PATHOLOGY RESPIRATORY SYSTEM

- Pathology of upper respiratory passage
  - Nasal polyps
  - Nasal granuloma
  - Tracheitis
  - Bronchitis
- Pathology of lungs
  - Atelectasis
  - Emphysema
  - Pulmonary oedema
  - Pneumonia
  - Pulmonary adenomatosis
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- Pathology of air sacs
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- Pathology of pleura
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PATHOLOGY OF UPPER RESPIRATORY TRACT

In many infectious diseases, there is inflammation of mucosa of upper respiratory passage leading to nasal discharge which is catarrhal, purulent or fibrinous depending on the type of infection. The infection may extend to lower parts of respiratory tract and reach in lungs causing pathological alterations. Rhinitis is the inflammation of nasal mucosa (Fig. 15.1). Sinusitis is the inflammation of sinuses e.g. Frontal sinusitis in dehorned cattle. The larvae of botfly Oestrus ovis enters in nasal passage and migrate upto frontal sinuses and turbinate bones and cause mucopurulent inflammation. Similarly leeches (Dinobdella ferox) is known to cause nasal cavity inflammation in domestic animals and suck blood. Rhinitis caused by Bordetella bronchiseptica in pigs and characterized by mucopurulent exudate, disappearance of nasal septum, retarded growth of snout and plugging of passage by solidified exudate and dead tissue. This condition is known as porcine atrophic rhinitis. Epistaxis is bleeding from nasal passage due to trauma, neoplasm and ulcerative lesions as a result of infections. Pharyngitis is the inflammation of pharynx while laryngitis is the inflammation of larynx.

NASAL POLYPS

Nasal polyps are the inflammatory condition of respiratory mucosa resembling neoplastic growth caused by fungus and characterized by formation of new growth simulating benign neoplasm in nasal passage.

Etiology

• Rhinosporidium sceberi, a fungus most commonly prevalent in southern India.

Macroscopic features

• Formation of a single polyp in respiratory mucosa, pedunculated, elongated, fills nasal cavity.
• Cauliflower like growth may cause bleeding.

Microscopic features

• Fibrous covering by mucous membrane and heavily infiltrated by neutrophils, lymphocytes, eosinophils, macrophages around fungus.

NASAL GRANULOMA

Nasal granuloma is the granulomatous inflammation of respiratory mucosa in nasal cavity caused by blood flukes and characterized by the presence of granulomatous growth filling the nasal passage causing obstruction (Fig. 15.2 & 15.3).

Etiology

• Schistosoma nasalis, a blood fluke.
• Type II hypersensitivity reaction of nasal mucosa to plant pollens, fungi, mites etc (Fig. 15.4).

Macroscopic Features

• Nasal pruritus
• Small tiny nodules on nasal mucosa later becomes cauliflower like growth filling the cavity and causing obstruction.

Microscopic features

• Oedema in lamina propria
• Infiltration of eosinophils, mast cells, lymphocytes and plasma cells and absence of epithelioid cells.
• Proliferation of fibroblasts.
• The lesion is covered by squamous epithelium.
• Mucous glands may have metaplastic pseudostratified columnar epithelium.

TRACHEITIS

Tracheitis is the inflammation of trachea. In canines, it is tracheobronchitis while in poultry it is manifested by laryngotracheitis (Fig. 15.5).

Etiology

• Canine tracheobronchitis caused by adenovirus, influenza virus and herpes virus.
• Avian infectious laryngotracheitis (ILT) is caused by herpes virus.
Fig. 15.1. Photograph showing rhinitis in a camel and catarrhal nasal discharge.

Fig. 15.2. Photograph showing nasal granuloma (ARS/USDA)

Fig. 15.3. Photomicrograph showing nasal granuloma (ARS/USDA)

Fig. 15.4. Photomicrograph showing causative fungus in nasal granuloma (ARS/USDA)

Fig. 15.5. Photograph showing haemorrhagic tracheitis in poultry.

Fig. 15.6. Photograph showing presence of caseous exudate in larynx and trachea.

Fig. 15.7. Diagram showing presence of caseous exudate in larynx and trachea.

Fig. 15.8. Diagram showing lesions of Infectious bronchitis in poultry.
Macroscopic features
- Canine tracheobronchitis or *kennel cough* includes congestion of trachea and presence of catarrhal exudate.
- In poultry, haemorrhage in trachea and caseous plug in trachea towards larynx causing obstruction (Fig. 15.6 & 15.7).

Microscopic features
- Inclusion bodies in tracheal and bronchial epithelium in canines
- Haemorrhagic tracheitis, presence of intra nuclear basophilic inclusions in tracheal epithelial cells in infectious laryngotracheitis.

**BRONCHITIS**
Bronchitis is the inflammation of bronchi, characterized by catarrhal, suppurative, fibrinous or haemorrhagic exudate.

Etiology
- Bacteria e.g. Pasteurella
- Virus e.g. Infectious bronchitis in poultry
- Parasites
- Allergy/ Inhalation of pollens etc.

Macroscopic features
- Coughing, dyspnoea
- Mucous exudate in lumen
- Congestion and / or haemorrhages in bronchi
- Presence of caseous plugs at the point where bronchi enters in lungs in infectious bronchitis of poultry (Fig. 15.8).

Microscopic features
- Mucous exudate alongwith inflammatory cells in the lumen of bronchi.
- Hyperplasia and/or necrosis of bronchiolar epithelium
- Accumulation of mononuclear cells in the bronchial mucosa and in peribronchiolar area.

**EMPHYSEMA**
Emphysema is the increase in amount of air in lungs characterized by dilation of the alveoli. It may be acute or chronic and focal or generalized.

Etiology
- Bronchitis
- Atelectasis in adjoining area of lung
- Pneumonia
- Allergy to dust, Pollens etc
- Pulmonary adenomatosis

Macroscopic features
- Lungs are enlarged and flabby
- Imprints of ribs can be seen. Colour of lungs becomes pale.
- Cut surface is smooth and dry.

Microscopic features
- Alveoli are distended (Fig. 15.10).
- Some alveoli may rupture and form giant alveoli.
- Alveolar wall becomes thin due to stretching.
- Mild bronchitis.
- Hyperplasia of lymphoid tissue.
Fig. 15.12. Photomicrograph of lung showing oedematous fluid in alveoli.

Fig. 15.14. Diagram showing bronchogenous spread of causal agent in lung.

Fig. 15.10. Photomicrograph lung showing emphysema.

Fig. 15.16. Diagram showing hematogenous spread of causal agent in lung.

Fig. 15.11. Photograph of lung showing edema.

Fig. 15.15. Photomicrograph showing bronchopneumonia.

Fig. 15.9. Photomicrograph of lung showing atelectasis.

Fig. 15.13. Photograph of lamb showing signs of pneumonia.
PULMONARY OEDEMA

In pulmonary oedema, there is accumulation of serous fluid in alveoli of lungs (Fig. 15.11 & 15.12).

Etiology
- Bacteria
- Virus
- Allergy

Macroscopic features
- Lungs become enlarged
- Weight of lungs increases
- Cut surface releases fluid and frothy exudate in trachea and/or bronchi.

Microscopic features
- Serous fluid accumulation in alveoli of lungs
- Fluid may also be seen in some bronchi/bronchioles.
- Infiltration of inflammatory cells.
- Congestion of lungs.

PNEUMONIA

Pneumonia is the inflammation of lungs characterized by congestion and consolidation of lungs. The pathological lesions in lungs are produced in a similar way irrespective of the type of etiological agent and includes various stages like congestion, red hepatization, grey hepatization and resolution.

Stage of congestion: This stage of lung is characterized by active hyperemia and pulmonary oedema. The capillaries are distended with engorged blood and alveoli are filled with watery serous exudate. This requires 2 minutes to few hours to initiate the congestion.

Stage of red hepatization: This stage of lung is characterized by the consolidation of lungs due to accumulation of blood in blood vessels (congestion). The consolidated lungs are firm and looking like liver and hence the name “red hepatization”. Such affected lung always sinks in water. Alveoli are filled with serous or serofibrinous exudate giving hardness to lungs. In inflammatory condition, the neutrophils, macrophages and lymphocytes along with erythrocytes infiltrate the affected area of lungs. This stage of red hepatization takes 2 days for development of firmness of lung.

Stage of grey hepatization: The lung remains hard but due to lysis and removal of erythrocytes, it becomes grey or less red in colour. Firmness/hardness of lung remains same and thus, the name grey hepatization. There is increase in infiltration of inflammatory cells like macrophages, lymphocytes, epithelioid cells depending on the virulence of etiological agents.

Stage of resolution: After a week, the recovery starts in the form of resorption of fluid; autolized cells and debris is removed by phagocytic cells. The causative organism is neutralized or removed from the lungs through immunity of body. After few days the lung parenchyma becomes normal and starts functioning. If the causative agent is more virulent, it may cause death of animal due to respiratory failure or may cause permanent lesions like formation of scar, carnification, granuloma etc. There are various types of pneumonia caused by bacteria, virus, fungi, parasites, allergens, chemicals and all such affections of lungs are classified as under.

BRONCHOPNEUMONIA

Bronchopneumonia is the inflammation of lungs involving bronchi or bronchioles along with alveoli. It is thought to be spread through bronchogenous route and is the common type of pneumonia in animals (Fig. 15.14 & 15.15).

Etiology
- Virus
- Bacteria
- Chemicals
- Mycoplasma
- Chlamydia
- Parasites
- Fungus
- Mainly through bronchogenous route
Fig. 15.17. Photomicrograph showing interstitial pneumonia

Fig. 15.19. Photomicrograph of fibrinous pneumonia

Fig. 15.19. Photomicrograph showing hyaline membrane pneumonia

Fig. 15.20. Photomicrograph showing verminous pneumonia

Fig. 15.21. Photomicrograph showing aspiration pneumonia (ARS/USDA)

Fig. 15.22. Photograph showing mycotic pneumonia

Fig. 15.23. Photomicrograph showing mycotic pneumonia.

Fig. 15.24. Photograph of lung showing tubercle/granulomatous lesion (ARS/USDA)
Macroscopic features
- Congestion and consolidation of anterior and ventral parts of lungs (Lobular pneumonia).
- Patchy lesions on one or several lobes and adjacent area shows emphysema.
- Mediastinal lymphnodes are swollen.

Microscopic features
- Congestion, oedema or haemorrhage in lung.
- Infiltration of neutrophils, mononuclear cells in and around bronchioles/bronchi.
- Catarrhal inflammation of bronchi.
- Proliferation of bronchiolar epithelium

INTERSTITIAL PNEUMONIA
Interstitial pneumonia is the inflammation of the lungs characterized by thickening of alveolar septa due to serous/fibrinous exudate along with infiltration of neutrophils and/or mononuclear cells and proliferation of fibroblasts. It is also known as lobar pneumonia (Fig. 15.16 & 15.17).

Etiology
- Bacteria
- Virus
- Chlamydia
- Parasites
- Mainly through hematogenous route

Macroscopic features
- Lungs are pale or dark red in colour.
- Oedema, dripping of fluid from cut surface

Microscopic features
- Alveoli may have serous or fibrinous exudate.
- Thickening of alveolar septa due to accumulation of exudate, inflammatory cells and in chronic cases, proliferation of fibrous tissue.
- Infiltration of mononuclear cells in alveolar septa.

FIBRINOUS PNEUMONIA
Fibrinous pneumonia is the inflammation of lungs characterized by the presence of fibrin in alveoli or bronchioles and may give rise to hyaline membrane formation over the surface of alveoli or bronchiole.

Etiology
- Bacteria
- Virus
- Parasites
- Toxin/Poisons

Macroscopic features
- Antero-ventral portion of lung is congested and consolidation.
- Colour of lungs become deep red due to congestion
- Surface of lungs is covered by fibrin sheet.
- Interlobular septa are prominent due to accumulation of plasma and fibrin.

Microscopic features
- Principal exudate is fibrin, fills alveoli, bronchioles and bronchi (Fig. 15.18).
- Congestion and/or haemorrhages
- Infiltration of neutrophils, macrophages and giant cells
- Formation of eosinophilic false membrane of fibrin over the surface of alveoli and bronchiole and then known as “hyaline membrane pneumonia” (Fig. 15.19).

VERMINOUS PNEUMONIA
Verminous pneumonia is caused by parasites and characterized by the presence of lesions of broncho-pneumonia along with parasites or their larva (Fig. 15.20).

Etiology
- Metastrongylus apri in pig.
- Dictyocaulus filariae in sheep and goat.
- D. viviparus in cattle and buffaloes.
- Capillaria aerophila in dogs and cats.
- D. arnfieldi in horse and donkeys.

Macroscopic features
- Multiple petechial haemorrhage in lungs at the site of parasite penetration.
Fig. 15.25. Photomicrograph of lung showing tubercle.

Fig. 15.26. Photomicrograph of lung showing granulomatous lesions.

Fig. 15.27. Photomicrograph of lung showing granulomatous lesions and giant cells.

Fig. 15.28. Photograph showing pulmonary adenomatosis (ARS/USDA)

Fig. 15.29. Photomicrograph showing pulmonary adenomatosis (ARS/USDA)

Fig. 15.30. Photograph showing deposition of carbon particles in trachea in chicks.

Fig. 15.31. Photomicrograph showing pneumoconiosis.

Fig. 15.32. Photograph showing air sacculitis in poultry.
• Mature worms in alveoli, bronchioles and bronchi.
• Mucopurulent exudate in alveoli/bronchi.
• Pulmonary oedema, emphysema.

Microscopic features
• Dilation of bronchiole/bronchi
• Lesions of chronic suppurative bronchiolitis
• Focal areas of inflammation in the vicinity of parasites and around bronchioles.
• Hyperplasia of bronchiolar epithelium.
• Infiltration of eosinophils and lymphocytes.

ASPIRATION PNEUMONIA
Aspiration pneumonia is caused by faulty medication through drenching which reaches in lungs instead of target place (digestive track) and characterized by necrosis and gangrene of lung parenchyma.

Etiology
• Drugs, food, foreign body and oil drench which reaches in lungs through trachea.
• Paresis of throat predisposes the animal for aspiration pneumonia.

Macroscopic features
• Congestion and consolidation of anterior and ventral portion of lung.
• Affected part becomes green/black in colour, moist gangrene.
• Affected lungs are often foul smelling.
• Presence of foreign body like heads of wheats, parts of corn, oil, milk etc.

Microscopic features
• Thrombosis of blood vessels.
• Necrosis in lungs.
• Presence of saprophytes, leucocytes and bacteria cause liquefaction and gangrene.
• Gangrenous lesions surrounded by intense inflammation (Fig. 15.21).
• Congestion

MYCOTIC PNEUMONIA
Mycotic pneumonia is caused by a variety of fungi and characterized by the presence of chronic granulomatous lesions in lungs (Fig. 15.22 & 15.23).

Etiology
• Aspergillus fumigatus
• Blastomyces sp.
• Cryptococcus sp.
• Coccidioidomyces immitis

Macroscopic features
• Nodules in lungs
• On cut, cheese like caseative mass comes out from nodules.
• Caseation involves both bronchiole and alveoli.
• Such lesions may also present in trachea, bronchi and air sacs.

Microscopic features
• Presence of granulomatus lesions i.e. caseative necrosis, macrophages, epithelioid cells, lymphocytes, giant cells, fibroblasts etc.
• Presence of branched hyphae of fungi in the necrosed area.

TUBERCULOUS PNEUMONIA
Tuberculous pneumonia is caused by Mycobacterium sp. and characterized by the presence of chronic granulomatous lesions in the lungs (Fig. 15.24 to 15.27).

Etiology
• Mycobacterium tuberculosis
• M. bovis

Macroscopic features
• Grey, white or light yellowish nodules in lungs.
• Nodules are hard, painful and/or calcified.
• Animal carcass is cachectic, weak or emaciated.
• On cut, the cheesy material comes out from the nodules.
Microscopic features
- Presence of tubercle/granuloma in lungs which comprises a central necrosed area surrounded by macrophages, epithelioid cells, lymphocytes, Langhan’s giant cells and covered by fibrous covering.
- Acid-fast rod shaped bacteria may present in necrosed area.
- Central area may be calcified.

**PULMONARY ADENOMATOSIS**
Pulmonary adenomatosis is a slow viral disease of sheep and characterized by metaplasia of alveolar squamous epithelium to cuboidal and/or columnar epithelium leading to glandular appearance of alveoli (Fig. 15.28 & 15.29).

**Etiology**
- Retrovirus
  - Pulmonary adenomatosis virus

**Macroscopic features**
- Multiple focal areas of consolidation in lungs.
- Imprint of ribs on lungs.
- Congestion and hardening of mediastinal lymphnodes.

**Microscopic features**
- Metaplasia of alveolar epithelium leading to formation of glandular structures in alveoli.
- Metaplasia of simple squamous epithelium to cuboidal or columnar epithelium which gives alveoli a gland like look.
- Mild inflammatory reaction.
- Proliferation of fibrous tissue.

**HYPERSENSITIVITY PNEUMONITIS**
Hypersensitivity pneumonitis is the inflammation of lung caused by an allergic reaction of antigen (allergen) and characterized by interstitial pneumonia, emphysema, hyaline membrane formation and hyperplasia of alveolar epithelium.

**Etiology**
- Allergens
- Parasites – *Dictyocaulus viviparous*

- Moldy hay
- Fungus - *Aspergillus sp.*

**Macroscopic features**
- Lobes may contain small grey foci
- Presence of yellow and dense mucus in lumen of bronchi
- Excessive accumulation of air in lungs due to emphysema
- Presence of worms/larvae.

**Microscopic features**
- Extensive infiltration of lymphocytes, monocytes and eosinophils around the bronchi and bronchioles.
- Accumulation of catarrhal exudate in bronchi/bronchiole.
- Emphysema as a result of widening of alveoli.
- Hyperplasia of bronchiolar musculature.
- Inflammatory cells in interalveolar septa may form small granulomas.
- Formation of hyaline membrane over alveolar and bronchiolar epithelium.

**PNEUMOCONIASIS**
Pneumoconiosis is the granulomatous inflammation of lungs caused by aerogenous dust particles of sand, silica, beryllium, carbon or asbestos. It is also known as anthracosis (Fig. 15.30 & 15.31).

**Etiology**
- Silica
- Asbestos
- Beryllium
- Bauxite
- Graphite
- Carbon
- Bronchogenous/aerogenous administration of particles inhaled with air, mostly around mines/factories.
- Generator smoke.

**Macroscopic features**
- Dense fibrous nodules in lungs.
- Presence of carbon particles in trachea/bronchi mixed with mucous exudate.
Microscopic features
- Granuloma formation around the particles of silica/asbestos infiltrated by macrophages, lymphocytes and giant cells
- Silica produces cellular reaction ‘Silicosis’.
- Beryllium granuloma looks like tubercule without caseation.
- Asbestosis is characterized by the presence of club shaped filaments bearing cells in lesion.

PATHOLOGY OF AIR SACS

AIR SACCULITIS
Air sacculitis is inflammation of air sacs caused by E.coli, Mycoplasma, reovirus etc. and characterized by thickening of the wall of air sacs and presence of cheesy exudate (Fig. 15.32).

Etiology
- Escherichia coli
- Mycoplasma gallisepticum
- Avian reovirus

Macroscopic features
- Thickening of the air sac wall, which becomes dirty and cloudy.
- Presence of cheesy exudate in air sacs, congestion of lungs.
- Fibrinous pericarditis
- Liver is covered with thin fibrinous membrane.

Microscopic features
- Oedema and infiltration of neutrophils and lymphocytes in air sacs
- Caseous exudate in lungs and air sacs.

PATHOLOGY OF PLEURA

PLEURITIS
Pleuritis is the inflammation of pleura characterized by serous, fibrinous or purulent exudate. It is also known as pleurisy.

Etiology
- Mycobacterium tuberculosis
- Mycoplasma mycoides
- Haemophilus suis
- Organisms responsible for pneumonia/traumatic pericarditis may also cause pleuritis.

Macroscopic features
- Congestion of pleura
- Serous, fibrinous or purulent exudate.
- Accumulation of clear fluid in pleura/thoracic cavity is called as hydrothorax.
- Presence of blood in thoracic cavity is known as Hemothorax.
- Suppurative exudate in thoracic cavity is known as pyothorax.
- Presence of air in pleural cavity is termed as pneumothorax, while presence of lymph in pleural cavity is called as chylothorax.
- Tuberculous pleuritis is characterized by small nodules on pleura and is known as “peary disease”.
- In chronic cases, development of fibrous tissue causes adhesions and is known as adhesive pleuritis.

Microscopic features
- Congestion of blood vessels
- Infiltration of neutrophils and lymphocytes.
- Thickening of pleura due to oedema
- Proliferation of fibroblasts producing adhesive lesions.

MODEL QUESTIONS

Q. 1. Fill in the gaps with suitable word(s).
1. ............... is the inflammation of lungs characterized by ............... and ............... of lungs.
2. Lobar pneumonia is characterized by ............... of interalveolar septa.
3. Fibrinous pneumonia is characterized by the presence of ............... exudate in alveoli and may give rise to ............... formation which is ............. of fibrin over the surface of ............. and ............

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4. Aspiration pneumonia is caused by ................. of drugs/ milk and characterized by ................. and ................. formation in the lungs.

5. *Mycobacterium tuberculosis* produces ................. pneumonia in lungs characterized by ................. formation consisting of ................. central area surrounded by ................., ................., ................., ................., and covered by ................. capsule.

6. Pulmonary adenomatosi is caused by ................. and characterized by ................. of alveolar squamous epithelium to ................. or ................. leading to ................. appearance of alveoli.

7. Allergic reaction due to ................. may cause ................. characterized by ................., ................., ................. and ................. of alveolar epithelium.

8. Pneumoconiosis is ................. inflammation of lungs caused by aerogenous ................. of ................., ................. or ................. and it is also known as .................

9. Inflammation of air sacs in poultry is known as ................. caused by ................., ................. and ................. and characterized by ................. and .................

10. ................. pleuritis is also known as ................. while the presence of lymph in pleural cavity is termed as .................

**Q. 2. Write true or false against each statement and correct the false statements.**

1. ..........Bronchopneumonia is the inflammation of lungs characterized by thickening of interalveolar septa.

2. ..........Verminous pneumonia is caused by *Bordetella bronchiseptica*.

3. ..........Gangrenous pneumonia occurs due to faulty drenching of medicines.

4. ..........Mycotic pneumonia is caused by *E. coli*.

5. ..........Granulomatous pneumonia is produced by *Blastomyces* sp.

6. ..........Pearly disease is caused by *Mycoplasma myoides*.

7. ..........Atelectic lung floats in water.

8. ..........*Oestrus ovis* is the cause of nasal granuloma in sheep.

9. ..........*Metaplasia* of alveolar epithelium occurs in hypersensitivity pneumonitis.

10. ..........Air sacculitis is caused by *E. coli*.

**Q. 3. Define the followings.**

1. Rhinitis
2. Sinusitis
3. Laryngitis
4. Pharyngitis
5. Hydrothorax
6. Pyothorax
7. Epistaxis
8. Hyaline membrane
9. Silicosis
10. Asbestosis
11. Pleurisy
12. Chylothorax
13. Adhesive pleuritis
14. Tracheobronchitis
15. Pneumothorax
16. Red hepatization
17. Carnification
18. Lung worms
19. Atelectasis neonatorum
20. Bronchiolitis
21. Beryllium granuloma
22. Peribronchitis
23. Hemothorax
24. Alveolitis
25. Pearly disease
Q. 4. Write short notes on.
1. Porcine atrophic rhinitis
2. Nasal polyps
3. Nasal granuloma
4. Atelectasis
5. Pathogenesis of pneumonia
6. Lobar pneumonia
7. Hyaline membrane pneumonia
8. Gangrenous pneumonia
9. Infectious laryngotracheitis
10. Emphysema
11. Pulmonary adenomatosis
12. Bronchopneumonia
13. Mycotic pneumonia
14. Granulomatous pneumonia
15. Air sacculitis

Q. 5. Match the word(s) from four options given against each statement.
1. Nasal polyps are caused by .......... (a) Schistosoma nasalis  (b) Rhinosporidium scepteri (c) E. coli (d) Mycoplasma mycoides
2. Canine tracheobronchitis is caused by .......... (a) Adenovirus  (b) Influenza virus (c) Herpes virus (d) All of the above
3. Presence of caseous plugs in bronchi at the point of entrance in lungs in characteristic lesions of .......... (a) Infectious bronchitis (b) Infectious laryngotracheitis (c) Air sacculitis (d) Pleuritis
4. This is not the pathologic lesion of pneumonia .......... (a) Congestion (b) Red hepatization (c) Yellow hepatization (d) Resolution
5. Infection through aerogenous route may cause .......... pneumonia (a) Lobar  (b) Lobular (c) Hypersensitivity (d) Fibrinous
6. Verminous pneumonia is caused by .......... (a) Mycoplasma  (b) Chlamydia (c) Dictyocaulus sp. (d) E. coli
7. Langhan’s type giant cell is characteristic feature of .......... pneumonia (a) Tuberculous (b) Verminous (c) Broncho (d) Pulmonary adenomatosis
8. Atelectasis neonatorum is characteristic features of .......... (a) Premature birth (b) Aborted foetus (c) Still birth (d) None
9. Hypersensitivity pneumonitis is caused by .......... (a) Allergens (b) Parasites (c) Moldy hay (d) All of the above
10. Pneumoconiasis is characterized by .......... lesions in lungs (a) Serus (b) Fibrinous (c) Haemorrhagic (d) Granulomatous