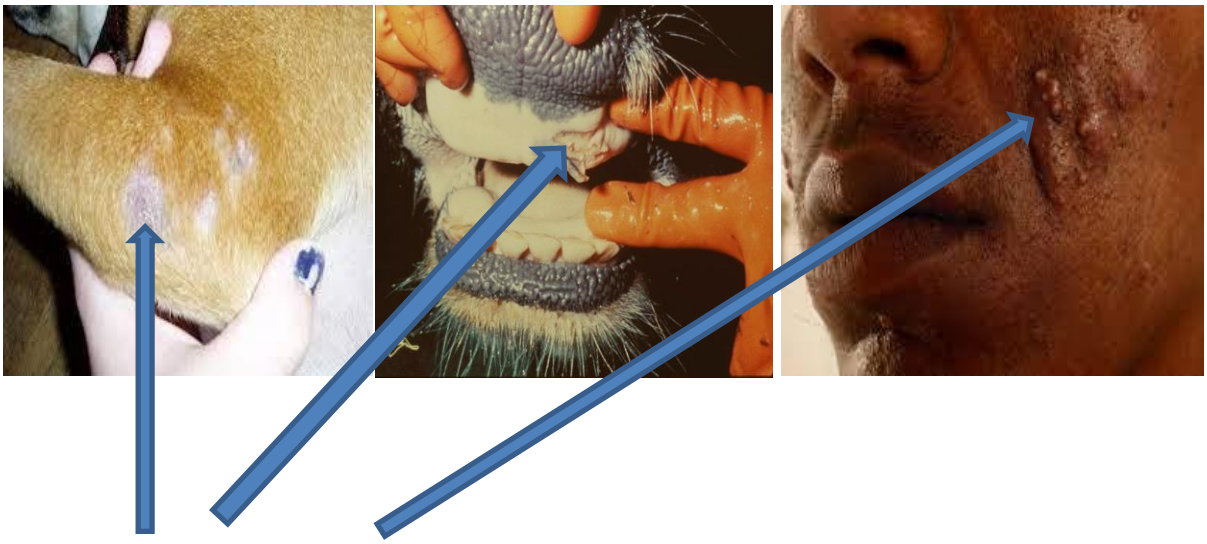

Figure 3: Cyst, Blister and **Acne** types of lesionsbl

Self-Check – 4	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short answer questions

1. What is inflammatory lesion is (2pts).

2. List and explain the cell lesions neatly (4pts).

Note: **Satisfactory rating – above 6 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 5 - Recognizing the process of tissue repair and healing

Introduction Tissue Repair and Healing

Wound Healing: Healing is the body response to injury in an attempt to restore normal structure and function. Two Distinct processes

1. **Regeneration:** - Complete restoration of the original tissues.
2. **Repair:** - When the healing takes place by proliferation of connective tissue element resulting in fibrosis and scarring.

5.1 Healing (Tissue Repair)

- **Tissue:** Proliferating potential of cell types
- **Labile cells** - Continuously dividing cells. E.g. epidermis, epithelial cells, bone marrow cells.
- **Stable cells**
  - Quiescent cells
  - Undergoes division occasionally
  - Liver, kidney pancreas, fibroblasts, endothelial cells
- **Permanent cells**
  - Non–dividing cells
  - Neurons, muscle cells (cardiac, skeletal)

Hence, healing occurs by:-

1. Healing by regeneration
2. Healing by substitution

Depending on the proliferation potential of the cells as described above.

Wound healing

Wound healing is not a separate process and occurs along with the inflammatory reaction. It is a complex but orderly phenomenon involving a number of processes. Namely,

- Acute inflammatory reaction following initial injury.
- Parenchymatous cellular regeneration
- Migration and production of parenchymatous and connective tissue cell
- Extracellular matrix, protein synthesis
- Remodelling of connective tissue

Healing by primary union or first intention

- This type of healing occurs in clean surgical approximated incision i.e. limited bleeding and tissue destruction.
- The sequence of events occurring in primary union is given below

0 hrs	Clot filling the incised area
3-24hrs.	Neutrophilic infiltration
48 hrs	Basal cell proliferation and epithelial closure takes place by 24-48 hours
72 hrs	Macrophages replace neutrophils. Granulation tissue begins to appear. Collagen is arranged vertically
120 hrs	Incised space is filled with granulation tissue. Neovascularisation is maximal. Collagen fibre begin to appear and epithelial proliferation is maximal
2 weeks	Proliferation of fibroblast with continuous collagen accumulation producing a scar. Type III collagen is deposited early in scar tissue and is replaced by adult type I collagen which accounts for wound strength. Newly formed blood vessels disappear.
8 week	Scar tissue consists of granulation tissue which is devoid of inflammation covering intact epidermis.

Healing by second intention

The wound involved shows extensive loss of cells and tissue. E.g. infarction, ulceration, abscesses, surface wound with large defects. The wound is filled with tissue debris, a few erythrocytes and bacteria. Abundant granulation tissue (soft, pink, granular appearance of wound surfaces) grows in from the margin to fill the defect but at the same time the wound contracts i.e., the defect is marked by depression and decrease from its original size. Microscopically granulation tissue consists of new capillaries, fibroblasts, collagen and proteoglycan rich ground substance. Initially granulation tissue is soft and spongy due to leaky blood vessels

Injury – open wound – excess loss of tissue – infected – necrosis – inflammation

↓

Blood clot

↓

24 hours – neutrophils infiltrate to destroy irritant

↓

48-72 hours – macrophages and lymphocytes infiltrate

↓

Removal of necrotic and cellular debris by liquefaction by macrophages

↓

Red granules from underneath (granulation tissue) represent proliferating capillaries.

Fibroblast also proliferate to fill the gap

↓

There is a definite order.

Base - capillaries grow vertically and project towards the surface. Fibroblast grows perpendicular to capillary and parallel to surface – pulling pressure of the wound

↓

Surface – fibroblasts are arranged parallel to capillaries exerting tension towards wound surface for easy closure. This arrangement differentiates granulation tissue from fibrosarcoma which lacks orderly arrangement.

↓

The surface is closed by the epithelium proliferating from the margin

↓

The tissue is devoid of sweat gland, sebaceous gland, hair and hair follicles and pigment. So, the scar appears dry and unpigmented white and puckered as it becomes avascular and shrinkage of collagen.

Exuberant granulation or proud flesh

- Sometimes the granulation continues to grow in abnormally large amount due to irritant, movement or trauma which prevents healing. This condition is called proud flesh or excess granulation tissue.

Keloid

- Keloid is another condition. Reason for its development is not known. The connective tissue below the epithelial covering continues to proliferate. This condition may recur after the removal. This is found in horses and black people having some genetic or familial predisposition.

Systemic and local factors influencing wound healing

Systemic factors

- Nutritional
  - ✓ Vitamins – vitamin C is required for collagen synthesis.
  - ✓ Proteins deficiency – starvation.
  - ✓ Sulphur containing amino acids (methionine and cystine) are important and required for intermediate forms of collagen.
  - ✓ Zinc – as metalloenzyme, it is essential for remodelling of extracellular matrix
- Metabolic factors
  - ✓ Diabetes mellitus – delays healing.
  - ✓ Hyperadrenocortism
- Circulatory stasis or adequacy of blood supply

- ✓ Inadequate blood supply – delays healing

- Hormones: concurrent glucocorticoid therapy hinders inflammatory and reparatory process

Local factors

- Infection can delay healing
- Mechanical – movements directly affect wound healing
- Foreign bodies impede healing
- Size, location and type of wound
- Cold inhibits wound healing

Others

- Old age-Healing is slower than young ones.
- Chemotherapeutic agents
- Radiation
- Immunodeficiency

Self-Check – 5	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Define what is cell repair and healing differences (4pts)?

2. What is term ‘regeneration and repair’ (2pts).

Note: **Satisfactory rating – above 6 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

LG #50	LO #4 - Identify pathological circulatory disorders

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following **content coverage** and topics:

- Identifying and describing impaired blood supply to tissues (ischemia and infarction) and its causes (thrombosis, embolism and vasoconstriction).
- Identifying the nature and characteristics of ischemia and infarctions on different organs of an animal body.
- Identifying abnormal findings including hyperaemia, congestion, jaundice /icterus; related to circulatory system.
- Identifying and describing the development, the causes and effects of oedema.
- Identifying shock and the different types and causes of shock.

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, **you will be able to**:

- Identify and describe impaired blood supply to tissues (ischemia and infarction) and its causes (thrombosis, embolism and vasoconstriction).
- Identify the nature and characteristics of ischemia and infarctions on different organs of an animal body.
- Identify abnormal findings including hyperaemia, congestion, jaundice /icterus; related to circulatory system.
- Identify and describe the development, the causes and effects of oedema.
- Identify shock and the different types and causes of shock.

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the “Information Sheets”. Try to understand what are being discussed. Ask your trainer for assistance if you have hard time understanding them.
4. Accomplish the “Self-checks” which are placed following all information sheets.
5. Ask from your trainer the key to correction (key answers) or you can request your trainer to correct your work. (You are to get the key answer only after you finished answering the Self-checks).

Information Sheet 1- Identifying and describing impaired blood supply to tissues (ischemia and infarction) and its causes

Introduction to ischemia and infarction

Ischemia is decreased blood flow to or from an organ, while **infarction** is the cellular response to lack of perfusion (blocked blood flow to or from organs). Ischemia can be caused by obstruction of arterial blood flow – the most common cause, or by decreased perfusion of tissues by oxygen-carrying blood as occurs in cardiac failure, hypotension, & shock.

Infarction

Definition: An infract is an area of ischemic necrosis caused by occlusion of either the arterial supply or venous drainage in a particular tissue. Nearly 99% of all infarcts result from thrombotic or embolic events. Other mechanisms include (all of them are arterial in origin):

- Local vasospasm
- Expansion of atheroma due to hemorrage in to athermotous plaque.
- External compression of the vessels. E.g. trauma
- Entrapment of vessels at hernial rings etc.

The development & the size of an infarct are determined by the following factors:

I. The nature of the vascular supply
II. The rate of development of occlusion
III. Susceptibility of the tissue for hypoxia
IV. Oxygen content of the blood
V. The severity and duration of ischemia

I. The nature of vascular supply

The following organs have a dual blood supply.

- Lung → pulumonary artery
  → Bronchial artery
- Liver → hepatic artery
  → Portal vein
- Hand & forearm → Radial arteries
  → Ulnar arteries.

The effect of such a dual blood supply is that if there is obstruction of one of the arterial supplies, the other one may offset the rapid occurrence of infarction in these organs unlike the renal & splenic circulations which have end arterial supply. Infarction caused by venous thrombosis is more likely to occur in organs with single venous outflow channels, such as testis &ovary.

II. Rate of development occlusion

Slowly developing occlusions are less likely to cause infraction since they provide time for the development of collaterals.

III. Tissue susceptibility to hypoxia

The susceptibility of a tissue to hypoxia influences the likelihood of infarction. Neurons undergo irreversible damage when deprived of their blood supply for only 3 to 4 minutes. Myocardial cells die after 20-30 minutes of ischemia. Fibroblasts are more resistant, especially those in the myocardium.

IV. Oxygen content of blood

Partial obstruction of the flow of blood in an anaemic or cyanotic patient may lead to tissue infarction.

V. The severity & duration of ischemia.

Types of infarcts

Infarcts are classified depending on:

1. The basis of their colour (reflecting the amount of hemorrhage) into:
   b. Hemorrhagic (Red) infarcts
   c. Anemic (White) infarcts

2. The presence or absence of microbial infection into:
   a. Septic infarcts
   b. Bland infarcts

Red infarcts occur in:

a. Venous occlusions as in ovarian torsion
b. Loose tissues such as the lung which allow blood to collect in infarct zone.
c. Tissues with dual circulations (e.g. the lung), permitting flow of blood from unobstructed vessel in to necrotic zone.
d. In tissues that were previously congested because of sluggish outflow of blood.
e. When blood flow is reestablished to a site of previous arterial occlusion & necrosis.

White infarcts occur in:

- Arterial occlusion in organs with a single arterial blood supply.
- Solid organs such as the heart, spleen, & kidney, where the solidity of the tissue limits the amount of hemorrage that can percolate or seep in to the area of ischemic necrosis from the nearby capillaries.

Morphology of infarcts

Gross: All infarcts are wedge-shaped with the occluded vessel at the apex and the periphery of the organ forming the base of the wedge. The infarction will induce inflammation in the tissue surrounding the area of infarction. Following inflammation, some of the infarcts may show recovery, however, most are ultimately replaced with scars except in the brain.

Microscopy: The dominant histologic feature of infarction is ischemic coagulative necrosis. The brain is an exception to this generalization, where liquefactive necrosis is common.

Clinical examples of infarction:

A. **Myocardial infarction**
   - Usually results from occlusive thrombosis supervening on ulcerating atheroma of a major coronary artery.
   - Is a white infarct.
   - Can cause sudden death, cardiac failure, etc.

B. **Cerebral infarcts**
   - May appear as pale or hemorrhagic
   - A fatal increase in intracranial pressure may occur due to swelling of large cerebral infarction, as recent infarcts are raised above the surface since hypoxic cells lack the ability to maintain ionic gradients & they absorb water & swell.
   - Is one type of cerebrovascular accidents (CVA) or stroke which has various clinical manifestations.

C. **Lung infarcts**
   - Are typically dark red & conical (wedge-shaped).
   - Can cause chest pain, hemoptysis, etc.

D. **Splenic infarcts**
   - Conical & sub capsular
   - Initially dark red later turned to be pale.

Etiology of ischemia and infarction

A. **Thrombosis:** is a formation of clotted mass of blood within the cardiovascular system, while **blood clot** is a clump of blood that has changed from a liquid to a gel-like or semisolid state. Differences between thrombus and blood clot

	Formation	Composition	Prognosis
Thrombus	• Blood vessels • Platelets • Blood clotting system	• Platelets • Fibrin	• Life threatening
Blood clot	• Blood clotting system	• Only fibrin	• Life saving

Causes for thrombosis

- Trauma : lacerations, contusion, rupture, o/r injection
- Toxins : Streptococci, erysipelothrix (vegetations)
- Degenerations : Atherosclerosis (damage to intima)
- Viruses : Hog cholera virus
- Parasites : Strongylus vulgaris in anterior mesenteric artery in horses
- Tumours : Invading tumours

Mechanism of thrombus formation

I. **Active**
   - Antithrombotic factors and prothrombotic factors are seen on surface of endothelium

II. **Passive**
   - Endothelium is thrombo-resistant whereas sub-endothelial connective tissue is highly thrombogenic.
   - Sub-endothelial connective tissue consists of collagen, elastic, fibrinonectin, laminin glycosoaminoglycans and thrombosporin.

- Damage to endothelium exposes the sub-endothelial connective tissue and activates intrinsic blood clotting pathway and platelet adhesion.

Antithrombotic factors (present on endothelial cells) - Inhibit thrombosis

- Anticoagulant properties
  - ✓ Thrombo modulin - Protect against action of heparin and thrombin which converts fibrinogen to fibrin
- Anti-platelet properties - Inhibit platelet aggregation
  - ✓ Prostacyclin (PGI2)
  - ✓ Nitric oxide (NO_2)
- Fibrinolytic properties
  - ✓ Tissue plasminogen activator (tPAs) - Promotes fibrinolytic activity in blood and reacts against blood clots

Thrombotic factors

- Tissue factor (Thromboplastin)
  - ✓ Present on endothelium in small amounts
  - ✓ Activate extrinsic clotting pathway
  - ✓ Stimulated by
  - ✓ Endotoxins
  - ✓ Cytokines (IL – 1)
  - ✓ Tumour necrosis factor (TNF)
- von Willebrand factor (vWF)
  - ✓ Protein helps in platelet adherence thrombus
- Platelet Activating Factor (PAF)
  - ✓ Helps in platelet aggregation thrombus
- Inhibitor of Plasminogen Activator
  - ✓ Prevents fibrinolysis thrombus

Notes

- **Normal homeostasis:** There will be a balance between antithrombotic and prothrombotic factors in normal endothelium.

B. Embolism

- An embolus is any foreign body floating in blood. The process is called embolism.

Location of embolism

- Artery / venous / capillaries / lymphatics
- In domestic animals emboli always occurs in arteries
- In human, venous embolism is common
- Thrombus in leg vein may form emboli to reach large blood vessel, right side heart and pulmonary artery embolism

Types / Causes of emboli

- Thrombotic emboli : Thrombo embolism – arteries (Thrombi detach to form emboli)
  - ✓ Heart – vegetations
  - ✓ Parasitic; atherosclerotic; bacteria
- Bacterial emboli : Septicaemia
- Parasitic emboli : *Dirofilaria immitis* - Pulmonary artery – dog
  - ✓ Schistosomes – Portal; mesenteric; Nasal blood vessels
  - ✓ Trypanasomes – If tartar emetic is given rapidly, it kills large number of organism and forms emboli on coronary vessels which is fatal.
  - ✓ Filarial - Lymphatics emboli in brain
- Neoplastic emboli: Clumps of tumour cells in circulation producing metastatic tumours.
- Fibrin - In blood transfusion, when blood is improperly defibrinated / inadequate anticoagulants
- Fat embolus - In fracture of long bones, fat in the marrow cavity gets dislodged and forms emboli. These are lodged in lungs and leads to death.
- “Fat embolism syndrome“ (Acute respiratory symptoms, tachycardia neurological symptoms)
- Air or gas emboli

C. **Vasoconstriction:** is the narrowing (constriction) of blood vessels by small muscles in their walls. When blood vessels constrict, blood flow is slowed or blocked. Vasoconstriction may be slight or severe. It may result from disease, drugs, or psychological conditions.

D. **Aneurysm:** (dilation or outpouching of a blood vessel wall) are pathologic abnormalities that can occur within the vasculature.

Self-Check – 1	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. List the pathological condition that occurred at blood cells (2pts)?

2. Define and differentiate ‘Ischemia and infarction’ (4pts)?

3. What are the causes of ‘Ischemia and infarction’ (4pts)?

Note: **Satisfactory rating – above 3 points** **Unsatisfactory - below 3 points**

You can ask your teacher the copy of the answer sheet

Information sheet 2 – Identifying the nature and characteristics of ischemia and infarctions

2.1 Nature and characteristics of ischemia

Ischemia or ischaemia is a restriction in blood supply to tissues, causing a shortage of oxygen that is needed for cellular metabolism (to keep tissue alive). Ischemia is deficiency of arterial blood in any part of an organ. It is also known as local anemia local anemia.

Description

- Decreased blood supply that cannot meet the oxygen demands of an organ or tissue
- If not corrected, ischemia can lead to cell death due to cellular swelling (orcosis).

Pathogenesis

- Decreased arterial perfusion (e.g., due to atherosclerosis, thromboembolism) in solid organs with only a single (end-arterial) blood supply (e.g., kidney, heart) → pale infarct
- Decreased venous drainage (e.g., venous occlusion, Budd-Chiari syndrome, testicular torsion, ovarian torsion) in tissues with more than one blood supply (e.g., intestine, lung, liver, testes) or reperfusion (e.g., following angioplasty) → red infarct
- Shock with the following variants:
  - ✓ Hypovolemic shock (e.g., hemorrhage) → ↓ intravascular volume → ↓ delivery of oxygen to tissue → ischemia
  - ✓ Cardiogenic shock (e.g., cardiac tamponade) → ↓ left ventricular function → ↓ forward flow of blood → ↓ delivery of oxygen to tissue → ischemia

- ✓ Distributive shock (e.g., septic, neurogenic, and anaphylactic shock) → systemic vasodilation → peripheral pooling of blood → ↓ delivery of oxygen to tissue → ischemia

Pathophysiology of ischemia

Ischemia results in tissue damage in a process known as ischemic cascade. The damage is the result of the:

- Build-up of metabolic waste products,
- Inability to maintain cell membranes,
- Mitochondrial damage, and
- Leakage of autolyzing proteolytic enzymes into the cell and surrounding tissues
- Cardiac arrhythmias (irregular heart beat; too fast or slow)
- Cellular self-destruction
- Damage white blood cells

Etiology

- Occlusion: The thrombi may dislodge and may travel anywhere in the circulatory system, where they may lead to pulmonary embolus, an acute arterial occlusion causing the oxygen and blood supply distal to the embolus to decrease suddenly.
- Trauma: traumatic injury to an extremity may produce partial or total occlusion of a vessel from compression, shearing, or laceration.
- External pressure on artery
- Narrowing / obliteration of lumen of artery
- Thrombi / emboli

Notes

- ***Ischemia:*** *Death of cell by absences of blood supply or lack of oxygenated blood*
- ***oncosis:*** *Death of cells with swelling.*

- ***Necrosis:*** *Cell death by environmental stimuli with uncontrolled release of inflammatory cellular contents.*
- ***infarction:*** *Cell death by occlusion of blood supply veins or arteries*

2.2 Nature and characteristics of infarctions

Infraction

Definition: An infract is an area of ischemic necrosis caused by occlusion of either the arterial supply or venous drainage in a particular tissue. Nearly 99% of all infarcts result from thrombotic or embolic events. Other mechanisms include:

- Local vasospasm
- Expansion of atheroma due to hemorrage in to athermotous plaque.
- External compression of the vessels. e.g trauma
- Entrapment of vessels at hernial sacks etc.

The development & the size of an infarct are determined by the following factors:

a. The nature of the vascular supply
b. The rate of development of occlusion
c. Susceptibility of the tissue for hypoxia
d. Oxygen content of the blood
e. The severity & duration of ischemia

Self-Check – 2	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Define the morphological changes occurred in ‘Ischemia and Infarction’ (4pts)?

Note: **Satisfactory rating – above 4 points** **Unsatisfactory - below 4 points**

You can ask your teacher the copy of the answer sheet

Information Sheet -3- Identifying abnormal findings circulatory system.

3.1 HYPERAEMIA AND CONGESTION

- Hyperaemia is increased volume of blood in affected tissue or part.

Hyperaemia (Active hyperaemia)

- Occurs in arterioles or arteries
- Increased blood flow in capillaries

Congestion (Passive hyperaemia)

- Occurs due to impaired venous drainage
- Stasis of blood in veins

Classification of Hyperaemia

1. **Active Hyperaemia**
   - Increased blood in arterial side
   - Usually due to inflammation
   - All active hyperaemia are acute
   - Chronic active hyperaemia does not occur
   - Occurs when there is a demand for oxygen and nutrients - increase metabolism
   - It is beneficial.

2. **Acute General Active Hyperaemia**
   - Increased blood throughout the body

Causes

- Various systemic diseases. E.g. Pasteurellosis, erysipelas
  - ✓ Rapidly beating heart → increased blood supply
- Renal diseases - due to retention of fluids

Macroscopically

- Bright red color or organs

Microscopically

- Arteries and capillaries dilated with blood

Result

- Disappears if cause is removed

3. Acute Local Active Hyperaemia

- Increased amount of blood in arterial system within a local area (leg, Stomach, lung)
- Most common type of hyperaemia

Causes

- Physiological
  - ✓ Occurs in stomach and intestine following a meal
  - ✓ Lactating mammary gland
  - ✓ Muscles during exercise
  - ✓ Genital tract during oestrus

Blushing

- Acute inflammation

Macroscopically

- Enlarged, swollen, heary
- ↑ warmth in Skin

Microscopically

- In live animals, arteries, arterioles and capillaries are distended with blood
- Difficult to detect in dead animals

CONGESTION

- Increased blood in the venous end due to improper drainage.
- GRNERAL - if interference is central (i.e.) lungs, heart
- LOCAL - if vein of an organ or body

- It can be acute or chronic
- Chronic venous congestion is more common

Types of congestion

1. **Acute**
    - Increase in the amount of blood on the venous side of circulatory system
    - Due to sudden obstruction to the flow of blood in heart and lungs.

Causes

- Heart failure
    - ✓ Degeneration and necrosis of myocardium
    - ✓ Myocardial infarction
- Pneumonia
- Pulmonary thrombosis or embolism
- Hydropericardium, Haemopericadium, etc.
- Hydrothorax, Haemothorax, etc.

Macroscopically

- Organs are blue in color (Unoxygenated blood)
- Veins distended with blood
- Organs enlarged, heavy
- Upon incision, blood oozes out

Result

- Causes are mild → Recovery
- Causes are severe → Death

2. **Chronic**

Increased blood on venous end persisting for long period of time causes Permanent changes (fibrosis, atrophy).

Causes due to central lesions in heart and lungs

- Heart lesions
  - ✓ Stenosis of valvular openings
- Valvular insufficiency
  - ✓ Failure of cusps of valves to close property
  - ✓ Inflammatory tissue
  - ✓ Thrombus
- Myocardial failure
  - ✓ Degeneration and necrosis of muscles

Contraction of muscles

↓

Blood pushed in arteries

↓

But accumulates in venous side

Anomalies of heart

- Persistent foramen orale
- Interventricular septal defects

Blood moves from one chamber to another

↓

Arterial blood pressure maintained

↓

Blood accumulates in venous end.

- Constrictive lesons in pericardium
  - ✓ Traumatic pericarditis in cattle
- Lesions of lungs
  - ✓ Obliteration of capillary bed in lungs
  - ✓ Prevents free flow of blood through the lungs
  - ✓ Retards flow through right side of heart
  - ✓ Blood back flows into Liver

- ✓ Causes
  - ❖ Chronic alveolar pulmonary emphysema in horses (BROKEN WIND)
  - ❖ Pneumonia
  - ❖ Hydrothorax, haemothorax
- Compression of major pulmonary vessels
  - ✓ Tumours
  - ✓ Cysts
  - ✓ Abscesses

3. Acute Local

Increase in blood in the veins of a portion (foot, tail, kidney etc) Due to sudden obstruction to blood flow

Causes

- Malposition of viscera
  - ✓ Volvulus, intussusception, torsion
- External pressure
  - ✓ Ligatures, tourniquets, bandages

Pathogenesis

- Pathogenesis

4. Hydrostatic

- Accumulation of blood in ventral portions of the body due to gravity.

Causes

- Occurs in heart diseases
- Recumbency
- Inactive animals
- Large animals
- Heart failure - Agonal congestion

Appearances

- Veins in ventral portion or organs distended with blood
- Lungs - increase capillary bed

- Intestine & kidneys – necrosis and gangrene
- Causes pneumonia and gangrene of intestine

Significance

- Indicates

✓ the side of animals which was ventral at the time of death

✓ Heart was not able to pump properly

✓ Location of body in medico–legal cases

Grossly and microscopically

Veins are engorged with blood

↓

Necrosis of endothelial cells

↓

Haemorrhage

5. Chronic Local

- Increase in amount of blood for a long time in veins
- Permanent tissue changes (atrophy, fibrosis)

Causes

- External pressure
  - ✓ Tumors, abscesses
- Obstruction from within
  - ✓ Thrombus (blood clot)

Gross and microscopic appearance

- Enlarged initially later undergoes atrophy
- Veins - bluish blood
- Oedema due to increase permeability of capillaries
- Fibrosis

a. JAUNDICE /Icterus

Icterus is increased amount of bile pigments in blood circulation and is often called as hyper- bilirubinemia or jaundice. It is of three types.

i. Hemolytic

Hemolytic jaundice occurs as a result of excessive hemolysis in circulating blood. It is also known as prehepatic jaundice.

Etiology

- Piroplasmosis (Babesia bigemina)
- Anaplasmosis (Anaplasma marginale)
- Leptospirosis(Leptospira ictehaemmorrhagae)
- Equine infectious anemia virus
- Anthrax (Bacillus anthracis)
- Clostriduum hemolyticum
- haemolytic streptococci

ii. Toxic

Toxic jaundice occurs as a result of damage in liver leading to increased amount of unconjugated and conjugated bilirubin in blood. It is also known as hepatic jaundice.

a. Etiology

- Toxinj Poisons
- Copper poisoning
- Leptospirosis

iii. Obstructive Jaundice: occurs as a result of obstruction in bile duct causing hindrance in normal flow of bile. It is also known as post hepatic jundice.

Etiology

- Blocking of bile canaliculi by swollen hepatocytes
- Obstruction in bile duct (Liver flukes, tapeworms and ascaris)
- Biliary cirrhosis, Cholangitis and Cholelithiasis

- Pressure on bile duct due to abscess, neoplasm.
- Inflammation and swelling at duct opening in duodenum

Self-Check – 3	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. What are the pathological lesion occurred in blood circulation (2pts)?

2. Explain clearly the differences among hyperemia, congestion and icterus neatly (4pts)?

Note: **Satisfactory rating – above 3 points** **Unsatisfactory - below 3 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 4- Identifying and describing the development, the causes and effects of oedema.

4.1 INTRODUCTION TO OEDEMA

- Oedema denotes abnormal accumulation of fluid in the interstitial tissue spaces and body cavities.
- There is always certain amount of fluid in these structures, but any excess is removed by the lymphatics as lymph.
- In health condition there is a constant passage of fluid from the capillaries to the tissues and vice versa, and the chief factors concerned in the process are hydrostatic (capillary blood-pressure) and osmotic pressure of the protein.
- At the arterial end of capillaries blood pressure exceeds osmotic pressure of the blood and fluid diffuses to the tissue spaces.
- At the venous end of capillaries osmotic pressure of blood increases owing to increased concentration of colloids, whereas, blood pressure falls hence facilitating re-absorption of fluid.
- If the hydrostatic or osmotic balances in the blood vessels and tissues are impaired or the permeability of capillaries is increased oedema would develop.

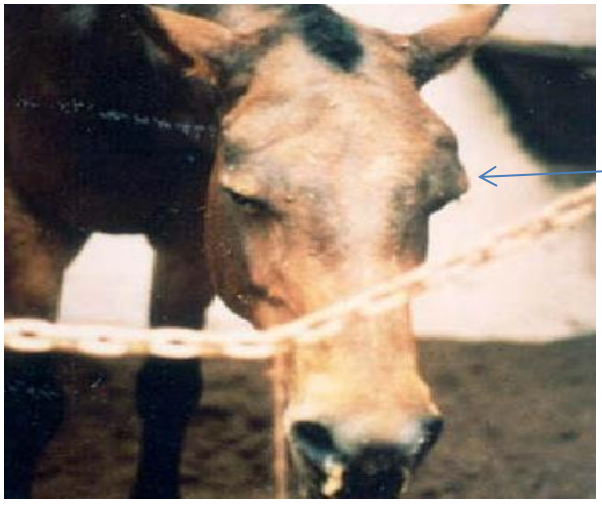

Figure 4. Oedema of Supra orbital fossa: African Horse sickness

Factors concerned with production of oedema:

- Increased capillary blood pressure (hydrostatic pressure)
- Decreased colloid osmotic pressure of blood plasma
- Increased permeability of capillary wall
- Increased colloid osmotic pressure of the tissue
- Lymphatic obstruction- only causes local oedema

NB: colloid osmotic balance of blood is controlled by a protein called albumin. Damage to the liver, which is responsible for the synthesis of albumin, would lead to oedema.

Fluid nature of oedema:

- Oedematous fluid is transudate.
- Resembles plasma and has pale or straw-colour
- It is watery, but contains protein and has lower specific gravity
- It does not clot in any way

Structural changes due to oedema:

When subcutaneous tissues are involved:

- Puffy swelling that pits on pressure
- Freely moving fluid that oozes on cutting
- Generalized subcutaneous oedema is called **anasarca.**

In the involvement of serous cavities:

- **Ascites-** fluid in the peritoneum
- **Hydro pericardium-** fluid in the pericardium
- **Hydrothorax**- fluid in the thoracic cavity

Types of oedema

1. **Renal oedema-** renal diseases may lead to filtration of protein from glomeruli leading to hypoproteinaemia hence causing oedema.

2. **Parasitic oedema** – this develops due to blood sucking parasites like *Hemonchus* in sheep. In cases of hemonchosis fluid accumulates under the lower jaw known as bottle-jaw/submandibular oedema due to lowering of the neck during grazing.
3. **Nutritional oedema-** mal-nutrition leads to hypoproteinaemia.
4. **Liver diseases-** albumin and other proteins are synthesised in the liver. Any disease in the liver may interfere with albumin synthesis
5. **Cardiac oedema**- this is due to lack of increased hydrostatic pressure. E.g. heart failure (congestion)
6. **Inflammatory oedema**- there histamine production in inflammation. Histamine causes dilatation of vessel wall leading to leakage of fluid and colloid.

Outcome of oedema:

1. Recovery if the cause is removed
2. Inflammation due to bacterial infection
3. Death due to:
   - ✓ Pressure of accumulating fluid
   - ✓ Inability of vital organs to function. E.g. oedema of lungs that fills the alveoli with fluid
   - ✓ Asphyxia- oedema of larynx

Self-Check – 4	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Define Oedema (2pts)

2. List the types of oedema (2pts).

Note: **Satisfactory rating – above 4 points** **Unsatisfactory - below 4 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 5 - Identifying shock, types and causes

Shock

Definition: Shock is a state in which there is failure of the circulatory system to maintain adequate cellular perfusion resulting in widespread reduction in delivery of oxygen & other nutrients to tissues. In shock, the mean arterial pressure is less than 60 mmHg or the systolic blood pressure is less than 90 mmHg. Adequate organ perfusion depends on arterial blood pressure (BP) which, in turn, depends on:

- Cardiac output.
- Peripheral vascular resistance.

Cardiac output = stroke volume X heart rate

In turn, stroke volume depends on:

a. Preload i.e. blood volume,
b. Afterload i.e. arterial resistance, &
c. Myocardial contractility.

Therefore, shock (i.e. widespread decreased perfusion of tissues) occurs when the preload (i.e. the blood volume) is decreased, or when the afterload (the peripheral vascular resistance) is decreased, or when the myocardium fails to contract. These basic mechanisms of shock are used to classify it. Next, we will look at the classification of shock.

Types of shock: Shock can be divided into:

1. Hypovolemic shock
2. Cardiogenic shock
3. Distributive shock

A. Hypovolemic shock

Definition: This is shock caused by reduced blood volume. Reduction in circulating blood volume results in the reduction of the preload which leads to inadequate left ventricular filling, reflected as decreased left & right ventricular end diastolic volume and pressure. The reduced preload culminates in decreased cardiac output which leads to widespread tissue perfusion (shock).

Causes of hypovolumic shock include:

i. Haemorrhage
ii. Diarrhoea and vomiting
iii. Burns
iv. Trauma and etc.

The effect of haemorrhage depends on the rate and amount of blood loss. Hypovolumic shock is the most common type of shock in clinical medicine .A normal healthy adult can lose 550ml (10%of blood volume) without significant symptoms.

B. Cardiogenic shock

Definition: This is shock that results from severe depression of cardiac performance. It primarily results from pump failure (myocardial failure). Causes of cardiogenic shock can be divided into:

1. Myopathic
2. Mechanical

1. Myopathic causes of cardiogenic shock include:
   - Acute myocardial infraction. Usually shock occurs in this conditioin if ≥ 40% of the left ventricular mass & more on the right ventricle is involved by infarction.
   - Mycocarditis
   - Dilated cardiomyopathy/hypertrophic cardiomyopathy
   - Myocardial depression in septic shock and etc.

III. Mechanical

a. Intracardiac

1. Left ventricle outflow obstruction E.g.Aortic stenosis, hypertrophic cardiomyopathy
2. Reduction in forward cardiac output E.g. Aortic or mitral regurgitation
3. Arrhythmia

b. Extracardiac

This can be called obstructive shock. The extracardiac causes of cardiogenic shock can be caused by:

1. Pericardial tamponade (gross fluid accumulation in the pericardial space) results in a decreased ventricular diastolic filling → ↓CO
2. Tension pneumothorax (gas accumulation in pleural space). This decreases the venous return by creating a positive pressure.
3. Acute massive pulumonary embolism occupying 50-60% of pulumonary vascular bed.
4. Severe pulumonary hypertension (10 pulmonary hypertension).

C. Distributive shock

Definition: Distributive shock refers to a group of shock subtypes caused by profound peripheral vasodilatation despite normal or high cardiac output.

Causes of distributive shock:

1. Septic shock – the commonest among the group & clinically very important.
2. Neurogenic shock
   - Usually occurs in the setting of anaesthetic procedure [cephalo-caudal migration of anaesthetic agent] or spinal cord injury owing to loss of vascular tone & peripheral pooling of blood.
3. Anaphylactic shock
   - Initiated by generalized IgE – mediated hypersensitivity response, associated with systemic vasodilatation & increased vascular permeability.

4. Endocrine shock
    - This is a type of shock that typically occurs in adrenal insufficiency.

Notes

- **Bactermia:** is the presence of viable bacteria in the blood as evidenced by blood culture.
- **Septicemia:** is systemic infection due the presence of microbes and their toxin the blood.
- **Sepsis:** is a systemic response to severe infection mediated via macrophage-derived cytokines that target end organ receptors in response to infection.

Self-Check – 5	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Define shock (2pts)?
2. List and define the types of shock (4pts)?
3. What is the cause of cardiogenic shock (2pts)?

Note: **Satisfactory rating – above 8 points** **Unsatisfactory - below 8 points**

You can ask your teacher the copy of the answer sheet

LG #51	LO #4- Identify disorders of cell/tissue growth

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following **content coverage** and topics:

- Seeing the congenital defects of the different body systems.
- Identifying all adaptive changes in cells which help the cell to cope with an alteration in its environment.
- Identifying and describing the common types of neoplasia occurring in animals.
- Identifying and describing the appearance and classification of the different types of neoplasia.

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, **you will be able to**:

- See the congenital defects of the different body systems.
- Identify all adaptive changes in cells which help the cell to cope with an alteration in its environment.
- Identify and describe the common types of neoplasia occurring in animals.
- Identify and describe the appearance and classification of the different types of neoplasia.

Learning Instructions:

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Information Sheet 1- See the congenital defects of the different body systems.

Introduction to congenital defects

Congenital anomalies can be defined as structural or functional anomalies that occur during intrauterine life. Also called birth defects, congenital disorders, or congenital malformations, these conditions develop prenatally and may be identified before or at birth, or later in life.

figure : Congenital defeacts occurred at animals level

2. **Disturbances in Development**
   (Anomalies & Monster)

- Anomaly is developmental defect affecting an organ or part of the body.
- Anomaly is the disturbance of development that involves an organ or a portion of an organ.
- Monster is an animal in which extensive abnormal developments are present.
- A Congenital disease is one in which the patient is born with the disease whereas an inherited disease is one which is due to factors in the genetic materials received from the parents.

Classification of Anomalies

A. Arrest of Development

1. ***Agenesia*** is an incomplete and imperfect development of an organ or part and **aplasia** is absence of an organ or part. There some exaamples of agenesis and aplasia lists:
    - **Acrania** is absence of most or all of the bones of the cranium.
    - **Amelia** is absence of one or more limbs.
    - **Agnathia** is absence of lower jaw.
    - **Anencephalia** is absence of the brain
    - **Hypocephalia** is incomplete development of the brain.
    - **Anophathalmia** is absence of one or both eyes.
    - **Hemicrania** is absence of half of the head.
    - **Atresia** is absence of normal opening e.g. Atresia ani is absence of anus opening and Atresia coli is absence of rectum.
    - **Exencephalia** is defective skull with brain exposed or extruded. If the protruding brain contains a ventricle which is filled with excessive amount of fluid, the malformation is a hydrencephalocele.
    - **Arhinencephalia** is absence or rudimentary development of the olfactory lobe with corresponding lack of development of the external olfactory organs.
    - **Agnathia** is absence of the lower jaw.
    - **Anophthalmia** is absence of one or both eyes.
    - **Abrachia** is absence of the forelimbs.
    - **Abrachiocephalia** is absence of forelimbs and head.
    - **Adactylia** is absence of digits.

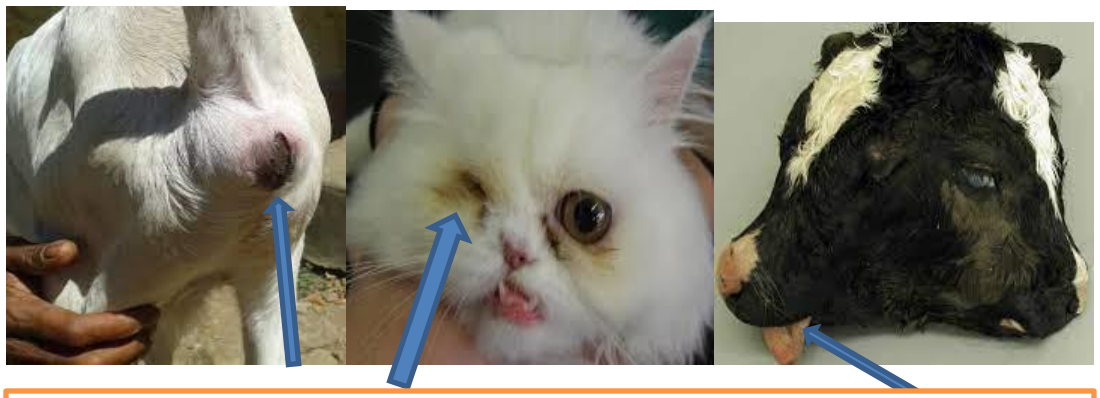

Figure 5: Anomalies of Atresia, Anophthalmia and Anencephalia

2. **Fissures on the median line of the head, thorax, and abdomen.**
   - Craniooschisis (skull)
   - Cheiloschisis (lip), often referred to as harelip.
   - Palatoschisis (oral) cavity, often called cleft palate. Harelip and cleft palate result from faulty development of the maxillary process derived from the first visceral arch.
   - Rachischisis (spinal column).
   - Schistorrachis or spina bifida (spinal column)
   - Schistothorax (thoraz or sternum).
   - Schistosomus (abdomen).
   - Schistocormus (thorax, neck or abdominal wall). Results from arrested development of the amnion.

Figure 5: Organs monasteries indications of some animals

3. **Fusion of paired organs**
   - Cyclopia (eyes)
   - Ren arcuatus (kidneys), often referred to as horseshoe kidney.

B. Excess of Development

i. Congenital hypertrophy

ii. Hemi hypertrophy (partial)

- ✓ Increase in the number of a part.

- Polyotia (ears)
- Polyodontia(teeth)
- Polymelia (limbs)
- Polydactylia(digits)

- Polymastia (mammary gland)
- Polythelia(teats)

DISPLACEMENTS DURING DEVEOPMENT

I. Displacements of organs

- Dextrocardia is transposition of the heart to the right side.
- Ectopia cordis cervicalis is displacement of the heart into the neck.

II. Displacements of tissues

- Teratoma is inclusion of multiple displaced and also neoplastic tissues within an individual.
- Dermoid is inclusion within an individual of a mass containing skin, hair, feathers, or teeth depending on the species and often arranged as an epidermal cyst (Dermoid cyst).
- Odontoid cyst is inclusion within an individual of a mass of dental enamel and cement.
- Dentigerous cyst is inclusion within an individual of one or more imperfectly formed teeth.
- Fusion of Sexual Characters
- Hermaphrodite is an individual having both testicular and ovarian tissue. Pseudohermaphrodite is an animal having unisexual development of the sex glands (either testicular or ovarian tissue), but having also either a unisexual or bisexual development of the other parts of the genitalia.
- Freemartin is a female calf having arrested development of the sex organs and being the twin of perfect male.

Monsters

A monster or monstrosity is a disturbance of development that involves several organs and causes great distortion of the individual. For the most part monsters possess a duplication of all or most of the organs and other parts of the body. They develop from a single ovum. They are therefore the product of incomplete twinning.

Classification of the Monsters

- Twins Entirely Separate
  - ✓ Although separate, these twins are in a single chorion. One twin as a rule is well developed; the other is malformed (acardius). In the malformed foetus there is arrested development of the heart, lungs, and trunk. Such monsters may lack a head (acephalus), limbs and other recognizable features (amorphous), or the trunk (acormus).
- Twins United
  - ✓ These twins are more or less completely united and are of symmetrical development.

Twins United

I. **Anterior Twinning:** The anterior part of the individual is double, the posterior single.

- Pygopagus – united in the pelvic region with the bodies side by side.
- Ischiopagus – united in the pelvic region with the bodies at an obtuse (not pointed) angle.
- Dicephalus – two separate heads; doubling may also affect the neck, thorax and trunk.
- Diproosopus – doubling in the cephalic region without complete separation of heads; only the face doubled.

II. **Posterior Twinning:** The posterior part is double, the anterior single.

- Craniopagus – brains usually separated; bodies as a rule at an acute angle.
- Cephalothoracopagus – union of head and thorax.
- Dipygus – doubling of posterior extremities and posterior part of body.

Figure 6: twins united anomalies

III. Twinning Almost Complete

Duplication of the whole trunk or the anterior or posterior extremities with parallel, ventral arrangement of the foetuses. The pair is joined in the region of the thorax, and also often in the abdominal region.

- Thoracopagus – united only by the thorax.
- Prosopothoracopagus – besides the union the thorax the abdomen, the head and neck are united.
- Rachipagus – thorax and lumbar portion of the spinal column united.

Self-Check – 1	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

3. Define congenital in pathological concept (4pts).

4. List atleast 5 congenital organs and explain it (3pts).

5. What is aplasia and freemartins' (4pts).

Note: **Satisfactory rating – above 6 points** **Unsatisfactory - below 5 points**

You can ask your teacher the copy of the answer sheet

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Information Sheet 2- Identifying all adaptive changes in cells

2.1 Cellular changes and adaptive responses

Cellular adaptation is the ability of cells to respond to various types of stimuli and adverse environmental changes. Tissues adapt differently depending on the replicative characteristics of the cells that make up the tissue. For example, labile tissue such as the skin can rapidly replicate, and therefore can also regenerate after injury, whereas permanent tissue such as neural and cardiac tissue cannot regenerate after injury. If cells are not able to adapt to the adverse environmental changes, cell death occurs physiologically in the form of apoptosis, or pathologically, in the form of necrosis. This article provides an overview of the main cellular adaptive mechanisms and their different consequences in the human body. Cellular adaptations include:

- Hypertrophy (enlargement of individual cells),
- Hyperplasia (increase in cell number),
- Atrophy (reduction in size and cell number),
- Metaplasia (transformation from one type of epithelium to another), and
- Dysplasia (disordered growth of cells).

Tissues adapt differently depending on the replicative characteristics of the cells that make up the tissue. For example, labile tissue such as the skin can rapidly replicate, and therefore can also regenerate after injury, whereas permanent tissue such as neural and cardiac tissue cannot regenerate after injury. If cells are not able to adapt to the adverse environmental changes, cell death occurs physiologically in the form of apoptosis, or pathologically, in the form of necrosis.

2.2 Types of cellular adaptation

i. Hypertrophy

Hypertrophy is increase in the size of cells. Increased workload leads to increased protein synthesis & increased size & number of intracellular organelles which, in turn, leads to increased cell size. The increased cell size leads to increased size of the

organ. E.g.: the enlargement of the left ventricle in hypertensive heart disease & the increase in skeletal muscle during sternous exercise.

ii. Hyperplasia

Hyperplasia is an increase in the number of cells. It can lead to an increase in the size of the organ. It is usually caused by hormonal stimulation. It can be physiological as in enlargement of the breast during pregnancy or it can pathological as in endometrial hyperplasia.

iii. Atrophy

Atrophy is a decrease in the size of a cell. This can lead to decreased size of the organ. The atrophic cell shows autophagic vacuoles which contain cellular debris from degraded organelles. Atrophy can be caused by:

- Disuse
- Undernutrition
- Decreased endocrine stimulation
- denervation
- Old age

iv. Metaplasia

Metaplasia is the replacement of one differentiated tissue by another differentiated tissue. There are different types of metaplasia. Examples include:

A. **Squamous metaplasia**: this is replacement of another type of epithelium by squamous epithelium. For example, the columnar epithelium of the bronchus can be replaced by squamous epithelium in cigarette smokers
B. **Osseous metaplasia:** this replacement of a connective tissue by bone, for example at sites of injury.

v. Dysplasia

A term used to describe the presence of abnormal cells within a tissue or organ. Dysplasia is not cancer, but it may sometimes become cancer. Dysplasia can be mild, moderate, or severe, depending on how abnormal the cells look under a microscope and how much of the tissue or organ is affected.

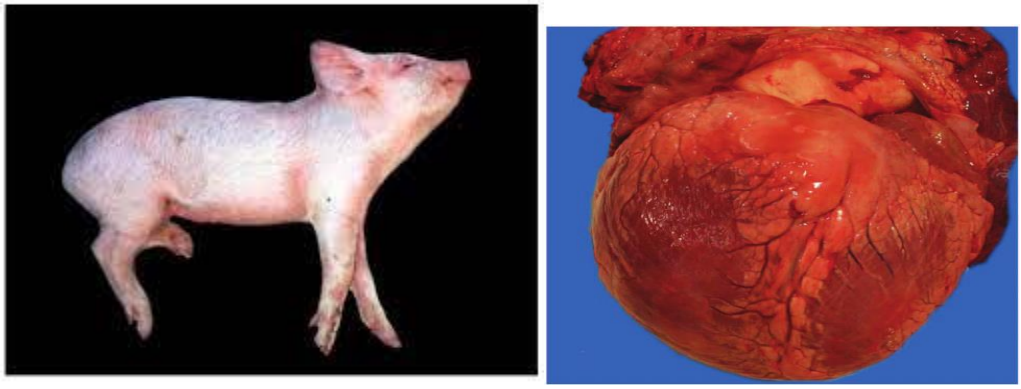

Figure 3: Organs and bovine heart hypertrophy

Figure 5: *Development of **metaplasia** in oral cavity of both animals and plant*

Self-Check – 2	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Compare & contrast the various types of cellular adaptation (4pts).

2. Define cell injury (2pts).

Note: **Satisfactory rating – above 6 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 3 - Identifying and describing the types of neoplasia

Definition and Nomenclature

Recently, neoplasia means new growth and technically, defined as abnormal mass of tissues the growth of which exceeds and persists in the same excessive manner after cessation of the stimulus, evoking the transformation.

Nomenclature: Neoplasms are named based upon two factors:

- On the histologic types → ***mesenchymal*** and ***epithelial***
- On behavioral patterns : ***benign*** and ***malignant*** neoplasms

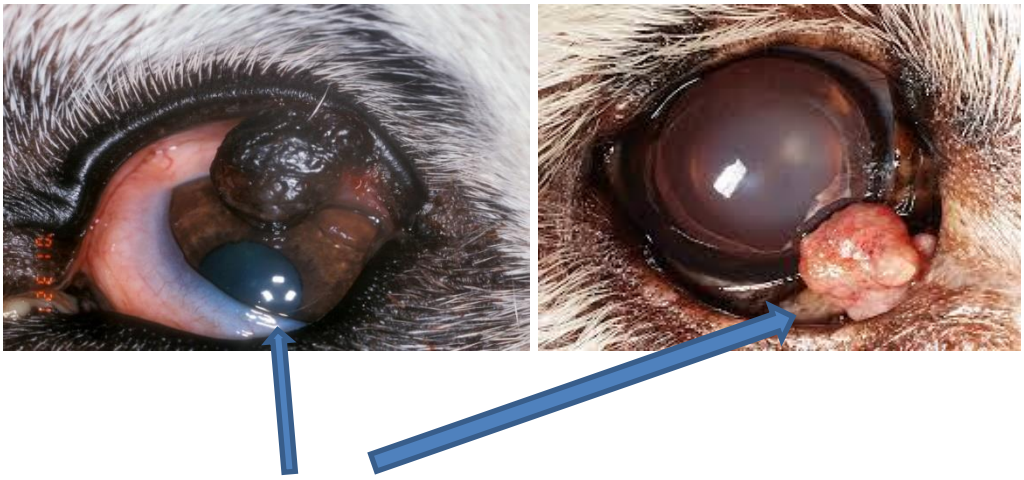

Figure 3: *Neoplasia* *of dog eye and eyelid*

Neoplasms are classified so that one can:

- Deduce a prognosis
- Investigate the cause
  - ✓ Perhaps with a view to prevention
- Assess the results of treatment.

- It must be remembered that there are several types of proliferative change, and neoplasia must be distinguised from these.
- In addition to neoplasia, hyperplasia and dysplasia are also proliferative changes.
  - ✓ Inflammatory, repair and granuloma lesions may also masquerade as neoplasms.
- However, destructive or necrotic tumours may have inflammation present.
  - ✓ Unlike inflammation, hyperplasia or dysplasia, neoplastic cells show uncontrolled proliferation in the absence of a triggering stimulus.

Types of Classification

Tumours are classified by:

1. **Histogenesis:** by identification of the cells/ tissue of origin.
2. **Anatomical origin**: by site of anatomical growth.
3. **State of differentiation**: by relative development or maturity of the cell type involved.
4. **Behaviour**: by host-tumour growth patterns. e.g. ***benign*** *or* ***malignant****.*

A. Behaviour

- The terms 'benign' and 'malignant' represent two ends of a spectrum of behaviour patterns in tumours.
- Certain features exhibited by the neoplastic cells allow tumours to be labelled as benign or malignant.

Characteristics of Benign and Malignant Neoplasms

Benign neoplasia: Thus, the suffix -oma denotes a benign neoplasm. Benign mesenchymal neoplasms originating from muscle, bone, fat, blood vessel nerve, fibrous

tissue and cartilages are named as Rhabdomyoma, osteoma, lipoma, hemangioma, neuroma, fibroma and chondroma respectively. Benign epithelial neoplasms are classified on the basis of cell of origin for example adenoma is the term for benign epithelial neoplasm that form glandular pattern or on basis of microscopic or macroscopic patterns for example visible finger like or warty projection from epithelial surface are referred to as papillomas.

Malignant neoplasm nomenclature essentially follows the same scheme used for benign neoplasm with certain additions. Malignant neoplasms arising from mesenchymal tissues are called sarcomas (Greed sar =fleshy). Thus, it is a fleshy tumour. These neoplasms are named as fibrosarcoma, liposarcoma, osteosarcoma, hemangiosarcoma etc. Malignant neoplasms of epithelial cell origin derived from any of the three germ layers are called carcinomas.

Eg. Ectodermal origin: skin (epidermis squamous cell carcinoma, basal cell carcinoma) Mesodermal origin: renal tubules (renal cell carcinoma).Endodermal origin: linings of the gastrointestinal tract (colonic carcinoma). The difference in characteristics of these neoplasms can be conveniently discussed under the following:-

a. Differentiation & anaplasia
b. Rate of growth
c. Local invasion
d. Metastasis

A. Differentiation & anaplasia

Differentiation refers to the extent to which parenchymal cells resemble comparable normal cells both morphologically and functionally. Thus, well-differentiated tumours cells resemble mature normal cells of tissue of origin. In general, benign neoplasms are well differentiated. Malignant neoplasms in contrast, range from well differentiated, moderately differentiated to poorly differentiate types. Malignant neoplasm composed of undifferentiated cells are said to be anaplastic, literally anaplasia means to form backward.

- Morphology of anaplastic cell includes large Pleomorphic; hyperchromatic nucleus with high nuclear cytoplasmic ratio 1:1(normally 1:4 to 1:6). The cell usually

reveals large nucleoli with high and often abnormal mitoses. Tumour giant cells and frequent loss of polarity of epithelial arrangements are encountered.

- On functional differentiation, the well differentiated the neoplasm, the more completely it retains the functional capabilities found in its normal counterparts thus, endocrine tumours produce hormone (ex. Thyroid, adrenal) so also, well differentiated squamous cell carcinoma and well differentiated hepatocellular carcinomas produce keratine and bile respectively.
- However, highly anaplastic or undifferentiated cells of what cell tissue of origin come to resemble each other functionally and morphologically more than the normal cells which they have arisen this is called chemical convergence.

B. Rate of growth

- Most benign tumours grow slowly whereas; most malignant tumours grow rapidly sometimes, at erratic pace.
- Some benign tumours for example uterine leiomyoma increase in size during pregnancy due to probably steroidal effects (estrogen) and regress in menopause.
- In general, the growth rate of neoplasms correlate with their level of differentiation and thus, most malignant neoplasms grow more rapidly than do benign neoplasms.
- On occasions, cancers have been observed to decrease in size and even spontaneously disappear. E.g. renal cell carcinoma, malignant melanoma, choriocarcinoma.

C. Local invasion

- Nearly all benign neoplasms grow as cohesive expansile masses that remains localized to their site of origin and do not have the capacity to invade or metastasize to distant sites, as do malignant neoplasms.
- Rims of fibrous capsules encapsulate most benign neoplasms. However, hemangiomas and neurofibromas are exceptions. Thus, such encapsulations tend to contain the 193 benign neoplasms as a discrete, rapidly palpable and easily movable mass that can easily surgically enucleated.

- The growth of malignant neoplasms is accompanied by progressive infiltration, invasion and destruction of the surrounding tissue. Generally, they are poorly demarcated from the surrounding normal tissue (and a well-defined cleavage plane is lacking).
- Next to the development of metastasis, invasiveness is the most reliable feature that differentiates malignant from benign neoplasms.
- Even though, malignant neoplasms can invade all tissues in the body, connective tissues are the favoured invasive path for most malignant neoplasms, due to the elaboration of some enzymes such as type IV collagnases & plasmin, which is specific to collagen of basement membrane. Several matrix-degrading enzymes including glycosidase may be associated with tumour invasion.

D. Metastasis

- It is defined as a transfer of malignant cells from one site to another not directly connected with it (as it is described in the above steps).
- Metastasis is the most reliable sign of malignancy. The invasiveness of cancers permits them to penetrate in to the blood vessel, lymphatic and body cavities providing the opportunity for spread.
- Most malignant neoplasm metastasies except few such as gliomas in the central nervous system, basal cell carcinoma (Rodent ulcer) in the skin and dermatofibrosarcoma in soft tissues.
- Organs least favoured for metastatic spread include striated muscles and spleen.
- Since the pattern of metastasis is unpredictable, no judgment can be made about the possibility of metastasis from pathologic examination of the primary tumour.

Pathways of spread:

Dissemination of malignant neoplasm may occur through one of the following pathways.

1. Seeding of body cavities and surfaces (transcoelomic spread)
2. Lymphatic spread: The most common pathway for the initial dissemination of carcinomas
3. Hematogenous spread

Self-Check – 3	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Define neoplasia (3pts).

2. Discuss the differences between benign and malignant neoplasms (3pts).

Note: **Satisfactory rating – above 6 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 4 - Identifying and describing the appearance and types of neoplasia.

4.1 Neoplasia

Neoplasia is the abnormal growth of a tissue into a mass that is not responsive to normal control mechanisms and may be benign or malignant. Growth of this mass is not affected when the inciting stimulus is removed.

Overview: The terms **tumor, nodule, and mass** are nonspecific terms that refer to an abnormal proliferation of cells. The term **neoplasm** means new growth and *does not* imply benign or malignant (i.e., there are benign neoplasms, and there are malignant neoplasms).

Nomenclature for general categories of neoplasms

1. **Adenoma:** Benign neoplasm derived from glandular cells.

Adenoma is **a type of non-cancerous tumor or benign** that may affect various organs. It is derived from the word “adeno” meaning 'pertaining to a gland'. Every cell in the body has a tightly regulated system that dictates when it needs to grow, mature and eventually die off.

2. **Carcinoma:** Malignant neoplasm derived from epithelial cells.

- Carcinoma refers to a malignant neoplasm of epithelial origin or cancer of the internal or external lining of the body. Carcinomas, malignancies of epithelial tissue, account for 80 to 90 percent of all cancer cases.
- Epithelial tissue is found throughout the body. It is present in the skin, as well as the covering and lining of organs and internal passageways, such as the gastrointestinal tract.

- Carcinomas are divided into two major subtypes: adenocarcinoma, which develops in an organ or gland, and squamous cell carcinoma, which originates in the squamous epithelium.
- Adenocarcinomas generally occur in mucus membranes and are first seen as a thickened plaque-like white mucosa. They often spread easily through the soft tissue where they occur. Squamous cell carcinomas occur in many areas of the body.
- Most carcinomas affect organs or glands capable of secretion, such as the breasts, which produce milk, or the lungs, which secrete mucus, or colon or prostate or bladder.

3. **Sarcoma:** Malignant neoplasm derived from mesenchymal cells (e.g., fat, muscle).

Sarcoma refers to cancer that originates in supportive and connective tissues such as bones, tendons, cartilage, muscle, and fat. Generally occurring in young adults, the most common sarcoma often develops as a painful mass on the bone. Sarcoma tumors usually resemble the tissue in which they grow. Examples of sarcomas are:

- Osteosarcoma or osteogenic sarcoma (bone)
- Chondrosarcoma (cartilage)
- Leiomyosarcoma (smooth muscle)
- Rhabdomyosarcoma (skeletal muscle)
- Mesothelial sarcoma or mesothelioma (membranous lining of body cavities)
- Fibrosarcoma (fibrous tissue)
- Angiosarcoma or hemangioendothelioma (blood vessels)
- Liposarcoma (adipose tissue)
- Glioma or astrocytoma (neurogenic connective tissue found in the brain)
- Myxosarcoma (primitive embryonic connective tissue)
- Mesenchymous or mixed mesodermal tumor (mixed connective tissue types)

4. **Lymphoma:** Malignant neoplasm derived from lymphocytes.

Lymphomas develop in the glands or nodes of the lymphatic system, a network of vessels, nodes, and organs (specifically the spleen, tonsils, and thymus) that purify bodily fluids and produce infection-fighting white blood cells, or lymphocytes. Unlike the leukemia's which are sometimes called "liquid cancers," lymphomas are "solid cancers." Lymphomas may also occur in specific organs such as the stomach, breast or brain. These lymphomas are referred to as extra nodal lymphomas. The lymphomas are sub-classified into two categories: Hodgkin lymphoma and Non-Hodgkin lymphoma. The presence of Reed-Sternberg cells in Hodgkin lymphoma diagnostically distinguishes Hodgkin lymphoma from Non-Hodgkin lymphoma.

5. **Melanoma:** Malignant neoplasm derived from melanocytes.

Myeloma is cancer that originates in the plasma cells of bone marrow. The plasma cells produce some of the proteins found in blood.

5.1. Leukemia

Leukemias ("liquid cancers" or "blood cancers") are cancers of the bone marrow (the site of blood cell production). The word leukemia means "white blood" in Greek. The disease is often associated with the overproduction of immature white blood cells. These immature white blood cells do not perform as well as they should, therefore the patient is often prone to infection. Leukemia also affects red blood cells and can cause poor blood clotting and fatigue due to anemia. Examples of leukemia include:

- Myelogenous or granulocytic leukemia (malignancy of the myeloid and granulocytic white blood cell series)
- Lymphatic, lymphocytic, or lymphoblastic leukemia (malignancy of the lymphoid and lymphocytic blood cell series)
- Polycythemia vera or erythremia (malignancy of various blood cell products, but with red cells predominating).

6. **Germ cell tumor:** Malignant neoplasm derived from germ cells.

The type components may be within one category or from different categories. Some examples are:

- adenosquamous carcinoma
- carcinosarcoma
- mixed mesodermal tumor
- teratocarcinoma

Nomenclature for benign neoplasms

In general, the name of a benign neoplasm often ends with - oma.

- **Examples:** Adenoma (benign neoplasm of glandular epithelium), fibroadenoma (benign neoplasm of the breast), and leiomyoma (benign neoplasm of smooth muscle).
- **Some exceptions:** Hepatoma (malignant neoplasm of liver), melanoma (malignant neoplasm of melanocytes), mesothelioma (malignant neoplasm of mesothelial cells), and seminoma (malignant germ cell neoplasm of testis).

Nomenclature for malignant neoplasms

In general, the name of a malignant neoplasm often ends with –carcinoma or –sarcoma.

- **Examples:** Adenocarcinoma (malignant neoplasm of glandular tissue), rhabdomyosarcoma (malignant neoplasm of skeletal muscle), and leiomyosarcoma (malignant neoplasm of smooth muscle).

Self-Check – 4	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Write the types of cancer (4pts)?

2. Explain the types of neoplasia neatly (6pts)?

Note: **Satisfactory rating – above 4 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

LG #52	LO # 6 – Assist in post-mortem examinations

Instruction sheet

This learning guide is developed to provide you the necessary information regarding the following **content coverage** and topics:

- Identifying reasons for conducting post mortem examinations.
- Identifying materials and equipment post mortem examination.
- Following procedures to conduct the post mortem examination is properly.
- Understanding post mortem changes.
- Observing and recording findings and abnormalities.

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, **you will be able to**:

- Identify reasons for conducting post mortem examinations.
- Identify materials and equipment post mortem examination.
- Follow procedures to conduct the post mortem examination is properly.
- Understand post mortem changes.
- Observe and record findings and abnormalities.

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5. Ask from your trainer the key to correction (key answers) or you can request your trainer to correct your work. (You are to get the key answer only after you finished answering the Self-checks).
6. If you earned a satisfactory evaluation proceed to “Operation sheets
7. Perform “the Learning activity performance test” which is placed following

"Operation sheets" ,

8. If your performance is satisfactory proceed to the next learning guide,
9. If your performance is unsatisfactory, see your trainer for further instructions or go back to "Operation sheets".

Information Sheet 1- Identify reasons for conducting post mortem examinations

1.1 Definition of Necropsy

A **necropsy** is the examination of an animal ***after death***. The purpose of a necropsy is typically to determine the cause of death, or extent of disease. This involves a careful process of dissection, observation, interpretation, and documentation. A thorough knowledge of normal anatomy is critical in distinguishing lesions from normal variations. Lesions are classified by their characteristics: location, distribution, size, shape, color and consistency.

1.1.1 Objectives of necropsy

- Necropsy requires observation of all parts of the carcass, dressing procedures, equipment, and facilities to prevent contamination of edible parts. The inspector must ensure that condemned carcasses and parts are disposed of safely.

The main reason for conducting post-mortem examination is:

i. Identify an Etiology and pathological lesions
ii. To evaluate the presence and extent of animals disease in patients and
iii. To evaluate the effectiveness of therapeutic procedures for the benefit of patient families, our staff, and the future practice of medicine.

Reason of Necropsy

- Necropsy contributes to the body of **scientific knowledge** by increasing our understanding of anatomy and physiology in health and disease.
- Autopsies are performed to determine the cause of death, for legal purposes, and for education and research. In the same way, clinical medicine contributes greatly to the necropsy process - a detailed clinical history can provide clues to direct the postmortem exam.

- Necropsies ***save lives***! They can alert us to the presence of diseases that may be transmissible to other animals (or humans!), and guide treatment decisions for at-risk individuals. Public health and regulatory veterinarians use the necropsy as a surveillance tool to monitor for emerging or foreign animal diseases. In a laboratory setting, postmortem examination is frequently used to help determine the safety and efficacy of new pharmaceuticals before they enter clinical trials.
- In some cases, necropsy findings can give comfort or closure to an owner, especially in the case of a seemingly sudden or unexplained death.
- Necropsy also serves important legal functions - photographic and written documentation of post-mortem findings is critical in cruelty and insurance investigations, and pathologists are occasionally called to testify as expert witnesses in such cases.

The difference between necrosis and post- mortem examination are summarized as:

S. No.	Post mortem autolysis	Necrosis
1	Absence of inflammatory reaction	Presence of inflammatory reaction
2	Autolytic changes are seen uniform throughout the tissue	Diffuse or focal adjacent living and dead tissues are seen

Self-Check – 1	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

3. Define the term ‘Necropsy’ (4pts)?

4. Write the reasons of conducting animals’ necropsy (4pts)?

Note: **Satisfactory rating – above 3 points** **Unsatisfactory - below 3 points**

You can ask your teacher the copy of the answer sheet

Information Sheet - 2- Identifying materials and equipment at post mortem examination

Use and maintain necropsy material and equipment

II. Post-mortem equipment

The minimum requirements for conducting a safe and satisfactory field post-mortem examination are as follows: –

- a curved knife for skinning
- a straight, pointed knife for dissection
- a pair of 25 cm rat-toothed forceps
- a pair of 15 cm pointed forceps
- a pair of 15 cm dissecting scissors
- a sterile scalpel and blades
- an enterotome
- a bone saw
- a large pair of bone forceps or bone-cutting shears
- an axe
- a sharpening stone and steel
- a spring balance to weigh to 10 kg
- a block and tackle
- some nylon rope
- A small gas or alcohol burner for sterilizing instruments. The kit may be packed in a stout, heavy, wooden box.

III. Specimen containers and sampling equipment

The following list of equipment is necessary for sampling:

- sterile disposable 5 ml syringes and sterile needles (20 g)
- culture tubes with sterile swabs
- microscope slides in box
- sterile Universal bottles
- sterile blood tubes
- plastic bags with closure tops (Whirlpack or Ziploc type)
- heavy duty plastic sealing tape
- 300 ml wide mouthed glass or plastic jars
- a measuring tape or ruler
- rubber or plastic gloves (and talc)
- aluminium foil
- a rabies kit (World Health Organization) (or drinking straw in a small jar of buffered glycerine)

- labels, string, waterproof marker pen/pencil.

IV. Transport equipment

For transportation, the following equipment is required:

- an insulated, plastic cooler box
- a leak-proof, screw cap, plastic containers
- absorptive packing material
- string and heavy duty plastic sealing tape
- sterile buffered 50% glycerine (see Appendix I for formulation)
- 'easy blood' (see Appendix I for formulation)
- 'Blue ice' freezer packs (pre-frozen).

V. Fixatives

The following fixatives are used:

- 10% buffered formalin
- 100% acetone for cytology
- 70% alcohol for parasites
- paradichlorobenzene.

VI. Disinfection materials

Disinfection materials include the following:

- a plastic bucket and brush
- a nailbrush, soap and towel
- borax
- 5% formalin
- Sodium hypochlorite (0.5%)
- 70% ethyl alcohol for disinfecting instruments.
- Sodium carbonates (5%).

VII. Additional equipment

The equipment listed below will also be very useful:

- field microscope (with mirror or car battery attachment for light source) for checking suspected anthrax cases before autopsy
- a portable centrifuge for serum separation
- a camera and film
- a notebook.

Self-Check – 2	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. List the main used material and tools during post-mortem examination (4pts).

2. Explain the methods of maintains of material after post-mortem examination (2pts).

Note: **Satisfactory rating – above 6 points** **Unsatisfactory - below 6 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 3 - Following procedures to conduct the post mortem examination

Post- mortem examination procedures

Post-mortem examination refers to inspection of carcass and organs by veterinary doctors after slaughtering and dressing. It should be carried out as soon as possible after dressing. The carcass is examined for evidence of bruises, injuries, or discoloration and diseased conditions. The veterinary doctor examines carcass and organs to ensure that carcass and organs are fit for human consumption. During inspection, care should to be taken not to contaminate the carcass and organs from diseased animals.

Post-mortem procedures

The procedures of post-mortem examination are:

Carcass examination

It will be necessary for the head, pluck (heart, pericardium, liver, spleen, and lung if possible), alimentary tract, genitalia and carcass (with neck and kidney) to be properly identified and presented separately for inspection. Since many viral and bacterial infections tend to be of a generalized nature, sound and professional meat inspection examination and judgement of the birds and carcass is of great importance.

- **Lungs** not removed during dressing procedures should be examined visually and by palpation in the thorax. To expose the lungs, two cuts above the lungs on the each side of the ribs should be made.
- **Head:** visual examination of the mouth, palate, eyes, lips and sinuses for icterus, sinusitis, crusting of eyelids and thrush (oral Candida infection)
- **Pluck**
  - ✓ Lungs: visual and palpation for haemorrhage, edema and pneumonia.
  - ✓ Heart: visual and palpation for haemorrhages; expose valves for endocarditis.

- ✓ Pericardium: visual, and incision if necessary; for pericarditis.
- ✓ Liver: visual and palpation; incise if necessary; for icterus, discolouration, adhesions, degeneration, abscess, fibrosis, inflammation and toxic condition.
- ✓ Spleen: visual and incision if necessary; for enlargement, haemorrhages and signs of febrile or septic conditions.
- ✓ Kidney: visual and palpation; for haemorrhages, degeneration, urate crystals.

- **Gastro-intestinal tract**
  - ✓ ***Oesophagus/proventriculus, gizzard***: visual and palpation; for foreign body penetration, impaction, inflammation and ulceration and parasitic conditions (nematode-Libyostrongylus) in glands of proventriculus.
  - ✓ ***Small intestine***: visual and palpation; impaction, volvulus, necrotic and catarrhal enteritis and small tapeworm (Houttynia).
  - ✓ ***Large intestine***: visual and palpation for faecal impaction, stones, inflammation and nematode (Condiostomum).

- **Reproductive organs** - visual for egg retention, rupture, prolapsed penis; Atrophic organs are found during non-breeding season.

Reason for carcass examination

- The carcass is observe for contamination, inadequate bleeding, bruising, haemorrhages, lacerations, fracture, dislocation, twisted legs, adhesions, icterus, arthritis, peritonitis, air sacculitis, abscesses (injection sites), foreign bodies.

Judgement: carcass should be:

- **Condemned** if affected with any of the following: death from any cause other than slaughter, extensive bruising and haemorrhages, general contamination, putrefaction, emaciation, edema, icterus, septicemia, aspergillosis, toxoplasmosis, malignant or multiple tumours, leucosis, poisoning.
- The parts of the carcass which show localized lesions may be **trimmed** and the rest of the carcass would then be approved.

4. **Body orifice examination**: this organs and mucous membrane were examined careful to diagnosis the availability of any pathological lesion. E.g. mucous membrane blood smear indicate an anthrax diagnosis.
5. **Blood examination:** the examined as reason identify a microbial infection and pathological changes in blood cells.
6. **Skin and pelage examination:** should be noted and an examination made for ulcers, shot holes, tooth marks and external injuries. The carcass should be turned over and any broken limb bones recorded. The upper eye is often removed by scavenging birds and the tail and muzzle may be damaged by foxes, jackals or dogs. An examination should be made for external parasites, noting abundance and location. Representative specimens should be collected and stored in 70% alcohol. Particular attention should be paid to predilection sites, e.g. around the muzzle, eyes, ears and genitalia, on the neck, brisket, tail switch, axillae, groins and hoof clefts. The mouth should be examined and the condition of the oral mucosa, tongue and teeth recorded. In cases of plant poisoning, parts of the plant may still be lodged between the teeth.

Self-Check – 3	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Write the reason of necropsy examination (2pts).

2. list and explain the procedure of post-mortem examination (4pts)

Note: **Satisfactory rating – above 3 points** **Unsatisfactory - below 3 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 4- Understand post mortem changes

Post-Mortem Changes

A. Somatic death

- Somatic death is the death of the body as a whole.
- When ***respiration*** and ***cardiac action*** have stopped, the animal is said to have undergone somatic death. After death, the cells undergo certain changes (post mortem changes), which a pathologist must have knowledge of to distinguish them from lesions found in disease. By a careful study of a postmortem changes one can determine the probable time of death and this is of great importance in medicolegal cases.
- Factors influencing the rate of postmortem necrosis
- ***Species of animal***: Pig-soft and moist muscle- rapid in onset, Horse-dry and firm muscle-slow in onset
- ***Organ involved:*** the degrees of the expression of postmortem changes vary from tissues to tissues. The presence of bacterial flora, enzyme secretions and the availability of moisture and substrates influence the rate of postmortem autolysis. Pancreas-high amount-rapid changes. Fibrous tissue-less amount-slow changes. Retina-most sensitive, separates from choroids. Adrenals, liver, testis-abdominal organs also show autolytic changes.

B. Putrefaction

Decomposition of tissues brought about by the protein splitting anaerobic saprophytic organisms, results in the formation of gas and variety of foul smelling substances ammonia, hydrogen sulphide, indol, skatol and putrescent amines-like “putriscience and cadaverine”. The tissue turns black or dark-green as a result of formation of iron sulphide from break down haemoglobin. The common putrefactive organisms are Clostridium spp. normally present in faeces, leads to pronounced postmortem changes in the body like gaseous distension, softening etc. Bacterial flora present in GIT and

respiratory tract bring about the post-mortem changes rapidly under favourable conditions.

Sequence of post-mortem changes

I. Algor mortis
II. Rigor mortis
III. Livor mortis- hypostatic congestion
IV. PM clotting of blood
V. Imbibition of hemoglobin
VI. Imbibition of bile
VII. PM desquamation
VIII. PM softening
IX. PM discoloration
X. PM distention
XI. PM displacement
XII. PM rupture of organ and tissue

1. **Algor mortis:** is cooling of the body. It commences at or before the stoppage of blood flow. The rate of cooling depends on the following factors:
    - External atmospheric temperature
    - Air currents
    - The thickness of hair coat or wool
    - Adiposity of the animal
    - Amount of fermentable ingesta in the digestive tract
    - Larger animals cool slowly; so also in sheep, with thick wool cooling occurs slowly. Limbs and other extremities cool more rapidly than the trunk.
2. **Rigor mortis:** is contraction of muscles after death.

This is a contraction of muscles after death so that the joints become stiff and body is rigid. Rigor mortis develops first in those muscles that are very active. E.g. heart, palpebral muscles, muscles of the head and neck. Gradually other muscles of the forelimbs, the trunk and the hind limbs, are affected in that order. It passes of also in this order, starting first in the head. Usually, rigor mortis appears in 1 to 8 hrs after death and may disappear from 20-30 hours. The following factors hasten the onset of rigor mortis.

- High atmospheric temperature
- Active exercise- hunting, fighting, racing or struggling

- Strychnine poisoning
- Tetanus

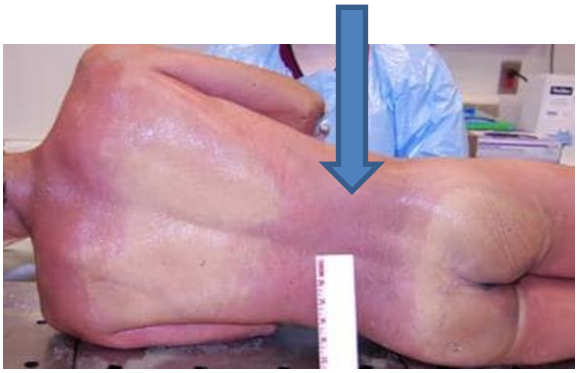
3. **Livor mortis:** Hypostatic congestion is, due to gravity, accumulation of blood in vessels of organs that are found on the lower side of the recumbent animal.
4. **Post-mortem clot:** is the coagulation of blood in the vessels after death. Chicken fat is the white clot while current jelly clot is the red clot seen in the clot. PM clot is formed after death of animal.
5. **Imbibition of hemoglobin:** PM staining is pinkish discolouration of endothelium of larger vessels due to haemoglobin (liberated from lysed erythrocytes) after death.
6. **PM imbibition of bile:** is the yellow pigmentation of the tissue occurring in the vicinity of gall bladder.
7. **PM softening**: is softening of tissues, after death, by the action of autolytic enzymes of the cells and the proteolytic ferments of the saprophytes and infecting bacteria.
8. **PM discoloration**: Pseudomelanosis coli is staining (blackish / greenish discolouration) of intestines due to formation of iron sulphide (H2s + Fe from Hb = Iron sulphide) after death of animals.
9. **PM bloats / PM emphysema** is accumulation of gas in the rumen and intestines due to fermentation of food after death.
10. **PM displacement of organs:** This may occur following handling of carcass by rolling etc.
11. **PM rupture of organ and tissue:** This may be attributed to softening and handling but devoid of any inflammatory reaction.

In equine practice, stud fee is payable only on the birth of a live foal. So, the veterinarian may be required to certify as to whether a foal was born alive or dead. The two criteria to be looked for are:

- Does the lung float in water? If it floats the foal was born alive since presence of air renders the lung buoyant. Air can be present in lung only if the animal had breathed and breathing can occur only if the foal was born alive.

- Did it suckle? Presence of milk or curds in the stomach is valid evident that the foal was alive at birth and had suckled**.**

Figure: Putrefaction, **emphysema** and **livor mortis** lesion appeared at post-mortem



Self-Check – 4	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Define what is death?

2. List and explain the sequence of post-mortem changes

Note: **Satisfactory rating – above 3 points** **Unsatisfactory - below 3 points**

You can ask your teacher the copy of the answer sheet

Information Sheet 5- Observe and record findings and abnormalities.

5.1 Record an observed abnormality

After all studies are completed, a detailed report is prepared that describes the autopsy procedure and microscopic findings, gives a list of medical diagnoses, and a summary of the case. The report emphasizes the relationship or correlation between:

- Slaughter method (Halal or Christ).
- Carcass condition (health or pathological status)
- Body orifices (health or pathological changes)
- Judgement categories (Approved as fit for human consumption, totally unfit for human consumption and partially condemned).
- Clinical findings (lesion)
- Laboratory result of post- mortem
- Radiology findings and
- Pathologic findings (those made from the autopsy).

Findings are documented, either in

- Writing,
- Photographs or both.

The information is then synthesized into a “best guess” interpretation of what the physical findings likely represent. Some findings are easily interpreted (hole in the stomach wall = gastric ulcer), while others may involve a list of likely differentials. Supplemental diagnostic tests, such as histopathology, microbiology, or serology, frequently aid in discriminating among lists of differentials.

Self-Check – 5	Written test

Name…… ID…… Date…….

Directions: Answer all the questions listed below. Examples may be necessary to aid some explanations/answers.

Test I: Short Answer Questions

1. Write the main record during you examined post-mortem (4pts)

Note: **Satisfactory rating – above 2 points** **Unsatisfactory - below 2 points**

You can ask your teacher the copy of the answer sheet

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This Teaching, Training and Learning Materials (TTLM) were developed on June 2021 at Adama, Pan Africa Hotel.

Operation Sheet 1– Make pathological lesion finding in abattoir

Objective: Finding pathological lesion in abattoir

Material: Necropsy (liver, lung, offal and heart tissue), table, knife, light, potable water, refrigerator, icebox, glove, eye google, gown

To carry out pathological lesions finding in tissues, 5 hours

The step finding pathological lesion is indicated as follow:

1. Viewing, incision, palpation and olfaction techniques.
2. Classifying the lesions into one of two major categories - acute or chronic.
3. Establishing whether the condition is localized or generalized, and the extent of systemic changes in other organs or tissues.
4. Determining the significance of primary and systemic pathological lesions and their relevance to major organs and systems, particularly the liver, kidneys, heart, spleen and lymphatic system.
5. Coordinating all the components of postmortem findings to make a final diagnosis.
6. Submitting the samples to the laboratory for diagnostic support, if abattoir has holding and refrigeration facilities for carcasses under detention.

LAP TEST	Performance Test

Name…….. ID…… Date……

Instructions: Given necessary templates, tools and materials you are required to perform the following tasks within 2 hour. The project is expected from each student to do it.

Time started: _____ Time finished: _____

Task-1: Perform successive 1/2 dilutions, 5 times

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