

Here are the complete answers to all "Q.5" long-answer questions from the 2024, 2023, 2022, 2019, and 2018 papers.



## 2024 Exam (Q.5)

### 5.1 Enlist egg borne diseases of poultry and describe etiology, symptoms, pathology and diagnosis of avian encephalomyelitis

#### Egg-Borne Diseases of Poultry:

- **Viral:** Avian Encephalomyelitis, Lymphoid Leucosis, Chicken Infectious Anemia, Avian Adenovirus (Inclusion Body Hepatitis).
- **Bacterial:** Pullorum Disease (*Salmonella Pullorum*), Fowl Typhoid (*Salmonella Gallinarum*), Mycoplasmosis (*M. gallisepticum*).

#### Avian Encephalomyelitis (AE)

- **Etiology:** Caused by a **Picornavirus** (genus *Hepatovirus*). It is a non-enveloped, single-stranded RNA virus.
- **Symptoms:**
  - **In Young Chicks (1-3 weeks):** The disease is also known as "Epidemic Tremor." Symptoms are primarily neurological, including dullness, ataxia (incoordination), leg weakness, paralysis, and characteristic **rapid tremors of the head and neck**.
  - **In Laying Hens:** The only signs are a transient **drop in egg production** (10-20%) and a significant decrease in hatchability.
- **Pathology (Lesions):**
  - **Gross Lesions:** There are **no specific or pathognomonic gross lesions** in any organ.
  - **Microscopic Lesions:** These are diagnostic.
    - **CNS:** The key lesion is **non-purulent encephalomyelitis**. This includes perivascular cuffing (lymphocyte infiltration) and gliosis (microglial proliferation). The most pathognomonic finding is **central chromatolysis**, which is the degeneration and loss of Nissl substance in neurons, especially in the brainstem and spinal cord.
    - **Other Organs:** Diffuse lymphoid aggregates (hyperplasia) are commonly found in the muscle of the **proventriculus and gizzard** and in the **pancreas**.
- **Diagnosis:**
  - **Presumptive:** Based on characteristic neurological signs (tremors) in young, un-vaccinated chicks.
  - **Histopathology:** The presence of characteristic microscopic lesions (central chromatolysis, perivascular cuffing) in the CNS is highly indicative.
  - **Virus Isolation:** Inoculation of the virus into embryonated chicken eggs.
  - **Serology:** ELISA or AGPT can detect antibodies in breeder flocks, confirming exposure or vaccination status.

### 5.2 Describe the common diseases of rabbit. Enumerate the causes of pregnancy toxemia in rabbit

#### Common Diseases of Rabbit:

- **Bacterial:**

- **Pasteurellosis ("Snuffles"):** Caused by *Pasteurella multocida*. It is the most common bacterial disease, causing rhinitis (snuffles), conjunctivitis, pneumonia, otitis media (leading to "wry neck"), and abscesses.
- **Tyzer's Disease:** Caused by *Clostridium piliforme*. (See 5.3 below).
- **Viral:**
  - **Myxomatosis:** A highly fatal disease caused by a *Poxvirus*. It is transmitted by insects and causes severe swelling of the head, face, and anogenital region (myxomatous masses).
  - **Rabbit Hemorrhagic Disease (RHD):** A highly contagious and fatal *Calicivirus* that causes massive liver necrosis and widespread hemorrhages.
- **Parasitic:**
  - **Coccidiosis (Hepatic):** Caused by *Eimeria stiedae*, which leads to the formation of white, nodular streaks in the liver due to bile duct hyperplasia.
  - **Coccidiosis (Intestinal):** Caused by other *Eimeria* species, leading to enteritis and diarrhea.
  - **Ear Canker:** Caused by the mite *Psoroptes cuniculi*, leading to thick, crusty exudates in the ear canal.
- **Non-Infectious:**
  - **Uterine Adenocarcinoma:** The most common neoplasm in female rabbits.
  - **Malocclusion:** Overgrowth of incisors or molars, leading to an inability to eat.

#### **Causes of Pregnancy Toxaemia in Rabbit:**

Pregnancy toxaemia (ketosis) is a fatal metabolic disorder of late-gestation rabbits. The primary cause is a state of **severe negative energy balance**, where the doe's energy demands (from the large litter) far exceed her energy intake.

This imbalance is triggered by:

1. **Obesity:** This is the most important predisposing factor. Obese does are often already in a state of hepatic lipidosis.
2. **Anorexia/Fasting:** A sudden decrease in food intake in the last week of gestation. This can be caused by:
  - Stress (e.g., transport, new environment).
  - Any other illness or pain (e.g., dental disease).
  - Unpalatable feed or a sudden change in feed.
3. **Large Litter Size:** Places a massive energy demand on the doe.

This negative energy balance forces the body to **mobilize large amounts of fat**. The liver becomes overwhelmed by these fatty acids, leading to **severe hepatic lipidosis** (fatty liver). The liver cannot function properly and overproduces ketones, leading to ketoacidosis, CNS depression, and death.

### **5.3 Briefly describe about Tyzer's Disease**

- **Etiology:** *Clostridium piliforme*, a motile, spore-forming, Gram-negative, obligate intracellular bacterium.
- **Hosts:** It is an acute, fatal disease primarily affecting foals, rabbits, and rodents (mice, gerbils, hamsters). It is often triggered by stress, poor sanitation, or immunosuppression.
- **Pathology (Lesions):** The disease is characterized by a "classic triad" of lesions affecting three primary organs:
  1. **Hepatitis:** The liver is enlarged and studded with numerous, multifocal, pinpoint, pale-yellow or white **necrotic foci** ("miliary necrosis").

2. **Enterocolitis:** Severe hemorrhagic and necrotic inflammation, particularly of the lower ileum, cecum, and colon (hemorrhagic typhlitis).
3. **Myocarditis:** Necrotic foci may also be found in the heart muscle.
- **Microscopic Lesion:** The pathognomonic finding is the presence of long, filamentous bacilli arranged in characteristic "**haystack**" or "**pickup sticks**" patterns. These are found *within* viable hepatocytes, typically at the margin of the necrotic foci. They are difficult to see with H&E stain and require a silver stain (like Warthin-Starry) to be visualized.



## 2023 Exam (Q.5)

### 5.1 Write down the etiology, mode of transmission, pathogenesis and lesions and diagnosis of Rabies in canines

- **Etiology:** Rabies virus, a neurotropic **Lyssavirus** from the family Rhabdoviridae. It is a bullet-shaped, enveloped, single-stranded RNA virus.
- **Mode of Transmission:** The virus is transmitted through the **saliva** of an infected animal, almost always via a **bite** that breaks the skin.
- **Pathogenesis:**
  1. **Replication:** The virus first replicates locally in muscle cells at the site of the bite.
  2. **Neuroinvasion:** It enters peripheral nerves via neuromuscular junctions.
  3. **Axonal Transport:** The virus travels via **retrograde axoplasmic flow** (moving backward along the nerve) to the spinal cord.
  4. **Ascension:** It rapidly ascends the spinal cord to the brain, where it causes a progressive, fatal **polioencephalomyelitis**. It has a high affinity for the limbic system, hippocampus, and cerebellum.
  5. **Centrifugal Spread:** From the brain, the virus travels centrifugally (outward) along autonomic nerves to other organs, most importantly the **salivary glands**, where it replicates and is shed in the saliva.
- **Lesions:**
  - **Gross Lesions:** There are **no specific or diagnostic gross lesions** in the brain or any other organ. Non-specific findings like self-inflicted trauma may be present.
  - **Microscopic Lesions:** The characteristic lesion is a **non-purulent meningoencephalomyelitis** (perivascular cuffing, gliosis, and neuronal degeneration). The pathognomonic finding is the presence of **Negri bodies**: well-defined, eosinophilic, **intracytoplasmic inclusion bodies** in neurons. In canines, Negri bodies are most reliably found in the **hippocampus**.
- **Diagnosis:**
  - **Gold Standard:** The **Fluorescent Antibody Test (FAT)** on fresh, un-fixed brain tissue (hippocampus and brainstem). This test detects viral antigens.
  - **Histopathology:** Demonstration of Negri bodies in brain tissue (less sensitive than FAT).
  - **PCR:** Detects viral RNA; highly sensitive.

### 5.2 Write down the etiology, mode of transmission, pathogenesis, lesions and diagnosis of FMD in cattle

- **Etiology:** Foot and Mouth Disease (FMD) Virus, an **Aphthovirus** from the family Picornaviridae. There are 7 main serotypes: **O, A, C, SAT 1, SAT 2, SAT 3, and Asia 1.**
- **Mode of Transmission:** FMD is one of the most contagious diseases known. It spreads via:
  - **Inhalation:** Aerosol transmission is a major route.
  - **Direct Contact:** Contact with infected animals or their secretions (saliva, vesicle fluid).
  - **Fomites:** Contaminated equipment, vehicles, feed, and people.
- **Pathogenesis:**
  1. **Entry:** Primary infection occurs via inhalation or ingestion.
  2. **Replication:** The virus replicates in the pharyngeal mucosa.
  3. **Viremia:** A rapid viremia (virus in the bloodstream) occurs, spreading the virus throughout the body.
  4. **Epitheliotropism:** The virus has a strong affinity for **epithelial tissue**, localizing in the mouth, feet, and mammary glands.
  5. **Vesicle Formation:** It causes ballooning degeneration of epithelial cells, leading to the formation of vesicles (blisters).
- **Lesions:**
  - **Gross Lesions:**
    - **Vesicles (Blisters):** The characteristic lesion. Found on the **tongue, dental pad, gums, hard palate, coronary band, and in the interdigital space.**
    - **Erosions:** Vesicles rupture within 24-48 hours, leaving painful, red, raw erosions. This causes profuse ropy salivation (drooling) and severe lameness.
    - **"Tiger Heart":** In young calves, the virus is myocardiotropic, causing acute heart failure. The myocardium shows greyish-yellow necrotic streaks, known as **tigroid heart.**
  - **Microscopic Lesions:** Intraepidermal vesicles, ballooning degeneration of epithelial cells (stratum spinosum), and (in calves) severe myocardial necrosis.
- **Diagnosis:**
  - **Field:** Based on clinical signs (vesicles, drooling, lameness) in multiple cloven-hoofed animals.
  - **Laboratory:**
    - **ELISA:** Detects viral antigen from vesicle fluid or antibodies in serum.
    - **PCR:** Rapidly detects and serotypes the viral RNA.
    - **Virus Isolation:** Inoculation onto cell cultures.

### 5.3 Write down the etiology, mode of transmission, pathogenesis and gross and microscopic lesions of IBD in poultry

- **Etiology:** Infectious Bursal Disease (IBD) Virus, a **Birnavirus**. It is a highly resistant, non-enveloped, double-stranded RNA virus.
- **Mode of Transmission:** The virus is highly contagious and spreads horizontally via the **fecal-oral route**. It is very stable and persists in the environment, spreading via contaminated feed, water, and fomites.
- **Pathogenesis:**
  1. **Ingestion:** The virus is ingested and absorbed in the gut.

2. **Replication:** It undergoes initial replication in gut-associated macrophages and lymphocytes.
  3. **Viremia:** A viremia spreads the virus throughout the body.
  4. **Tropism:** The virus has a specific tropism for the **Bursa of Fabricius**, where it targets and destroys **immature B-lymphocytes**.
  5. **Immunosuppression:** This massive destruction of B-cells leads to severe, often permanent, immunosuppression, making the birds susceptible to secondary infections.
- **Gross Lesions:** The lesions in the **Bursa of Fabricius** are sequential:
    - **Acute Phase (3-5 days post-infection):** The bursa is **severely enlarged**, swollen, edematous, and may show hemorrhages. It has a "creamy" or "gelatinous" appearance.
    - **Chronic Phase (after 8-10 days):** The bursa undergoes progressive, **severe atrophy**, shrinking to a small, fibrotic remnant.
    - **Other Lesions:** Hemorrhages in the **thigh and breast muscles** (brush-like hemorrhages), swollen kidneys, and dehydration.
  - **Microscopic Lesions:**
    - Severe, diffuse **necrosis of lymphocytes** within the bursal follicles.
    - Infiltration of heterophils and macrophages.
    - Follicular depletion, edema, and hemorrhage. In the chronic stage, there is follicular atrophy and fibrosis.



## 2022 Exam (Q.5)

### 5.1 a) Enlist important mycobacterial diseases in Cattle. b) Discuss pathogenesis and pathology of Bovine tuberculosis.

#### a) Important Mycobacterial Diseases in Cattle:

1. **Bovine Tuberculosis:** Caused by *Mycobacterium bovis*.
2. **Paratuberculosis (Johne's Disease):** Caused by *Mycobacterium avium* subspecies *paratuberculosis*.
3. **Avian Tuberculosis:** Caused by *Mycobacterium avium* subspecies *avium*. (Cattle can be infected, but it's less common).

#### b) Pathogenesis and Pathology of Bovine Tuberculosis

- **Pathogenesis:**
  1. **Transmission:** Primarily via **inhalation** of aerosolized droplets from an infected animal. Ingestion (e.g., in calves drinking infected milk) is another route.
  2. **Primary Infection:**
    - **Inhalation:** Bacteria are phagocytosed by alveolar macrophages in the lungs.
    - **Replication:** *M. bovis* survives and replicates intracellularly, spreading to the regional lymph nodes (tracheobronchial and mediastinal).
    - **Ghon Complex:** The initial small lesion in the lung, plus the affected draining lymph node, is called the "**Ghon complex**".
  3. **Immune Response:** A **Type IV (cell-mediated) hypersensitivity** is mounted. Macrophages and T-lymphocytes wall off the infection, forming a **granuloma (tubercle)**.
  4. **Lesion Formation:** The center of the tubercle becomes necrotic (**caseous**

- necrosis**) and often undergoes **calcification**.
5. **Progression:** If the immune system fails, the tubercles enlarge, coalesce, and can spread (disseminate) via the bloodstream or airways (**miliary tuberculosis**).
- **Pathology (Lesions):**
    - **Gross Lesions:** The characteristic lesion is the **tubercle**. This is a firm, greyish-white, nodular granuloma. When cut, it reveals a yellowish, cheesy, **caseous necrotic center** that is often **calcified** (feels gritty when cut).
      - **Lungs & Lymph Nodes:** These are the most common sites.
      - **"Pearl Disease":** A classic presentation where miliary tubercles form grape-like clusters on serosal surfaces (pleura, peritoneum).
    - **Microscopic Lesions:** The tubercle has a classic structure:
      - A central core of **caseous necrosis** and calcification.
      - Surrounded by a zone of **epithelioid macrophages** and **Langhans giant cells**.
      - An outer rim of **lymphocytes**, **plasma cells**, and a **fibrous capsule**.
      - **Acid-fast stains** (Ziehl-Neelsen) can be used to visualize the red-staining mycobacteria within the lesions.

## 5.2 a) What are different forms of New Castle disease. b) Discuss in details the pathology of Doyle's form of New Castle disease

**a) Different Forms of New Castle Disease (Ranikhet Disease):** The disease is caused by Avian Paramyxovirus-1 (APMV-1). Strains are classified by their pathogenicity (pathotypes):

1. **Velogenic:** Highly pathogenic, causing high mortality.
  - **Viscerotropic Velogenic (VVND):** Also known as **Doyle's form**. Targets the digestive tract.
  - **Neurotropic Velogenic (NVND):** Targets the respiratory and nervous systems.
2. **Mesogenic:** Moderately pathogenic. Causes respiratory signs and low mortality, but neurological signs in young birds.
3. **Lentogenic:** Low pathogenicity. Cause mild or subclinical respiratory infection. (Used for live vaccines, e.g., LaSota, B1).
4. **Asymptomatic:** Subclinical enteric infection.

### b) Pathology of Doyle's Form (Viscerotropic Velogenic ND)

Doyle's form (VVND) is the most acute and fatal form, characterized by severe **hemorrhagic and necrotic lesions** in the digestive tract.

- **Pathogenesis:** The virus is highly **endotheliotropic**, meaning it targets and destroys the endothelial cells lining blood vessels, leading to widespread vascular damage, hemorrhage, and tissue necrosis.
- **Gross Lesions:**
  - **Proventriculus:** The most characteristic lesion is **hemorrhage**, especially on the mucosal papillae at the **junction of the proventriculus and gizzard**.
  - **Intestine:** Severe, multifocal, **necrotic and hemorrhagic foci** in the intestinal lymphoid tissue, especially the **cecal tonsils** and Peyer's patches. These can ulcerate.
  - **Other:** Hemorrhages on serosal surfaces, cyanosis of the comb and wattles, and splenic necrosis.
- **Microscopic Lesions:**

- Severe **necrosis of endothelial cells** in small blood vessels.
- Widespread **hemorrhage, thrombosis**, and **necrosis** of lymphoid tissue (cecal tonsils, bursa, spleen).
- Focal necrosis of the intestinal epithelium overlying lymphoid patches.

### 5.3 a) Name different serotypes of FMD Virus. b) Discuss in details the pathology of FMD in cattle

a) **Different Serotypes of FMD Virus:** There are seven immunologically distinct serotypes:

1. **O**
2. **A**
3. **C**
4. **SAT 1** (Southern African Territories 1)
5. **SAT 2**
6. **SAT 3**
7. **Asia 1**

b) **Pathology of FMD in Cattle:** (*This is a repeat of question 5.2 from 2023*).

- **Pathogenesis:** FMD is an epitheliotropic virus. It enters via the pharynx, causes a viremia, and then localizes in the epithelium of the mouth, feet, and mammary glands, where it causes vesicles.
- **Gross Lesions:**
  - **Vesicles (Blisters):** The hallmark of the disease. They appear on the **tongue**, dental pad, gums, **coronary band**, and in the **interdigital space**.
  - **Erosions:** The vesicles rupture, leaving painful, red, raw erosions, which leads to profuse ropy salivation (drooling) and severe lameness.
  - **"Tiger Heart":** In young calves, the virus is myocardiotropic, causing acute fatal myocarditis. The heart muscle shows greyish-yellow necrotic streaks, known as **tigroid heart**.
- **Microscopic Lesions:**
  - **Epithelium:** Ballooning degeneration of epithelial cells (acantholysis) in the stratum spinosum, leading to the formation of fluid-filled intraepidermal vesicles.
  - **Myocardium (Calves):** Acute, multifocal necrosis of cardiac muscle fibers with a mild inflammatory infiltrate.



## 2019 Exam (Q.5)

### 5.1 Describe etiology, pathology, gross and microscopic lesions and diagnosis of Anthrax in Cattle

- **Etiology:** *Bacillus anthracis*, a large, Gram-positive, spore-forming, non-motile rod.
- **Pathology (Pathogenesis):**
  1. **Infection:** Usually via ingestion of spores from contaminated soil.
  2. **Germination:** Spores are phagocytosed by macrophages and germinate in regional lymph nodes.
  3. **Septicemia:** The vegetative bacteria multiply rapidly, enter the bloodstream, and cause a massive **septicemia**.
  4. **Toxin Production:** The bacteria produce a potent exotoxin complex (Protective

Antigen, Edema Factor, Lethal Factor), which causes widespread vascular damage, edema, hemorrhage, and tissue necrosis, leading to shock and rapid death.

- **Gross Lesions:**
  - **NOTE:** Necropsy is **strongly contraindicated** due to the risk of human infection and environmental contamination with spores.
  - **External:** Sudden death is common. There is often **bloody, non-clotting discharge** from all natural orifices (nose, mouth, anus). Rigor mortis is often absent or incomplete.
  - **Internal:** If accidentally opened, the carcass shows:
    - **Blood:** Dark, thick, "tarry" blood that **fails to clot**.
    - **Spleen:** The most characteristic lesion is a massive **splenomegaly**. The spleen is dark, "pulpy," and has a "blackberry jam" consistency.
    - **Hemorrhages:** Widespread **petechial and ecchymotic hemorrhages** on all serosal surfaces.
- **Microscopic Lesions:**
  - Widespread hemorrhage, edema, and necrosis in all tissues.
  - Massive numbers of large, **blue-staining (basophilic) bacilli** are visible in blood vessels (capillaries) and tissues.
  - **M'Fadyean's Reaction:** A stain of a blood smear reveals the large, square-ended rods surrounded by a distinct **pink, polypeptide capsule**.
- **Diagnosis:**
  - **Blood Smear:** A thin smear from a superficial ear vein (the safest method) stained with M'Fadyean's (polychrome methylene blue) stain to demonstrate the capsule.
  - **Ascoli's Test:** A thermoprecipitin test that detects anthrax antigens in tissue samples.
  - **Bacterial Culture:** Culture from the blood.

## 5.2 Describe etiology, pathology, gross and microscopic lesions and diagnosis of Rabies in Dog

*(This is a repeat of question 5.1 from 2023).*

- **Etiology:** Rabies virus (a **Lyssavirus**, family Rhabdoviridae).
- **Pathology (Pathogenesis):** Transmitted by a bite. The virus replicates in muscle, enters peripheral nerves, and travels via **retrograde axoplasmic flow** to the brain, causing fatal encephalomyelitis. It then spreads to the salivary glands.
- **Gross Lesions:** **None** are specific to rabies.
- **Microscopic Lesions:**
  - Non-purulent meningoencephalomyelitis (perivascular cuffing, gliosis).
  - The pathognomonic lesion is the **Negri body**: an eosinophilic, **intracytoplasmic inclusion body** in neurons, especially in the **hippocampus** of dogs.
- **Diagnosis:**
  - **Gold Standard: Fluorescent Antibody Test (FAT)** on fresh brain tissue.
  - Histopathology to find Negri bodies.

## 5.3 Describe etiology, pathology, gross and microscopic lesions and diagnosis of Avian influenza



- **Etiology:** Avian Influenza Virus (AIV), a Type A **Orthomyxovirus**. Strains are classified by their hemagglutinin (H) and neuraminidase (N) surface proteins (e.g., H5N1, H7N9).
- **Pathology:**
  - **Low Pathogenic (LPAI):** Infection is localized to the respiratory or GI tract, causing mild disease.
  - **High Pathogenic (HPAI):** (Usually H5 or H7 subtypes). These strains cause a rapidly fatal, **systemic disease**. The virus is **endotheliotropic**, replicating in and destroying vascular endothelial cells, leading to massive edema, hemorrhage, and multi-organ failure.
- **Gross Lesions (HPAI):**
  - **Edema and Cyanosis:** Severe, dark blue-to-purple discoloration and swelling of the **comb and wattles**.
  - **Hemorrhages:** Widespread **petechial and ecchymotic hemorrhages** throughout the body, especially on the **proventriculus**, in subcutaneous tissue, on serosal surfaces, and in body fat.
  - **Necrosis:** Multifocal necrotic foci in the **liver, spleen, kidney, and pancreas**.
- **Microscopic Lesions (HPAI):**
  - Severe **vasculitis** and **endothelial necrosis**.
  - Widespread edema, hemorrhage, and **thrombosis**.
  - Severe, multifocal necrosis in multiple organs, especially the **pancreas, heart, and brain**.
- **Diagnosis:**
  - **Virus Isolation:** Inoculation of oropharyngeal or cloacal swabs into embryonated chicken eggs.
  - **PCR (RT-PCR):** The most common, rapid test. It detects viral RNA and can identify the H5 and H7 subtypes.
  - **Serology:** AGID or ELISA to detect antibodies (used for flock surveillance).



## 2018 Exam (Q.5)

### 5.1 Write etiology, pathogenesis, clinical signs, gross lesions, microscopic lesions and diagnosis of Ranikhet disease

*(This is a repeat of question 5.2 from 2022).*

- **Etiology:** Avian Paramyxovirus-1 (APMV-1), also known as Newcastle Disease Virus.
- **Pathogenesis:** Infection is via inhalation or ingestion. The virus replicates in the respiratory or GI tract, causes a viremia, and then localizes to target organs (gut, CNS, respiratory tract) depending on the strain.
- **Clinical Signs:**
  - **Viscerotropic (Doyle's):** Sudden death, watery-green diarrhea, prostration.
  - **Neurotropic:** Respiratory distress followed by neurological signs: **torticollis (twisting of the neck)**, paralysis of legs/wings, tremors.
  - **Lentogenic:** Mild respiratory signs, coughing, sneezing.
- **Gross Lesions (Velogenic forms):**

- **Proventriculus: Hemorrhages** at the junction with the gizzard.
- **Intestine:** Severe **hemorrhagic and necrotic lesions** in lymphoid tissue, especially the **cecal tonsils**.
- **Respiratory:** Tracheal inflammation and congestion.
- **Microscopic Lesions:** Necrosis of lymphoid tissues, endothelial cell necrosis, and in neurotropic forms, a non-purulent encephalomyelitis.
- **Diagnosis:**
  - **Virus Isolation:** In embryonated chicken eggs.
  - **Serology:** Hemagglutination Inhibition (HI) test.
  - **PCR (RT-PCR):** To detect and pathotype the virus.

## 5.2 Enlist the bacterial disease of domestic animals. Write etiology, pathogenesis, clinical signs, gross lesions and microscopic lesions [of] Anthrax.

### Bacterial Diseases of Domestic Animals (Examples):

- **Anthrax** (*Bacillus anthracis*)
- **Clostridial Diseases:** Black Quarter (*C. chauvoei*), Tetanus (*C. tetani*), Enterotoxemia (*C. perfringens*)
- **Tuberculosis** (*Mycobacterium bovis*)
- **Paratuberculosis / Johne's Disease** (*M. avium* ssp. *paratuberculosis*)
- **Brucellosis (Bang's Disease)** (*Brucella abortus*)
- **Hemorrhagic Septicemia** (*Pasteurella multocida*)
- **Salmonellosis** (*Salmonella* spp.)
- **Leptospirosis** (*Leptospira* spp.)
- **Glanders** (*Burkholderia mallei*)
- **Strangles** (*Streptococcus equi*)
- **Lumpy Jaw** (*Actinomyces bovis*)

**Anthrax** (*This is a repeat of question 5.1 from 2019*).

- **Etiology:** *Bacillus anthracis*.
- **Pathogenesis:** Ingestion of spores -> germination in lymph nodes -> massive septicemia -> release of edema and lethal toxins -> vascular damage, hemorrhage, shock, and death.
- **Clinical Signs:** **Sudden death** is the most common sign. Also, high fever, ataxia, and **bloody discharge from natural orifices**.
- **Gross Lesions:** (Necropsy contraindicated)
  - Dark, "tarry," **non-clotting blood**.
  - Massive **splenomegaly** (spleen is dark and "pulpy" or "blackberry jam"-like).
  - Widespread **petechial hemorrhages** on all serosal surfaces.
- **Microscopic Lesions:**
  - Massive numbers of large, **blue-staining bacilli** in blood vessels.
  - **M'Fadyean's Reaction:** (Blood smear) Demonstrates the pink, polypeptide capsule.
  - Widespread tissue hemorrhage, edema, and necrosis.

## 5.3 Write etiology, pathogenesis, clinical signs, gross lesions,

## microscopic lesions and diagnosis of Rabies in wild animals

*(This is a repeat of question 5.1 from 2023 and 5.2 from 2019. The principles are identical).*

- **Etiology:** Rabies virus (a **Lyssavirus**). Key wildlife reservoirs include raccoons, skunks, foxes, and bats.
- **Pathogenesis:** Transmitted by a **bite**. The virus replicates locally in muscle, enters peripheral nerves, and travels via **retrograde axoplasmic flow** to the brain. It causes a fatal encephalomyelitis and then spreads to the salivary glands, making the animal infectious.
- **Clinical Signs:** Highly variable. The most common sign is a **change in behavior**.
  - Nocturnal animals are seen during the day.
  - Loss of fear of humans.
  - **"Furious form"**: Aggression, agitation, attacking inanimate objects.
  - **"Dumb/Paralytic form"**: Ataxia, lethargy, paralysis (especially of the throat, causing drooling).
- **Gross Lesions:** **None** are specific to rabies. Non-specific findings include trauma or emaciation.
- **Microscopic Lesions:**
  - Non-purulent meningoencephalomyelitis.
  - **Negri bodies** (eosinophilic, intracytoplasmic inclusions in neurons). In wild animals, they are most reliably found in the **cerebellum (Purkinje cells)** or brainstem, in addition to the hippocampus.
- **Diagnosis:**
  - **Gold Standard: Fluorescent Antibody Test (FAT)** on fresh brain tissue (brainstem and cerebellum are critical).
  - Histopathology to find Negri bodies.