

DISEASES OF THE RESPIRATORY SYSTEM

PRINCIPALES OF RESPIRATORY INSUFFICIENCY

- (I) Anoxia**
- (II) Hypercapnea**
- (III) Respiratory failure**

(I) ANOXIA:

Definition:

It means failure of the tissues to receive an adequate supply of oxygen

Types of anoxia:

1-Anoxic anoxia:

- *Occurs when there is defective oxygenation of the blood in the pulmonary circulation.
- *It is usually caused by primary disease of the respiratory tract.

2-Anemic Anoxia:

- *Occurs when there is a deficiency of hemoglobin per unit volume of the blood. The percentage saturation of the available hemoglobin & oxygen tension are normal, but the oxygen carrying capacity of the blood is reduced.
- *It is usually caused by anemia due to any cause."e.g."poisoning by nitrites or carbon monoxide .

3-Stagnant Anoxia:

- *Occurs when the rate of blood flow through the capillaries is reduced.
- *It usually occurs in cases of congestive heart failure, peripheral circulatory failure & venous obstruction.

4 –Histotoxic Anoxia:

- *Occurs when the blood is fully oxygenated, but because of the failure of the "tissue oxidation system," the tissues can not take up oxygen .
- *It usually occurs as a result of cyanide poisoning.

Special causes of Anoxic Anoxia:

- *When the oxygen tension of the inspired air is too low, that it can not oxygenate the pulmonary blood.
- *Any lesion or dysfunction of the respiratory tract reducing the supply of alveolar air such as :
 - Pneumonia.
 - Pulmonary atelectasis.
 - Pneumothorax.
 - Pulmonary edema & congestion.
 - Any decrease in the chest movement due to pain in the chest wall.
 - Obstruction of air passage by accumulation of the exudates.
- *Depression of the respiratory center by drugs or toxins.
- *Congenital defects of the heart & large blood vessels, when mixing of arterial & venous blood occurs through shunts between the two circulation .
- *Paralysis of respiratory muscles.
- *Botulism.
- *Tetanus.
- *Strychnine poisoning.

Complications of anoxia:

- *Increase in depth of respiratory movement “hyperpnea” which is mediated by chemoreceptors in the aortic arch & paroreceptors in the carotid sinus.
- *Stimulation of splenic contraction.
- *Erythropoiesis in the bone marrow.
- *Increased heart rate.
- *Signs of dysfunction of various organs appears, cerebral anoxia, myocardial dysfunction, renal and hepatic dysfunctions as well as reduction in motility and secretory activity of alimentary tract.

(II) CARBON DIOXIDE RETENTION “HYPERCAPNIA”

It means that there is an accumulation of CO₂ in the blood and tissues which cannot be eliminated via the lungs and that is due to respiratory insufficiency. This CO₂ stimulates the respiratory center.

(III) RESPIRATORY FAILURE:

Respiratory movements are controlled by respiratory center in the medulla & this center is controlled by afferent impulses from cerebral cortex, heat regulatory center in the hypothalamus, stretch receptors in lungs via the vagus & from chemo-receptors in the carotid body.

The activity of respiratory center is also regulated by: pH, oxygen & carbon dioxide tensions of the cranial arterial supply. So, stimulation of the above afferent nerves may cause reflex changes in respiration & causing stimulation of the pain fibers.

Definition:

Respiratory failure is the terminal stage of respiratory insufficiency, in which the activity of respiratory center is diminished to the point where the movement of respiratory muscles is completely stopped.

Types & causes:

Respiratory failure may be tachypenic, dyspneic, a sphixial or paralytic depending on the primary disease.

A- Asphyxial respiratory failure:

Causes:

- 1-Pneumonia .
- 2-Pulmonary odema.
- 3-Upper respiratory tract obstruction.

Clinical signs:

- 1- Hypercapnia → stimulate respiratory center → stimulation respiration
- 2- Anoxia.
- 3- Gaspings.
- 4- Apnea → death

B-Paralytic respiratory failure:

Causes:

- 1- Poisoning with respiratory center depressants.

- 2- Nervous shock.
- 3-Acute heart failure.
- 4-Hemorrhage.

Clinical signs:

- 1- Variable degree of dyspnea & gasping.
- 2- Paralysis of the respiratory center →shallow respiration & less frequent then complete stop of respiration .

C- Tachypneic respiratory failure:

Causes:

- 1-Increased pulmonary ventilation "hypoxia " but no carbon dioxide retention "acapnio".

Clinical signs:

Because of the lack of carbon dioxide to stimulate the respiratory movement; Rapid & shallow respiratory & shallow tachypnea are evident.

Treatment of respiratory failure:

- *In paralytic type → stimulants of respiratory center are given .
- *In asphyxial type → oxygen is provided .
- *In tachypneic type → oxygen & CO₂ are provided .

"PRINCIPAL MANIFESTATIONS OF RESPIRATORY INSUFFICIENCY"

The principal manifestations of respiratory dysfunction are those, which derive from anoxia .

1-Hyperpnea & dyspnea:

- Hyperpnea is defined as increased pulmonary ventilation.
- Dyspnea means difficulty of respiratory.

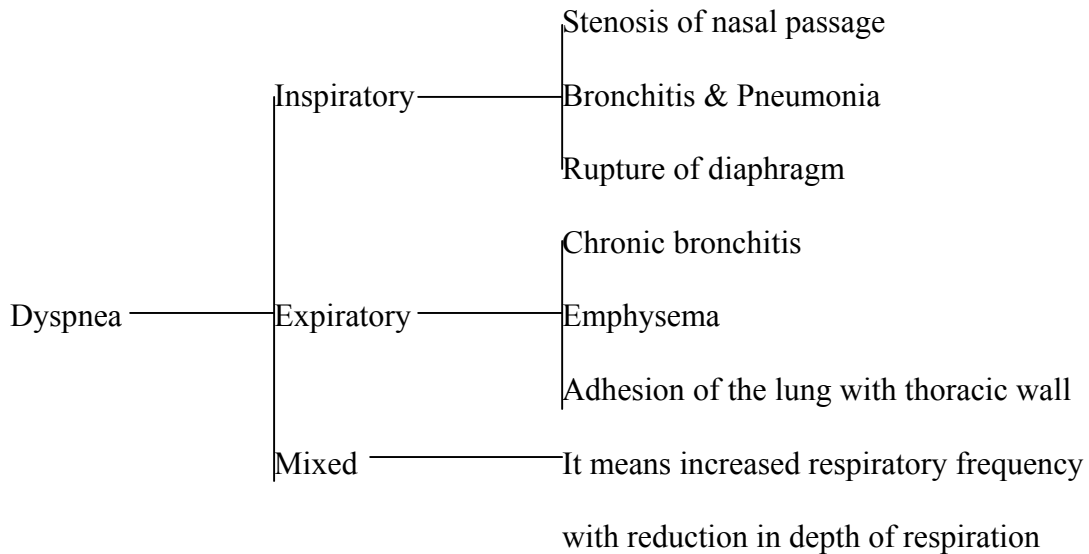
Causes:

- 1- Anoxia.
- 2- Hypercapnia arising most commonly from diseases of respiratory tract.
- 3- Congestion of the pleura leads to rapid & shallow respiration.
- 4- Pulmonary emphysema " caused by anoxic anoxia " .
- 5- Cardiac dyspnea results from backward failure of the left ventricle with congestion & edema of the lungs. Stagnant anoxia play a role in the production of this type of dyspnea .
- 6- Acidosis causes liberation of CO₂ and stimulation of respiratory center results in dyspnea .
- 7- Encephalitis causes neurogenic dyspnea.

Clinical signs:

- 1-Abnormalities of respiratory cycle such as prolongation of expiration than inspiration.
- 2-Abnormal movements of the two sides of the chest.

3-Evidence of pain during respiratory movement.



2-Cyanosis:

Definition:

It is a bluish discoloration of the skin, conjunctiva & visible mucosa.

Causes:

- 1- Increased amounts of reduced hemoglobin in the blood. It occurs only when the Hb concentration of the blood is normal & there is incomplete oxygenation of hemoglobin.
- 2- It occurs in all cases of anoxic anoxia , stagnant anoxia, but not in anemic anoxia because there is insufficient hemoglobin .
- 3- Polythemia, congenital cardiac defect & heart diseases.

Diagnosis:

The bluish discoloration should disappear when pressure is exerted on skin or mucosa & blood flow is stopped temporarily. Mat haemoglobinaemia is accompanied by discoloration of the skin & mucosa but color is more brown than blue.

3- Cough:

It is a sudden expulsion of the air from the lung preceded by deep inspiration & it is initiated by irritation of the respiratory mucosa of the air passages. It has a primary expulsive function. It is an important sign indicating the presence of primary or secondary disease of the respiratory system.

4-Nasal discharge:

*Abnormal nasal discharge is usually an indication of respiratory diseases. Mucoid or purulent discharge indicates the presence of inflammation in the nasal cavities or paranasal sinus. Frothy exudates indicates pulmonary congestion or odema.

*Small amount of serofrothy exudates in equine infectious anemia and infectious equine pneumonia.

- *Color may be greenish in gangrene, yellowish rusty in pneumonia & pleurisy.
- *Amount could be slight in T.B., profuse in rhinitis or intermittent in sinusitis.
- *Odour may be offensive in gangrene, bad in cyanosis.

5-Abnormal respiratory sounds:

These are abnormal sounds produced from the lung, bronchi, bronchioles & pleura. These sounds may be classified into rales & frictional sounds.

Rales:

Are abnormal respiratory sounds indicating the presence of secretions or aspired fluids in the bronchi & bronchioles. These fluids include "exudates, transudate, blood & aspired fluid". According to the viscosity of the secretion, rales may be dry or moist and crepitant.

NORMAL RESPIRATORY SOUNDS ON AUSCULTATION

*The normal respiratory sound heard over the respiratory area consists of vesicular sound & bronchial sound:-

*vesicular respiratory sound "vesicular murmur" The vesicular murmur resembles the sound produced when the letter "V" is whispered softly & it occurs during inspiration, but during expiration the vesicular murmur changes its character & resembles the sound of the letter "F".

*The vesicular murmur may be exaggerated or feeble "soft".

Exaggerated vesicular sound:

- *Increased respiratory frequency "polypnea".
- *Increased depth of respiration "hyperpnea".
- *Occurs normally in young animals.

Feeble vesicular sound:

- *Thickening of thoracic wall & pleura due to any cause
- *Reduced air content of the lung as in early stages of pneumonia.
- *In old animals.
- *Hydrothorax, hemothorax & pulmonary neoplasms.

Bronchial respiratory sound:

It resembles the sound produced by the letter "CH". It is heard clearly in small animals and very lean old animals but in large animals it is less distinct. The occurrence of bronchial sound in the lung are indicating of a diseased condition. It is audible when the lung contains less air with increase in the structural density of the inflammatory area which acts as a good conductor of the sound as in cases of hydrothorax, hemothorax & pleurisy "Exudative stage".

Crepitant rales:

Occurs when the bronchial mucosa is sufficiently swollen & affection extends to involve the alveoli. So, opposing walls become adherent to one another but the stream of air still pass through small communication between them it resembles the sound produced by rubbing a tuft of hair held between fingers close to the ear. It occurs in cases of: 1- Bronchiolitis. 2- Early stages of pneumonia. 3- Pulmonary edema

3-Frictional sound:

Normally the visceral & parietal pleura glide smoothly over each other, since both membranes are smooth & lubricated by clear lymph like fluid, when these surfaces are dry, frictional sound occurs. It resembles the sound produced by rubbing two pieces of leather against each other or by pressing the finger against the ear & stretching the finger nail of other hand. It occur in cases of: **1-** Preexudative stage of pleurisy. **2-** Pericarditis.

4-Emphysematous sound (Harch sound), or (Sharp sound):

Resembles the sound produced by collection of a piece of paper between fingers & hand.

5-Girgling sound :

Resembles sound produced by gases & air bubbles, as in cases of diaphragmatic hernia (in the chest) & bloat (in the rumen).

***Moist rales:**

Occurs when the bronchi & bronchioles contains thin watery mucous secretions, they are obtaining as when air is drawn from the end of the tube under the surface of water, so it is called bubbling sound. According to the site of affection moist rales are classified into:

a-Fine moist rales:

Occurs when the terminal parts of respiratory tract "alveoli" are involved. They are of unfavorable prognosis.

b-Coarse moist rales:

Occurs when the affections are confined to the bronchi & bronchioles only. It occurs in cases of:

- Bronchitis "acute".
- Bronchiolitis.
- Bronchopneumonia.
- Aspiration "drenching pneumonia".
- Hydrothorax.
- Haemothorax.
- Exudative stage of pleurisy.

***Dry rales:**

Are heard when air is forced through the bronchial tube which is partially thickened by the thick consistency exudate as by the severe swelling of the mucous membrane. It resembles the sound produced by the movement of two tightly stretched papers against each other. It occurs in cases of: **1-** Early acute stages of bronchitis.
2- Chronic bronchitis. **3-** T.B

DISEASES OF THE UPPER RESPIRATORY TRACT

RHINITIS "CORYZA OR NASAL CATTARAH"

Definition:

Rhinitis means inflammation of the mucous membrane of the nose and usually involving the upper part of the trachea, it may be acute, chronic, croupous or follicular.

Etiology:

Primary causes:

- *Inhalation of irritant vapour as ammonia or chlorides.
- *presence of foreign bodies in the nose as dust particles.

Secondary causes:

*Sudden exposure from hot to cold, this will reduce the resistance of the body & enable the M.O. which are normally inhabitant or commensals, in the upper respiratory tract as strept., staph., coryne. & pasteurilla, to become pathogenic, active and then attack the mucous membrane.

*Extension of inflammation from other parts of respiratory tract as laryngitis or even pharyngitis.

*Bacterial causes, "in the course of some diseases as":

- Glanders.
- Strangles.
- Meliodosis of sheep.
- Necrotic rhinitis of sheep.

*Viral causes:

- Malignant catarrhal fever of cattle.
- Mucosal disease.
- Rinderpest.
- Blue tongue disease.
- Infectious bovine rhinotracheitis.(I.B.R.).
- Equine viral rhinopneumonitis.
- Swine influenza.

*Parasitic causes:

- Oestrus ovis of sheep.
- Blood flukes as Shestosoma Nasalis of cattle.

Fungal Aspergillosis of dogs

Allergic condition:

Pathogenesis:

Rhinitis is usually of minor importance except when the nasal discharge rises up and blocks the nostril. Its major importance arises when it accompanies some specific infectious disease.

Clinical signs:

- *Redness and swelling of mucous membrane of the nose.
- *Bilateral nasal discharge which usually begins watery in character then mucopurulent & purulent.
- *Swelling of the submaxillary L.N.
- *Difficulty of swallowing "dysphagia".
- *Sometimes the discharge rises up and blocks the nose leading to a condition of "snorting".
- *When there is irritation the animal rubs its nose against any hard objects.
- *There may be lacrimation and bleeding.

Prognosis:

Favorable but when the causes are not treated it leads to chronic rhinitis.

Diagnosis:

- Clinical findings.
- Sudden onset.
- The character of the discharge which usually begins watery. When the animal rubs its nose against objects, ulcerations & abrasions will be formed.

Complications:

- *Chronic rhinitis may extend to other parts as nasal sinus giving rise to sinusitis.
- *Enlargement of submaxillary L.N.
- *Conjunctivitis specially in sheep.
- *Extension of the inflammation to the lung.

P.M. findings:

Rhinitis is not a fatal disease, although the animal may die from a specific disease in which rhinitis is a prominent lesion.

Treatment:

- *Put the animal in a well ventilated space away from draughts.
- *Complete rest of the animal & give only laxative, easily digestible food.
- *Irrigate the nostrils with Sodium bicarbonate (1% solution).
- *Apply medicated steam inhalation, but contraindicated to be used in milk and meat producing animals.
- *Put Vaseline on the upper lip of the animal.

CHRONIC RHINITIS

This disease takes more longer time than the acute type & it means that the case was either acute type and neglected or the stimuli acted slowly till it produces the condition

Etiology:

- *Neglected acute cases of rhinitis.
- *Accompanied with some chronic diseases of respiratory tract as chronic alveolar emphysema of horses as well as glander & T.B.

Clinical signs:

- *Mucoid nasal discharge, sometimes it may be transparent in color.
- *The mucous membrane of the nose is swollen & bluish or brownish.

- *Sometimes there is stenosis of the nasal cavities, due to swelling of the mucous membrane, accompanied by snorting which is due to breathing from the mouth.
- *There may be ulcers & abrasions on the surface of the nose, due to the fact that the animal shakes its head and tries to get rid of the discharge by rubbing the nostrils against objects.

Diagnosis & differential diagnosis:

Chronic rhinitis need care in its diagnosis, since some infectious disease gives the same symptoms as:-

1-Glanders:

- *The nasal discharge is unilateral.
- *Non inflammatory swelling of the submaxillary lymph node.
- *Ulceration & abrasions in the nostrils.
- *Positive Malline test.

2-Strangles:

The submaxillary lymph node is inflamed and tend to form abscess.

3-Sinusitis:

- *On percussion, there well be dull sound with severe painful response.
- *The nasal discharge is unilateral & is intermittent.

4-Infections of the teeth:

Careful examination of the teeth with smelling of the mouth will be helpful in differentiation.

5-Tuberculosis(TB):

Must be excluded by application of Tuberclin Test.

Differentiation between acute & chronic rhinitis

Acute rhinitis	Chronic rhinitis
1-Watery nasal discharge	1-Mucoid nasal discharge
2-Mucous memb. Is swollen & redden	2-Mucous m. is swollen & bluish
3-Swelling of the submaxillary L.N.	3-Swelling of submaxillary L.N.followed by dysphagia
4-Stenosis of the nostril	4-The animal rubs its nose against objects

Treatment :- As acute rhinitis but takes more longer time.

CROUPOUS RHINITIS:

Definition:

It means inflammation of the mucous membrane of the nose with the formation of pseudomembrane.

Etiology:

- *Inhalation of irritant vapour as gases, hot fumes or smoke.

*Some m.o. as bacillus necrophorus.

Clinical signs:

- *Exactly as acute rhinitis with inflammation of the mucous membrane & appearance of some grayish patches on the m.ms. or yellow false membrane which lastly sheds off, leaving a bleeding surface, and scabs.
- *The nasal discharge contain shreds of the mucous membrane which distinguishes this type from the acute type.
- *Swelling of the submaxillary lymph gland.
- *There may be slight rise in the body temperature.

Prognosis:

Recovery within one week.

Treatment:

As acute rhinitis but, for the rise of the body temp. give a course of antibiotics & antipyretic & isolate the diseased animals.

Follicular rhinitis:

Definition:

It means inflammation of the mucous membrane of nose with the formation of pustules which later on forms ulcers. The inflammation may involve the maxillary gland & often the sebaceous gland.

Etiology:

Streptococcus equi.

Clinical signs:

- *Swelling of the mucous membrane of the nose with formation of small nodules on the septum nasi "flea bite" size which increase in size & number & becomes yellowish in color.
- *The adjacent nodules will coalesce forming bigger nodules which ruptured leaving a bright red erosions" heal in few days".
- *The regional lymph glands & lymph .v. become swollen & enlarged forming thick cords & sometimes suppurates.
- *Conjunctivitis.

Diagnosis:

Differentiate between this type and Glander & Pox.

Prognosis:

Recovery takes place within 2-3 weeks.

Treatment:

- *Medicated steam inhalation.
- *Injection of antibiotics such is:-
- *Streptopenicid.
- *Oxytetracyclines.
- *Local application of iodine ointment on the affected area.

Differentiation between croupous & follicular rhinitis

Croupous rhinitis	Follicular rhinitis
Definition: It means inflammation of the mucous membrane with formation of pseudo-membrane	It means inflammation of the mucous membrane with the formation of pastules
Etiology: 1-Inhalation of irritant gases 2-M.Os as Bacillus Necrophrus	1-Streptococcus equi
Susceptibility: Equines & Bovines	Equines
Clinical signs: 1-Nasal discharge contain shreds of pseudomembrane 2-Swelling of submaxillary L.N. 3-Presence of yellow false membrane on the mucous membrane	1-Swelling of the mucous membrane with appearance of small nodules 2-Enlargment of the regional L.N forming thick cord 3-Conjunctivitis
Prognosis: Recovery in one week	Recovery in 2-3 weeks
Diagnosis: From symptoms	Different from Pox & Glander
Treatment: As acute rhinitis, but in case of rise of body temperature give a course of antibiotics & isolate the animals.	1-Medicated steam inhalation 2-Course of antibiotics 3-Local application of ointments.

OBSTRUCTION OF THE NASAL CAVITY

Obstruction of nasal cavities is rare in farm animals.

Etiology:

- *Acute rhinitis
- *Granulomatous lesions caused by a fungus “ Rhinosporidium”
- *Lesions caused by blood flukes “Schistosoma nasalis”
- *Foreign bodies entering the nasal cavities when the animal rubbing its nose against objects to relieve irritation of acute allergic rhinitis
- *Neoplasms of olfactory mucosa
- *Lesions usually occur in the posterior nares & are usually unilateral & may be bilateral

Clinical findings:

- *In cattle & pigs there is severe inspiratory dyspnea
- *The animal is excited & breathes through mouth
- *Loud noise “sound” occurs in each inspiration

- *Serious bloody stained nasal discharge “when there is foreign bodies, or purulent exudative nasal discharge when there is allergic rhinitis”
- *Snorting & shaking of head

Treatment:

- *Treat the cause & remove the foreign bodies by a long forceps if it is accessible
- *Administration of iodine preparation in chronic nasal obstruction as pot.iodide or sodium iodide

EPISTAXIS

Definition: It means bleeding from nostrils or from nasal sinuses

Etiology:

a-Primary cause:

- *Bad use of the tracheal tube
- *Traumatic injury of the nose, head, frontal nasal bore as well as sinuses
- *May be congenital
- *Over exhaustion of race horse
- *May occur without any apparent cause

b-Secondary cause:

- *Ulceration of the nose & septum nasi as in case of glanders in equine
- *Puncture of parasite in infection diseases as anthrax & hemorrhagic septicemia

Other causes:

- *Diseases of mucosa of the upper respiratory tract nasal cavity nasopharynx or guttural pouch
- *Erosion of the mucosa occurs in glanders & in granulomatous & neoplastic lesions in the nasal cavities.
- *By entry of foreign body in acute allergic rhinitis, or by accidental injury to the facial bones.
- *Purpuric diseases as purpura hemorrhagica, sweet clover poisoning, bracken fern poisoning & in congestive heart failure.

Clinical findings:

- *There is bleeding from nostrils either unilateral or bilateral. Blood is bright red in color, or may be mixed with mucous in case of glanders.
- *It may be scanty & stop by itself or profuse & the bleeding is profuse.
- *Pulse is rapid & weak & pale mucous membrane & anemia, loss of condition & death.

Diagnosis & differential diagnosis:

- *Try to locate the site of injury using endoscope
- *Unilateral bleeding is usually of nasal origin.
- *Bilateral bleeding is usually of nasal passage origin.
- *Bleeding caused by ulcers in the nostrils (Glanders) blood is scanty & mixed with mucous.
- *You have to identify the origin of epistaxis as following:
 - a-Blood from nostrils → bright red.

- b-Blood coming from lung → bright red & frothy.
- c-Blood coming from stomach → brownish red and acidic in reaction & mixed with ingesta.

Treatment:

- *Complete rest of the animal & keep it quite
- *Apply cold compresses on the head particularly on the frontal nasal bone & nose.
- *Apply astringent solution on the affected nostrils as:
 - Alum solution 2% or tannic acid 2%.
 - Piece of gauze soaked with adrenaline 2%.
- *In case of bilateral bleeding apply tracheotomy & plug the two nostrils with a piece of gauze soaked in adrenaline
- *Inject vitamin K to accelerate coagulation “ ½ -3 ml /20 lb.B.W.
- *Inject calcium chloride 10% 100 cc s/c.
- *Treat the cause if it is secondary epistaxis.
- *Fluid therapy “ Glucose sol. 20% I/V about one litre” or normal physiological saline 500 ml I/V.

Catarrh of the maxillary sinuses & Frontal sinus “ Sinusitis”

Definition:

It means inflammation of the mucosa of maxillary and/or frontal sinuses, manifested clinically by accumulation of mucopurulent exudates, and is usually unilateral.

Susceptibility:

The condition is common in old horses & is very rare in other animals.

Causes:

- *Traumatic
- *Extension of inflammation from nasal catarrh “ rhinitis”
- *Extension from diseases of teeth & alveoli.
- *In the course of some specific diseases as glanders & malignant head catarrh.

Clinical findings:

In horse:

- *Redness & swelling of nasal mucosa, followed by formation of granular & gelatinous nodules.
- *Swelling of submaxillary lymph node.
- *The animal lowers its head, snorts & cough.
- *Unilateral intermittent nasal discharge which is mucoid, mucopurulent and purulent and of foetid odour.
- *Painful palpation over the region of sinus.
- *Stenosis of nasal passage leading to dyspnea.
- *In later stages:

- *White streaks formed by secretion on the upper lip.
- *Conjunctivitis and lacrimation.

In cattle:

- *There is usually unilateral intermittent mucoid or mucopurulent nasal discharge, sometimes mixed with blood or threads of fibrin.
- *Accelerated breathings.
- *Violent movements of the head.
- *Expulsion of mucopurulent masses from the nasal cavities.

In pet animals:

- 1-Unilateral sero-mucoid or purulent nasal discharge, sometimes tinged with blood & of foetid odour.
- 2-The animals shake their heads and rub their noses with fore feet.

Diagnosis:

- 1-The presence of the unilateral intermittent nasal discharge, enlargement of sub maxillary L.N., conjunctivitis & painful palpation are evident.

Differential diagnosis: from glanders by Mallein test.

Treatment:

- *Irrigation of nasal cavities by astringent solution & physiological saline.
- *Removal of affected tooth.
- *Trephining may be needed.
- *Broad spectrum antibiotics are very helpful.

LARYNGITIS

Definition:

It means inflammation of the mucous membrane of the larynx and it may involve the upper part of the trachea. It may be acute or chronic.

Acute laryngitis:

Etiology:

- *Sudden exposure from hot to cold.
- *Inhalation of irritant vapour as ammonia, chloride & gases.
- *Extension of inflammation from other parts of respiratory tract.
- *Careless use of probing in case of relief of tympany.
- *In the course of some specific infectious diseases as Equine Influenza, Strangles, Swine fever & Canine Distemper.

Symptoms:

- *Redness & swelling of the mucous membrane of the nose.
- *Bilateral nasal discharge.
- *Swelling of submaxillary L.N.
- *Difficulty of respiration "dyspnea".

- *Hot painful larynx.
- *Short dry cough followed by long interval then long moist cough followed by short interval.
- *Slight rise of body temperature.

Course of the disease:

Few days but if neglected takes two weeks.

Differential Diagnosis :-

Simple laryngitis	Infectious disease
1-Laryngitis is the only symptoms 2-Slight rise of body temperature 3-Sporadic condition	1-There is other subsidiary symptoms 2-High rise of body temperature 3-More than one case
Laryngitis	Strangles
1-Affect any species of animals at any age. 2-Slight rise of body temperature 3-Swelling of sub maxillary L.N without abscess formation	1-Young horses only. 2-High rise of body temperature. 3-Abscess formation

Treatment:

Hygienic measures:

- *Complete rest of the animal.
- *Put the animal in a well ventilated space away from air draughts.
- *Only laxative food to be eaten.

Medical therapy:

- *Medicated steam inhalation.
- *Iodine ointment externally
- *Application of mustard poultice for large animals & antflugistine poultice for small animals.
- *Expectorants & respiratory stimulants as :-

R

Potassium citrate 8 gm.
 Ammonium carbonate 8 gm.
 Potassium iodide 4 gm. (in chronic cases only)
 Liquirice 4 gm.
 Puly campher 15 gm. (Not for milk & mean por.A)
 Sig./ To be mixed with sufficient quantity of treacle & given orally using Plunt object on the back of the tongue.

*Broad spectrum antibiotics as :

	Streptomycin	+	Penicillin
Equines	2 gm.		2 millions I.U.
Cattle	3 gm.		3 millions I.U.
S.Animals	½ gm.		400000 I.U.

*Sulphonamides as Sulphamezathine injection in a dose rate of 0.2 g/ kg followed by 0.1 g / kg in the next day & then 0.05 g / kg as a maintenance dose for three successive days.

CHRONIC LARYNGITIS

Definition:

It means chronic inflammation of the mucous membrane of the larynx.

Etiology:

- *Neglected acute cases.
- *Primary in nature when the stimuli acted slowly till producing the condition.
- *Secondary to some chronic infectious diseases as actinomycosis and T.B.

Symptoms:

- *Persisting cough.
- *Persisting nasal discharge.
- *Persisting loss of condition.
- *The body temperature may be rise or not.

N.B. (In horses there is whistling after coughing).

Treatment:

As the acute type but the prescription must include potassium iodide for the aim of liquification of the exudates.

ACUTE BRONCHITIS

Definition:

It means acute inflammation of the large and medium size bronchi.

Etiology:

- *Sudden exposure from hot to cold.
- *Inhalation of irritant vapor, as ammonia or chloride.
- *Bad sanitary stables as increased accumulation of ammonia, H₂S & CO₂.
- *Extension of inflammation from other parts of respiratory tract.
- *In the course of some specific infectious diseases as strangles, haemorrhagic septicaemia & canine distemper.
- *Presence of larvae of Dyticolous Flaria in sheep and goat, Dyticolous Viviparus in cattle & Oviparus in equines in the bronchi & bronchioles.

Symptoms:

- *Short dry painful cough at the beginning followed by long & moist cough.
- *Bilateral mucoid or mucopurulent nasal discharge containing pus cells.
- *Rise of body temperature. "continuous rise up to 7 days".
- *Increased pulse rate.
- *Increased respiratory frequency.
- *Congested mucous membrane.
- *In the early stages of acute bronchitis you can hear dry rales but latter on, it becomes moist.

The early is due to the swelling of the mucous membrane of alveoli or due to the thick consistency exudates, but latter on the secretion of bronchi & bronchioles

becomes watery in character.

Sometimes we hear crepitant rales because the bronchiols are sufficiently swollen & walls become adherent to one another but air still passes through small communication, as in cases of broncheolitis.

Prognosis:

Is favorable in acute bronchitis, but in broncheolitis is very difficult.

Diagnosis:

- *History of the case.
- *Clinical symptoms.
- *Auscultation.

Treatment:

Hygienic treatment “nursing”:

- *Complete rest of the animal.
- *Put the animal in a well ventilated space away from draughts.
- *Keep the bowl open by giving the animal easily digested food.
- *Apply rug on the chest of animal in cold weather.
- *Continuous supply of fresh water.
- *Apply bandage on the limbs to avoid edema & exchange the bandage daily to avoid pressure necrosis.

Medicinal treatment:

- *Medicated steam inhalation.
- *Apply iodoform on the chest “externally”.
- *Expectorant “internally”.
- *Heart tonic.
 - Campher in oil 20-25 g for horses only.
 - Cardiazol 1-2 ml sup./ cut.
 - Referine 5-10 ml for large animals.
1/2- 1 cc for small animals.
- *Antibiotics & sulphonamides.
- *Diuretics as :- Mixture of potassium citrate (15g) & sodium citrate (15g) in one liter of water orally.
- *Vitamin C .
- *Antihistaminic as avil ampoules.

Chronic bronchitis

Definition:

It means chronic inflammation of the bronchial tubes.

Etiology:

- *Neglected acute form.
- *Primary in nature when the stimuli acted slowly till producing the condition.

*Secondary to some chronic diseases as emphysema & T.B.

Clinical signs:

As acute form but without rise of body temperature & by auscultation only dry rales are heard.

Treatment:

As acute form but it takes more longer time.

Obstruction of the upper Respiratory tract

Etiology:

- *Edema of the larynx caused by allergic reactions or by inhalation of irritant gases or fumes or smoke.
- *Acute cellulites of throat “pharyngeal phlegmone”.
- *Anthrax in horse & pig.
- *Accidental obstruction when the horse or cattle vomit & a solid food material may lodge in the larynx or when sharp pointed foreign bodies lodged in the region of pharynx or larynx.
- *Obstruction in sheep specially caused by chronic laryngitis associated with corynebacterium pyogens infections.
- *Partial obstruction in horses caused by paralysis of vocal cords.
- *Enzootic nasal granuloma is of most important causes.

Clinical findings:

- *Dyspnea.
- *prolonged elevation of ribs & sinking of the flanks during inspiratory movements.
- *Cyanosis & mouth breathing.
- *Salivation & extension of the head.
- *Auscultation of the larynx reveals loud stenotic sound.

N.B.

Tracheotomy or labarotomy my be necessary to prevent fatal asphyxia until the obstruction subsides or is relived.

Chronic Obstructive Pulmonary Disease of Horse (COPD)

Chronic Obstructive pulmonary Disease (COPD) is a disease of horse characterized clinically by decreased work performance, chronic coughing, abnormal lung sounds and varying degree of pulmonary and cardiac dysfunction. Pathologically, there are varying degrees of bronchitis, bronchiolitis and pulmonary emphysema. The disease was formerly known as “heaves”, pulmonary emphysema or “bracken wind”.

Causes:

Chronic bronchiolitis which is present in affected horses is due to a hypersensitivity reaction to the allergens found in barn, dust, moldy and dusty feeds. Micropolyspores of fungi and *Asperigillus fumigatus* are recognized as common causes of Chronic Obstructive Pulmonary Disease of Horse.

Clinical findings:

*The disease occurs most commonly in adult horses 5-10 years old. The usual history is that the horse has been stabled for several weeks or months and has a chronic cough.

*The animal may have had an infectious disease 1-2 months previously, recovered from acute illness but began coughing recently. There may be a history of reduced exercise tolerance in some horses, but this is not the major feature initially.

*On clinical examination, the horse is usually bright and alert, the temperature and appetite is usually normal.

*Coughing is common and may consist of a single cough every few seconds or there is may be a paroxysm of coughing.

*An intermittent, bilateral nasal discharge is a common signs which may consist of serous fluid, mucous, mucopurulent, or bloody or a combination of their bilateral blood stained. Race horses with early COPD may develop exercise induced pulmonary hemorrhages, the so called "bleeder" and exhibit epistaxis.

*The resting respiratory rate is decreased with an increase in depth of respiration. In advanced cases the expiration which are normally biphasic become exaggerated.

*During expiration there is normal collapse of the rib cage followed by a clearly visible contraction of abdominal muscles of the flank. In long-standing cases, this results in the so called "heaves line" which is a trough that follows along the costal arch. In advanced cases the nostrils may be visibly dilated during inspiration and the force of the expiratory effort causes of the anus to protrude.

*In COPD, the heart rate is commonly within the normal range or only slightly increased. In advanced cases, pulmonary arterial hypertension may result in an increase in heart rate up to 50-60/min. The heart rate is significantly higher during exercise than in clinically healthy once.

*Auscultation of the lung in the early stage of the disease, may reveal only a slight increase in the amplitude of normal breath sounds. The abnormal sounds may be audible in the resting horses but they can be accentuated by 10-15/min. exercise on a long time. Also the placement of plastic bag over the horse nostrils for one minute, will cause the horse to hyperventilate and abnormal sounds may become clearly audible.

*The course of the disease is dependent on the removal of the continual presence of the precipitating factors. If the causes is removed in the early stage, complete recovery may occurs.

The degree of improvement will depends on how much emphysema is functional and how much is structural. In the continual presence of the precipitating causes relapse occurs commonly or the disease become progressive and affected horses become severely incapacitated, with good management and adequate housing, riding animals, and hunters or show jumpers can remain useful for many years.

The prognosis for race horses with exercise-induced epistaxis is less hopeful, because even a small degree of persistent emphysema is a serious handicap of a race horse.

*Most horses even with advanced Chronic Obstructive Pulmonary Disease do not die from the disease, but usually euthanized.

Clinical pathology and special examinations:

*Affected horses have an increased respiratory and expiratory flow rate, a decreased dynamic compliance, an increase in the maximum changes in the intrapleural pressure and elevated values for the effort of breathing.

*Precipitins against fungi have been identified in the serum of the affected horses and allergic skin tests have also been used.

*Cytological examination of transtracheal washing can aid in the differential diagnosis from infectious diseases of the lungs.

*The presence of eosinophils in the tracheal fluid samples suggests an allergic cause, however their absence does not preclude allergy as a cause.

*Endoscopic examination of the upper respiratory tract can aid in detection of horses with evidence of pulmonary hemorrhages secondary to this disease.

*Also, in the horse with advanced bronchitis, an excess of mucus may be seen in the pharynx, larynx or upper trachea.

*Radiography of the thorax may be useful in differentiating Chronic Obstructive Pulmonary Disease of Horse from others.

The important clinical features of COPD of Horse include the following:

1-Mature horse 5-10 years old.

2-History of prolonged confinement and exposure to a dusty environment.

3-Chronic coughing for several days or week.

4-Reduced exercise tolerance.

5-Dyspnoea.

6-Wheezing and crackling lung sounds.

7-An obvious expiratory effort following exercise.

8-Lack of toxemia.

The disease should be differentiated from acute pneumonia in which there is toxemia, fever, coughing and increased bronchial tones on auscultation. A tracheal wash will reveal the presence of exudates.

A concurrent pleurisy will result in a pleural effusion and muffling of lung sounds and a fluid line.

Pleurisy in horse is characterized by varying degree of toxemia, inappetence to anorexia, an increase in respiratory rate, a muffling or absence of lung sound over the ventral half or two – thirds of both lung fields a fluid line on percussion and ventral body edema. Thoracocentesis will reveal the presence of pleural fluid.

Pulmonary or mediastinal neoplasms will cause progressive weight loss, reduced exercise performance, dyspnea, hydrothorax, areas of absence of lung sound and dullness on percussion. Examination of pleural fluid may assist in the diagnosis of neoplastic cells.

It is worthy to say that viral infection of the upper respiratory tract of horses may result in animals, which continue to cough and have reduced exercise tolerance for 2-3 weeks following recovery from the acute illness.

Wheezing and cripitant sounds of the lungs may be audible and differentiation from bronchiolitis associated with COPD is difficult. With good management and adequate housing recovery is usual.

Treatment and control:

- 1-The best treatment for early cases is the provision of fresh air.
- 2-Ideally, the horse should be kept permanently in the open air, if not actually on pasture. A partly covered and well protected outside yard is also suitable. During the winter months, blankets may be necessary for horses kept outdoors.
- 3-For horses which must be housed, the boxes should be well ventilated. Every effort must be made to ensure that stable dust is kept to a minimum. All dust should be removed from roof rafters, windows and feed boxes.
- 4-If hay is fed, it should be of the best quality and must be throughout wetted with water prior to feeding. The feeding of hay as wet stuffs at ground level will encourage the horse to keep its head down for long periods where advanced cases of pulmonary emphysema in the horse are unlikely to recover but with proper care and feeding as outlined above they can be maintained as breeding animals for months and even years. Many drugs including corticosteroids, antihistaminics, expectorants, inhalants, bronchodilators and antibiotics have been used for the treatment of the COPD.
- 5-Of corticosteroids, dexamethasone is used at a dose are of 25 mg per animal intramuscularly every 2nd day for up to 2 weeks, this may give a remarkable results. Bronchodilators atropine sulfate can be used.
- 6-Administration of these bronchodilators by inhalation or atropine injection intravenously at a rate of 0.02 mg/kg body weight will result in a marked decrease in intrathoracic pressure, a decrease in respiratory rate, an initial decrease followed by an increase in arterial oxygen partial pressure and clinical improvement.

Pneumonia

Pneumonia is the inflammation of the pulmonary parenchyma usually accompanied by inflammation of the bronchioles and often by pleurisy. It is manifested clinically by an increase in respiratory rate, cough, abnormal breath sound on auscultation and in most bacterial pneumonia, by evidence of toxemia.

Causes:

a-Cattle:

- 1-Pneumonic pasteurellosis (Shipping fever). *Pasteurella hemolytica*, *P. multocida* with or without parainfluenza –3 virus (PI3).
- 2-Enzootic pneumonia of calve: PI3, adenoviruse 1,2, and 3 Rhinovirus, Bovine Syncytial Virus (BSV), reoviruses, Bovine herpes virus 1 (BSV virus) plus chlamydia spp. *Mycoplasma* spp. *Pasteurella* spp. *Corynebacterium pyogenes*, *Strept* spp. and *bedsomia* spp. (*Actinobacillus actinoides*).
- 3-Viral Pneumonia of yearling and adult cattle is caused by either PI3 or adenoviruses.
- 4-Contagious bovine pleuropneumonia caused by *Mycoplasma mycoides*.

- 5-Atypical interstitial pneumonia.
- 6-Massive infestation with ascarid larvae.
- 7-Lungworm pneumonia (*Dictycolus viviparus*).
- 8-*Klebsiella* pneumonia infection in calves and nursing cows suffering from mastitis caused by this organism.
- 9-Sporadically in T.B. (*Mycobacterium bovis*).
- 10-Sporadically in calf Diphtheria (*Spherous necrophorus*).
- 11-*Hemophilus somnus*, possibly in young cattle affected with the more common septicemic form of the disease. Its role as primary cause is uncertain.

Horses:

- 1-Newborn foals: Any of the septicemia which occur at those time, *Strep. Sp.*, *E. Coli*, *Actinobacillus equi*.
- 2-In immunodeficient foals, pneumonia is caused by adenoviruses or pneumocystis carini.
- 3-Older foals: *Corynebacterium*, *Rhodococcus equi* and equine herpesvirus.
- 4-*Dictycaulus arnifeldi* and *Parascarsis equorum* rarely cause significant pneumonia.
- 5-As a sequel to Strangles.
- 6-Rarely as sequele to equine viral arteritis or equine viral rhinopnumonitis in adult animals.
- 7-Glanders and epizootic lymphangitis (*Histomonus farcinicus*) usually include pneumonic lesions.

Sheep:

- 1-Pneumonic pasteurellosis (*Pasteurella sp.*) as acute primary pneumonia in feedlot lambs, or secondary to PI3 or *Chlamedia spp.*
- 2-Newborn lambs: Uncommonly *Strept. Zooepidemicus*, *Salmonella abortus ovis*.
- 3-*Mycoplasma spp.* (Severe pneumonia).
- 4-Symptomless pneumonia without secondary infection adenovirus, RSV, reovirus *Mycoplasma sp.*
- 5-*Corynebacterium pseudotuberculosis* (sporadic).
- 6-Melioidosis (*Pseudomonus pseudomallei*).
- 7-Lungworm pneumonia (*Dictycolus filarial*).
- 8-Progressive interstitial pneumonia.

Goats:

- 1-Pleuropneumonia caused by mycoplasma strain F38 or *M.Capri* is a devastating disease.
- 2-Chronic interstitial pneumonia with pulmonale as common sequel by a number of mycoplasma spp. *M . mycoides var. mycoids* appears to be the most commonly recorded.
- 3-Rotavirus infection.

All species:

- 1-Toxoplasmosis (sporadic cases).
- 2-Systemic mycosis lesion are focal only.
- 3-Aspiration pneumonia.
- 4-Secondary pneumonia caused by *Strept. Spp.* *Corynbact.spp.* *dermatophilus sp.*
- 5-Interstitial pneumonia, pulmonary consolidation and fibrosis by toxins in plants

(*Eupatorium glandulosum* in horses), *Zieria arborescens* (stinkwood) in cattle –
Astragalus sp. In all species.

Pathogenesis:

*Under normal condition the major airway and the lung parenchyma prevent the entry of and neutralize or remove injurious agents, so that the lung contains very few, if any organism beyond the terminal lung agents.

*Many infections of the respiratory tract originate from aerosolized particles carrying infectious agents, which arise external to or within the respiratory tract. Thus the pathogenesis of respiratory infections is related to the depletion of particles and infectious. Lung clearance mechanism:-

*The major defense mechanisms of the respiratory tract include aerodynamic filtration by the nasal cavities, sneezing, local nasal antibody, the laryngeal mechanism, the alveolar macrophages and the systemic and local antibody systems.

*Anything which interferes with the clearance of particles from the upper mucous will interfere with the clearance of particles from the upper respiratory tract. The cough reflex provide an important mechanism by which excess secretions and inflammatory exudates from the lungs and major airway can be removed from the airway and disposed of by expectoration or swallowing.

*In animal with relatively normal lungs, coughing represents a very effective means of expelling materials. In the presence of severe trachitis and pneumonia, coughing may results in retrograde movements of infected material to the terminal respiratory bronchioles and actually promotes spread of the infection to distal parts of the lungs.

*The lung clearance mechanism may be affected by a concurrent viral infection . This may have major implication in the control of some of the common infectious respiratory disease of farm animals.

* In high altitude and during periods of active physical or metabolic activity, the occurring low oxygen tension or hypoxia may follow mucociliary and macrophage activity and decrease pulmonary clearance rates. The basal ventilation activity is comparatively greater than other mammals, which results in that the inspired air becomes progressively more contaminated with infectious, allergic, or noxious substances.

*The bovine lung also has a higher degree of compartmentalization than other species. This may predispose to airway hypoxia peripheral to airway, which become occluded. This results in reduced phagocytic activity and retention or multiplication of infectious agents. In addition, because of the low number of alveolar macrophages in the bovine lung the pulmonary clearance mechanism may not be effectively as in other species. There is also a low level or atypical bioactivity of lysozyme in bovine respiratory mucous which may make cattle more susceptible to infection of the respiratory tract than other species.

Clinical findings:-

1-Rapid, shallow respiration, is the cardinal signs of early pneumonia, dyspnea occurring in the later stages.

2-Polypnoea may be quite maker with only minor pneumonic lesions and the rapidity of the respiration is an inaccurate guide to the degree of pulmonary involvement.

3-Cough is other important sign (the type of the cough varies with the type of the lesion). Bronchopneumonia is usually accompanied by a moist painful cough, interstitial pneumonia by frequent, dry, backing cough, often in paroxysms.

- 4-Cyanosis is not a common signs and occurs only when large of the lung are affected.
- 5-A nasal discharge may or may not be present in the bronchioles whether or not there is accompanying inflammation of the upper respiratory tract.
- 6-The odour of the breath may be informative. It may have an odour of the decay when there is a large accumulation of inspissated pus present in the air passages, or putrid, especially in horses, when pulmonary gangrene is present.
- 7-Auscultation of the thorax before and after coughing may detect exudate in the air passages.
- 8-By auscultation in the early congestive stage of bronchopneumonia and interstitial pneumonia the vascular murmur is increased. Moist rales develop in bronchopneumonia as bronchiolar exudation increases but in uncomplicated interstitial pneumonia, clear, harsh bronchial tones are audible. When complete consolidation occurs in either forms, bronchial tones are the only sounds audible over the affected lung but moist or cripitant rales can be heard at the periphery of the affected area in bronchopneumonia.
- 9-Consolidation also causes increased audibility of the heart sounds. When pleurisy is also present it causes a pleuritic friction rub in the early stages and muffling of the pulmonary sounds in the late exudative stages. Consolidation can be detected also by precussion of the thorax or by tracheal percussion.
- 10-There may be an observable difference in the amount of movement in the two side of the chest if the degree of consolidation is much greater in one lung. Additional signs evident in pneumonia include fever of variable severity, anorexia, depression, an increase in pulse rate.

In chronic bronchopneumonia in cattle:

- 1-There is toxemia with rough hair coat. Respiratory and heart rates are above normal and there is usually a moderate fever.
- 2-The depth of respiration is increased and both inspiration and expiration are prolonged. A grunt expiration and open mouth breathing indicate advanced pulmonary disease.
- 3-A copious bilateral mucopurulent nasal discharge and a chronic moist productive are common.
- 4-On auscultation of the lungs loud bronchial tones are usually audible over the ventral half of the lungs and moist dry rales are commonly audible over the entire lung fields, but are most pronounced over the ventral half.
- 5-With adequate treatment in the early stages, bacterial pneumonia usually responds quickly and completely but viral pneumonia may not respond as it may relapse after an initial response.
- 6-The treatment response is probably due to control of secondary bacterial invaders. In some bacterial pneumonia the same course is apparent due to either reinfection or persistence of the infection in necrotic foci out of the reach of usual therapeutic measures

Clinical pathology:

- 1-Anti-mortem lap. Examination consists largely of cultural examination of nasal swab or tracheal sputum and determination of the sensitivity of the isolated bacteria to antibacterial agents.
- 2-Transtracheal aspiration has been described earlier and is valuable tool for an intensive investigation of a respiratory tract infection.

- 3-Radiographic examinations is undertaken only in animals of suitable size.
- 4-Hematological examination usually reveals a leukocytosis with shift to the left in bacterial pneumonia.
- 5-A leukopenia and lymphopenia occurs in some cases of acute viral pneumonia. In viral pneumonia, the serological testing of acute and convalescent sera, in addition to the isolation of the virus, are useful tools support in evidence of the presence of an active infection.

Diagnosis:

There are two kinds of errors in the clinical diagnosis of pneumonia.

- One of that the pneumonia is not detected clinically, because the abnormal lung sound are apparently not obvious.
- The other is to make a diagnosis of pneumonia, because of the presence of dyspnoea which was due to disease in some other body systems.
- The major clinical findings of pneumonia are polypnoea in the early stage and dyspnoea later, abnormal lung sounds, and fever and toxemia in bacterial pneumonia. Polypnoea and dyspnoea may result from involvement of other body systems as congestive heart failure, terminal stage of anemia, poisoning by histotoxic agents such as hydrocyanic acid, hyperthermia and acidosis are accompanied by respiratory embarrassment, but not by the abnormal sounds typical of pulmonary involvement. Pulmonary edema and congestion, embolism of the pulmonary artery and emphysema are often mistaken for pneumonia, but can be usually differentiated by the absence of fever and toxemia, on the basis of the history and on auscultation findings.
- All of the practical lab. Aids described should be used when necessary. They are of particular importance when outbreaks of pneumonia are encountered. In a single routine cases of pneumonia the cause is usually not determined.
- However the age and class of the animal, the history and epidemiological findings and the clinical findings can usually be correlated and a presumptive etiological diagnosis done.

Differential diagnosis:

*Differential diagnosis must be done from pleurisy, that characterized by shallow, abdominal type of respiration, pleuritic friction sounds when effusion is minimal and a muffling of lung sound and a fluid line detectable by auscultation and percussion when fluid is plentiful.

* Differential diagnosis must be done from pneumothorax, where the later have inspiratory dyspnea and on the affected side the abnormalities include; an absence of the vesicular sounds, but bronchial tones are still audible over the base of the lungs, an increase in absolute intensity of the heart sound and increased resonance on percussion. Finally differential diagnosis must be done from diseases of the upper respiratory tract such as laryngitis, and tracheitis which are accompanied by varying degrees of inspiratory dyspnea which is often loud enough to be audible without stethoscope.

*In less severe cases, auscultation of the mid- cervical trachea will reveal moist wheezing sounds on inspiration. These sounds are transmitted down into the lungs and are audible on thorax auscultation.

*In some cases of severe laryngitis and tracheitis, the respiratory sound are audible over the trachea and over the lungs are markedly reduced because of almost total obliteration of these organs.

*In laryngitis and tracheitis there is usually a more frequent cough than in pneumonia and the cough can be readily stimulated by squeezing of the larynx or trachea.

*In pneumonia the abnormal lung sounds are audible on both inspiration and expiration. Examination of the larynx through oral cavity in cattle and with the aid of rhinolaryngoscope in the horse will usually reveal the lesions.

Treatment:

- 1-In specific infection isolation of affected animals and careful surveillance of the remainder of the group to detect cases on the early stages should accompany the administration of the specific antibacterial drugs or biological preparations to affected animals. The choice of antibacterial agents will depend on the tentative diagnosis, the experience with drugs in previous cases and the results of the drug sensitivity tests.
- 2-The common bacterial pneumonia of all species will usually recover quickly (24 hr.) if treated with an adequate dose of the drug of choice early in the course of the disease.
- 3-Animals with severe pneumonia will require daily treatment for the several days until recovery occurs.
- 4-Those with bacterial pneumonia and toxemia must be treated early on an individual basis. Each case should be identified and carefully monitored for failure to recover.
- 5-Antimicrobial agents in a long acting base may be used to provide therapy over a 4-6 day period instead of the daily administration of the shorter-acting preparations. However, the blood level from the long-acting preparations are not as high as the shorter-acting preparations and may not be as effective in severely affected animals.
- 6-The common causes for failure to respond favourably to treatment for bacterial pneumonia include: **A)** Advanced disease when treatment was undertaken, **B)** The development of pleurisy and pulmonary abscesses., **C)** Drug resistant bacteria.
- 7-There is no specific treatment for the viral pneumonia because viral and mycoplasmal pneumonia are commonly complicated by secondary bacterial infections. It is common practice to treat acute viral and mycoplasmal pneumonia with antibacterial until recovery is apparent.
- 8-In outbreaks of pneumonia where many animals are affected and new cases occur each day for several days, the use of mass medication of the feed and / or water supplies should be considered.
- 9-Mass medication may assist in the early treatment of subclinical pneumonia and is a labor-saving method of providing convalescent therapy to animals which have been treated individual.
- 10- When outbreaks of pneumonia occur and new cases are being recognized at the rate of 5-10 % per day of the total in the group, all the remaining in contact animals may help to treat subclinical cases before they become clinical and thus "abort" the outbreak.
- 11-Corticosteroids have been used for their anti-inflammatory. Effect in the treatment of acute pneumonia. However, there is no clinical evidence that they are beneficial. Affected animals should be housed in warm, well ventilated, draft-free accommodation, provided with ample, fresh water, and light nourishing food.

- 12-During convalescence condition, the return to work or exposure to bad or cold weather should be avoided. If the animal does not eat, oral or parenteral force-feeding should be initiated. If fluids are given intravenously care should be exercised in the speed with which they are administered. Injection at too rapid rate may cause overload on the heart ventricle and death may occur due to acute heart failure.
- 13-Supportive treatment may include the provision of O₂ supply to be available especially in the critical stages when hypoxia is evident. In foals, the oxygen can be administered through an intranasal tube passed back to the nasopharynx and delivered at the rate of about 3 liters / min. for several hours. Expectorants may be of value in chronic cases and during convalescence.

ASPIRATION PNEUMONIA **(Drenching pneumonia, Foreign body Pneumonia,** **Regulation pneumonia, Inhalation pneumonia,** **Mechanical pneumonia)**

It is a common series disease of farm animals characterized by short course & sudden death of affected animals.

Etiology:

- *Careless drenching.
- *Careless passage of stomach tube during relieve of other diseases.
- *Vomiting in ruminants and horses that followed by aspiration.
- *Paralysis of the pharynx & larynx.

Symptoms:

- *Death occurs quickly even when a small amounts of fluid are drenched & that depends on the character of fluid & velocity of absorption from the lung is very rapid & soluble substance as chloral hydrate or magnesium sulphate produce their pharmacological effect very quickly if the material is insoluble as a result of aspiration of a vomits toxemia develops & is usually fatal within 48-72 h.
- *Signs of pneumonia are present including coughing, rales, foamy nasal discharge accompanied by protrusion of tongue & feted odors breath, congested mucosa, rapid pulsation & febrile condition.
- *The condition is usually followed by gangrenous pneumonia.

Prognosis:

Very bad & unfavorable.

Treatment:

- *If the lesion is advanced, treatment is non effective, but treatment using broad spectrum antibiotic & sulfonamides may prevent the development of the disease if administered quickly after aspiration of fluids.
- *Care of the animals and supportive remedies & sedatives are reliable if the case is not advanced.

Lung worm infestation (Verminous broncho pneumonia)

Definition:

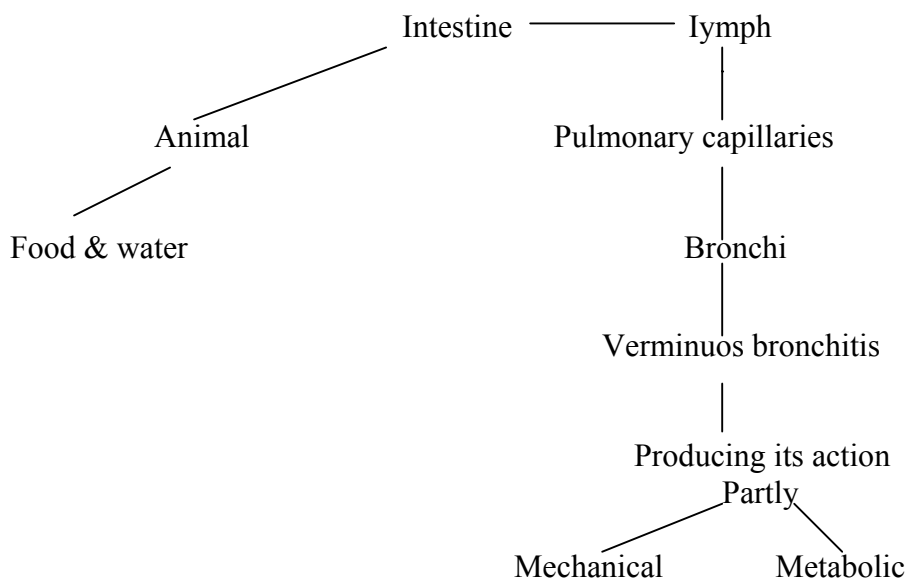
Lung worm infestation is an enzootic or epizootic affection manifested by bronchitis & bronchopneumonia & caused by infestation of lung by metastrongylides.

Etiology:

The lung worms are long thin smooth thread worms belonging to the family metastrongylidae except genus "cappillaria" which belongs to the family "trichinellidae".

- 1-Dictycaulus filarial of sheep, goat, camel & occasionally cattle.
- 2-Dictycaulus viviparous of cattle.
- 3-Dictycaulus oviparous of horse & donkey.
- 4-Metastrongylus elongatus of pigs & occasionally in sheep.
- 5-Prostrongylus rufescens: sheep, goat & rabbit.
- 6-Prostrongylus commintatus: sheep and goat.
- 7-Mallerious coppillaris: sheep & lambs.
- 8-Strongylus abstruses: cat.
- 9-Haemoststrongylus vasorum: dog.

Pathogenesis:



Clinical findings:

Incubation period between infestation & appearance of symptoms ranged between 4-8 weeks.

Sheep & goat:

- *The first symptom is cough.
- *Cough is accompanied by expectoration of masses of mucous containing adult worms or larvae.
- *Seromuroid nasal discharge which cause irritation & itching around the nares.

- *Respiration is labored.
- *Severe rise of body temperature reaching to 42 when the lung is involved.
- *Emaciation.
- *Anaemia.
- *Frequent diarrhea.
- *Enlargement & swelling of submaxillary region, lips & eye lids & sometimes all of the anteroventral part of the head.

In cattle “mostly in calves & newly born” and Horse:

- *Symptoms commences with cough which is accompanied by protrusion of tongue and expectoration of mucous containing the worms or larvae.
- *Respiration is accelerated & later on become labored.
- *Temp. is seldom raised.
- *Severe infestation may kill the animal within 3-8 days preceded by a period of anemia, emaciation & diarrhea.

Diagnosis:

- 1-Clinical signs.
- 2-Faecal examination.

Treatment:

- *Carbolic acid 1% I.M. injection of 5 ml for sheep & goat 20 cc for calves & equines.
- *Phenothiazine.
- *Copper sulphate.
- *Levamisole injection.

PULMONARY EMPHYSEMA

Definition:

Emphysema means over distension of alveoli without any change in the pulmonary tissues & with or without escape of air into the interstitial tissue causing reduction in the air space & losing the elasticity of the alveoli.

Classification:

- 1-Acute alveolar emphysema:- mostly in cattle & is “ temporary distension of the alveoli”.
 - 2-Interstitial emphysema:- in which rupture of one or more alveoli & the air escape to the interlobular & intralobular spaces “ mostly in cattle & is fatal”.
- *Chronic alveolar emphysema or “Heaves Disease” in horses, overdistension of the alveoli till completes loss of elasticity.

Etiology:

***In Cattle:-** It is an important lesion in atypical pneumonia, parasitic pneumonia & anaphylaxis.

a-Acute alveolar emphysema:

- *Persisting cough.

- *Obstruction of the alveoli with food particles, foreign bodies, larvae of ascaris, dictyocoulus viviparous or exudates in case of bronchitis.
- *Collapse of some parts of the lung “alveoli” and other parts are distended to compensate “compensatory emphysema”.
- *Red hepatized pneumonia.
- *Traumatic reticuloperitonitis & pulmonary abscess.

b-Interstitial emphysema:

- 1-Heavy infestation with lung worm.
- 2-Damage of the lung tissue.
- 3-In case of parturition* over distention & lead to rupture of the alveoli.

***In Horses:-** The cause is not known but it may be due to:-

- *Heavy work & exercise.
- *Supplying the animal with dusty foods causes continuous coughing distention of alveoli loses their elasticity.
- *Housed horses in barns for long time.
- *Chronic bronchitis associated with bronchial stenosis.
- *Chronic or proximal coughing & excessive expiratory movement due to over exertion.
- *A similar syndrome has been produced by I./V. injection of histamine.

Pathogenesis:

The general idea for the pathogenesis is that:-

Over stretching of the supporting & elastic tissue of the pulmonary parenchyma * leads to excessive dilatation of the alveoli.

There are two schools for clarifying the above pathogenesis which are:-

*One purpose that, there is primary deficiency in the strength of the supporting tissue & this tissue will be unable to support the alveolar wall during coughing or exertion.

*The second purpose that the chronic bronchitis, bronchiolitis or bronchial spasms due to allergy, causes obstruction of the air passage, but air still enters the alveoli through small communication between them, thus air accumulates causing overdistention and finally rupture of the alveoli.

On Both cases an initial lesion leads to an area of weakness from which emphysema spreads during coughing or exercise.

-In Compensatory emphysema * there is rupture of one or more of the alveoli due to excessive accumulation of air into the alveoli * then air escapes to the interstitial tissue.

-In Compensatory emphysema, the emphysema develops in association with other lesions such as atelectasis or edema.

It has two hypotheses of pathogenesis:-

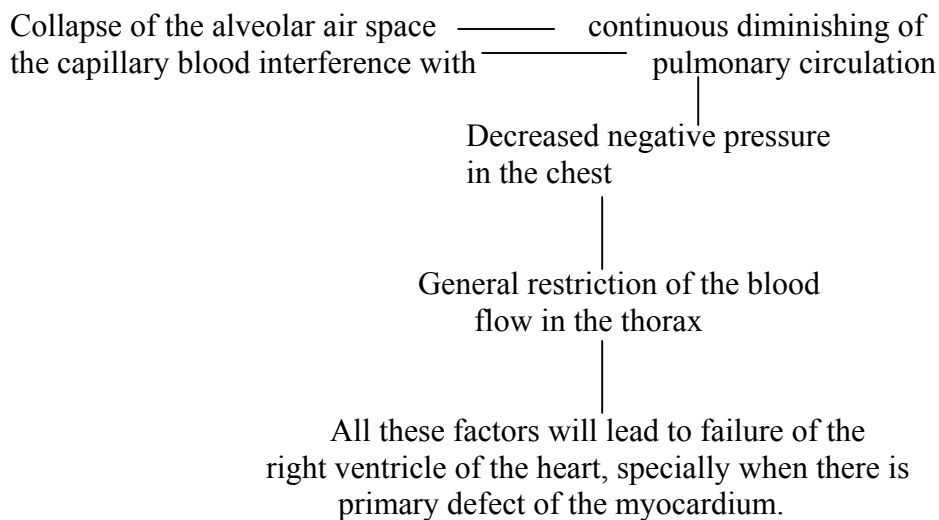
- *When a primary lesion occurs in some parts of the lung collapse of these parts, so the alveoli of the other parts are distended to compensate.
- *The agent which causes the primary lesion may also reduce the strength of the

surrounding supporting tissue, so causes obstruction of the neighboring bronchiols accumulation of air as illustrated before * emphysema.

Physiologically emphysema is due to:-

- 1-Insufficiency of evacuation of pulmonary air.
- 2-Failure of the normal gaseous exchange in the lung, so diminished the force of elastic tissue * increase the residual volume to maintain normal gaseous exchange * incomplete evacuation during expiration.
- 3-The retention of CO₂ * stimulate the depth of respiratory development of anoxia & reduced metabolism of all body tissues.

Also the retention of the carbon dioxide may cause acidosis.



Clinical findings:

1-Acute alveolar emphysema

- *Signs are similar to those of the chronic case but dyspnea is more severe.
- *On percussion * Hyperresonant sound in the anterior & posterior parts of the lung.
- *On auscultation * Rales associated with bronchitis.

2-Interstitial emphysema

- *Dyspnea.
- *Subcutaneous emphysema over the trunk, shoulder & inlet of thorax and may spread all over the lung (in cattle), but in other species there may be perforation of the pleura.
- *On percussion * Hyperresonant sound.
- *On auscultation * Crepitant rales.

3-Chronic alveolar emphysema

- Signs appear gradually and in the early stages are evident only during exercise.
- *Prolonged expired phase in each respiratory cycle.
- *Increased depth of respiration & during expiration, there is normal collapse of the ribs followed by an expiratory uplift of the abdominal wall.
- *Short weak cough.

- *Affected horses are of little use for work.
- *On percussion * hyperresonant sound over the diseased area. On auscultation crepitant rales.
- *Expansion of the lung over the heart * absence of heart sound * may lead to congestive heart failure.
- *Prolonged cough.
- *Difficulty in breathing.
- *Abdomen is barrel in shape.
- *Animal shows double expiratory movements, the 1st is costoabdominal and the 2nd is wholly abdominal in which the animal tries to get rid of the expired air by movements of the abdominal wall.
- *Extension of the lung till 14th rib “ normally is up to 11th rib”
- *Groove in the flanks along the line of the costal arch.
- *Course of chronic case some months or even some years.

Clinical pathology: →

The retention of CO₂ increases the alkali reserve.

Necropsy findings:

- *Lungs are distended & became pale in colour.
- *In interstitial emphysema * the interalveolar septae are distended with air which may spread to beneath the pleura.
- *There may be evidence of congestive heart failure.
- *Histopathologically → bronchitis is present in most cases.

Diagnosis:

From clinical signs in which acute emphysema in cattle & horses is often accompanied by pulmonary oedema with the presence of consolidation & fluids, as well as rales in the ventral parts of the lung.

Differential diagnosis:

- *The condition may be confused with acute pulmonary congestion, edema of the lung, pneumonia & pneumothorax.
- *Pneumonia is characterized by fever & localization of abnormal respiratory sound.
- *Crepitant rales may occur in chronic pneumonia, but only at the periphery of the consolidation area.
- *Pneumothorax is accompanied by forced inspiration & absence of normal breath sound.

Diagnosis “Detectable”:

Difficult in early stages, but in advanced cases:

1. Clinical symptoms.
2. By test helping in detection the animal suffering from the disease, “Exercise test”:
 - *Count the respiration per minute before exercise.
 - *Left the animal to pull a moderate weight.
 - *Allow it to exercise.
 - *Count the respiration every 5 minutes.
 - *Then put the animal in the stable for the rest.

*Again count the respiratory movements every 5 minutes.

*Follow up the animal till the respiratory rate returns back to the normal pattern.

N.B:- The normal respiratory rate in horses is 10-14 movements/minute. While the rate after exercise is 50-60 movements/minute. This rate will come back to the normal pattern after 15-18 minutes, but the rate in diseased horses after exercise is 80-120/minute and not return back before 30 minutes.

Prognoses:

Unfavorable, but you may be helpful in stopping the progress of the disease by putting the animal in good hygienic condition & allowing good nourished food free from dust as well as light work.

Treatment:

*Administration of Beladonna 25-40 gm. daily for adult horses suffering from chronic alveolar emphysema.

*In interstitial emphysema * Slaughter the cattle.

*In acute alveolar emphysema * Treat the cause.

*Complete rest of the animal away from air draughts.

*Put the animal in a well ventilated space.

*Oxygen therapy may be recommended.

*Injection of Atropine * relieve of dyspnea specially if odema is present.

*Antihistaminics therapy in acute alveolar emphysema in cattle & may be of value in chronic alveolar emphysema in horses, because it is known that histamine release induce bronchoconstriction.

PULMONARY EDEMA & CONGESTION

Pulmonary congestion is caused by an increase in the amount of blood in the lungs due to the engorgement of the pulmonary vascularization. Sometime It is followed by pulmonary edema when the intra-vascular fluid escapes into the surrounding parenchyma & alveoli & this will lead to respiratory disturbance & the degree of disturbance depends on the amount of alveolar air space which is lost.

Etiology:

*Primary * The basic lesion is in the lung.

*Secondary * Accompanied by many other diseases.

*Pulmonary congestion & edema is the terminal stage of many diseases.

Primary congestion:

Is the first stage in most cases of pneumonia.

- After inhalation of smoke & irritant fumes.
- In anaphylactic reactions.
- As hypostatic congestion in recumbent animals.

Secondary congestion:

Is usually a manifestation of congestive heart failure affecting the left ventricle when the blood flow through the lungs increased by increased pressure in the pulmonary vessels.

Overexhaustion of horses “not by hard work”, is characterized by pulmonary congestion which is partly primary & partly secondary due to acute Myocardial asthenia.

Pulmonary edema:

- As a result of pulmonary congestion in acute anaphylaxes.
- Hog fever.
- Congestive heart failure.
- After inhalation of smoke.
- In some toxaemias.
- Poisoning with organo – phosphorus compounds.
- Poisoning by “alphanaphthyle thiourea” “A.N.T.U”

The congestion of pulmonary capillaries leads to loss of amount of alveolar air space which leads to reduced vital capacity reduced oxygenation of the blood which is also due to reduced blood flow through the pulmonary vs. & this will lead to congestion of lung.

The vital capacity still reduced till appearance of pulmonary edema which is also result from the anoxic anoxia caused by reduced oxygenation of the blood & by passage of fluids due to increased hypostatic pressure.

Filling of the alveoli & in severe cases the bronchi, prevents the gaseous exchange.

Clinical findings:

- *Dyspnea with extended head & mouth breathing.
- *Exaggerated respiratory movements & increased respiratory rate.
- *There is marked abdominal & thoracic movements during inspiration & expiration.
- *Spreading of legs “fore limbs” wide apart.
- *Abducted elbow.
- *Increased pulse rate.
- *The mucous membrane of the nose is congested.
- *By auscultation in acute congestion sharp vesicular sound but no “rales” but when edema occurs “there is rales especially on the ventral parts of the lungs”.
- *There may be emphysema of the dorsal parts of the lungs.
- *By percussion “normal in early congestion & dull in edema”.
- *Soft & moist cough.
- *Serous nasal discharge “in congestion” & frothy may be bloody tinged “in edema”.
- *Death by asphyxial respiratory failure.

Prognosis:- Recovery occurs unless there is damage to the alveolar epithelium & presence of myocardial asthenia.

Clinical pathology:

- *Laboratory examination is only of value in differentiating the cases of edema and congestion.
- *Bacteriological examination of nasal swap.
- *Complete hematological examination “presence of more eosinophile than normal”.

Necropsy:

In congestion:

- *Lungs are dark red in colour & excessive quantities of venous blood exude from the surface.
- *Histologically pulmonary capillaries are engorged with blood.

In edema:

- *Swelling & loss of elasticity of the lung which pits down on pressure.
- *Lungs are usually pallor than normal & excessive quantities of serous fluids exude from the cut surface of the lungs.
- *Histologically there are accumulations of fluids in the alveoli & parenchyma.

Diagnosis:

- *From symptoms & causative agents.
- *Response to antibacterial treatment.

Treatment:

Hygienic treatment:

- *Treat the cause.
- *Complete rest to the animal at dry & clean environment.
- *Avoid exercise to the animals.

Medicinal treatment:

- *Venous suction with removal of 4m 1/ 11Lb.B.W. of blood in severe congestion.
- *Oxygen therapy in cases of anoxia as well as parenteral respiratory expectorants.
- *In severe cases Atropine sulphate to reduce fluid transudation.
- *Administration of Ethyle Alcohol 45% + mixture of Silicons & polyhydric alcohol, for the aim of treating the frothy air passages "The same as frothy bloat in ruminants".

PULMONARY ABSCESS

The presence of one or more abscess in the lung leads to chronic toxemia, cough, emaciation and may lead to suppurative bronchopneumonia.

Etiology:

- *Infected emboli originating from other organs may localized in the pulmonary capillaries are the most common causes of pulmonary abscess.
- *Endocarditis, metritis & mastitis are primary lesions often associated with pulmonary embolism.
- *In T.B. & Actinomycosis also are accompanied by pulmonary lesion & lead to pulmonary abscess.
- *Systemic mycosis may also accompanied by pulmonary lesions & may lead to pulmonary abscess.
- *Aspiration pneumonia.
- *Bacterial pneumonia specially Contagious Bovine Pleuropneumonia.

Pathogenesis:

Pulmonary abscess may be present in many cases of pneumonia, but are not recognized clinically.

In the absence of pneumonia pulmonary abscess is usually a chronic disease when spread, is hematogenous & large number of small abscesses develop simultaneously increase in size obliteration of large area of lung tissue.

In many cases there is a period of illness when abscess are walled or surrounded by connective tissue focus. Under certain condition, this focus is ruptured and produce a fatal suppurative bronchopneumonia.

Clinical finding:

- *Dullness, anorexia, emaciation & drop in milk production.
- *Slight & fluctuated elevation of body temperature.
- *Harsh & sharp cough & not accompanied by signs of pain.
- *Respiratory signs depend on the size of lesions.
- *Percussion carefully—→circumscribed area of dullness. Auscultation no breath sound can be heard & crepitant rales can be heard at the periphery of the lesions.
- *Dyspnea & purulent nasal discharge.
- *Bronchopneumonia may be developed from the extension of the abscess.

Clinical pathology:

- 1-Examination of the nasal or tracheal mucous.
- 2-Hematological examination gives an lesion, on which there is leukocytosis & shift to left.

Necropsy finding:

Accumulation of necrotic material in a thick walled fibrous capsule, is usually present in the ventral borders of the lung surrounded by a zone of bronchopneumonia. In sheep there may be an associated emphysema.

There may be encapsulated lesions showing evidence of recent rupture of capsule. Moreover a multiple small abscesses may be present when hematogenous spread has been occurred.

Diagnosis:

The diagnosis is difficult, but when a multiple small abscesses are present it may cause toxemia.

Differential diagnosis:

- *Toxaemia is produced from multiple small pulmonary abscess & it must be differentiated from that developed from splenic or hepatic abscess.
- *Exclude T.B. by tuberculin test.
- *The focal parasitic lesions such as hydatid cysts are not accompanied by toxemia or hematological changes.

Treatment:

Is not successful, but broad spectrum antibiotics are recommended for several days followed by enzyme therapy.

Pulmonary Neoplasms

Primary & metastatic neoplasms of lungs are rare in animals, although sporadic cases are found to have tumors, for examples:

Goats* Papilloma “in diaphragmatic lobes”.

Horse* 1- Granular cell myoplastoma.
2-Malignant melanosis in adult.

Cattle* 1- Lymphomatoses in young.

Clinical finding:

1-Decrease in the vital capacity of the lungs.

2-Dyspnea & cough.

3-Percussion & auscultation * localized consolidation.

4-There is no fever or toxemia.

5-There may be no evidence of inflammation.

6-There may be congestive heart failure “due to the compression & displacement of the apex of the heart”.

Complication:

Metastasis to the bronchial lymph node may cause obstruction of the esophagus dysphagia & “ruminal tympany of cattle”.

Diagnosis:

1-From symptoms.

2-Total differential count of leukocytes (may be normal).

Differential diagnosis:

*The disease may be mistaken with chronic encapsulated pulmonary abscess.

*Jugular engorgement. Ventral edema, tachycardia. Tympany & hydropericardium may cause a confusion with traumatic pericarditis.

“Pneumothorax”

Definition:

It means entry of the air in the pleural cavity in a sufficient quantity to cause collapse of the lung.

Etiology:

*Puncture of the chest wall from exterior by a sharp foreign body.

*Rupture of the lung by a sharp end of broken rib.

*Coughing for long time.

*Severe exercise.

*Chronic irritant emphysema.

Pathogenesis:

*The condition is unilateral except in horse.

*Entry of the air in the pleural cavity by any way absence of the negative pressure in the pleural sac collapse of the lung “on the affected side” pneumothorax.

Complication:

- 1-Hemothorax.
- 2-Pleurisy.

Clinical findings:

- *Acute dyspnea.
- *If the condition is unilateral, the chest cage on the affected side are collapse showing decreased movement.
- *Bulging of the chest wall on the an affected side.
- *Auscultation in complete absence of normal vesicular sound, but the bronchial sounds are normal.
- *The mediastinum bulges towards the unaffected side & may cause displacement of the apex of the heart.
- *The heart gives metallic sound.
- *Percussion over the effected side metallic & tympanic sound.

Clinical pathology:

Lab exam. Is of no value. But “X” ray show displacement of the mediastinum & heart and collapse of the lung.

P.M:

- 1-The lung of the affected side is collapsed.
- 2-There may be heamothorax.

Diagnosis:

The clinical findings & x-rays are diagnostic.

Diff. Diagnosis:

- *Diaphragmatic hernia may cause similar signs in small animals, but is rare in farm animals.
- *In cattle: hernia is a associated with traumatic reticulitis & not accompanied by respiratory disturbances.

Treatment:

- *Closure of the thoracic wound by surgical means & keep the animal quite.
- *Give prophylactic treatment.

DISEASES OF PLEURA HYDROTHORAX & HEMOTHORAX

Definition:

Hydrothorax: Means presence of non inflammatory fluid in the pleural sac.
Hemothorax: Means presence of blood in the pleural cavity. The accumulation of edematous transudate or whole blood in the pleural sac is accompanied by respiratory manifestation caused by collapse of the ventral parts of the lung.

Etiology:

- Hydrothorax accompanies:
- *Genenal edema caused by congestive heart failure & hypoproteinaemia.

*Lymphomatosis in cattle. Haemothorax: is rare & occurs when pleural adhesions are ruptured & or there is traumatic injury of the chest wall.

Pathogenesis:

Accumulation of fluids in the pleural sac * compression atelectasis of the ventral parts of the lung * dyspnea
* the pressure causes compression on the veins increased venous pressure.

Clinical findings:

In both diseases there is absence of systemic signs except of the acute hemorrhagic anemia due to excessive bleeding in the pleural cavity.

*Dyspnea & absence of breath sound.

*On percussion on the low parts of the chest dull sound.

*These conditions always are bilateral in horses but may be unilateral in other spp. causing absence of movements of ribs on the affected side.

*In thin animals the intercostals spaces are bulged.

*Presence of jugular pulsation because the amount of fluid is great enough to cause compression on the artery.

Clinical pathology:

*Thoracic puncture will be followed by a flow of:

a- Clear serous fluid in hydrothorax.

b- Blood in cases of hemothorax “recent cases”.

*Bacteriological examination of the fluid, the fluid is negative but may contain proteins.

Necropsy finding:

In animals which die of acute hemorrhagic anemia resulting from hemothorax.. The pleural cavity is filled with blood “not clotted” & the clot has been broken down by the constant respiratory movements.

Hydrothorax is not fatal but it may accompanied by other fatal diseases.

Diagnosis:

Both diseases must be differentiated from:

*Pleurisy by absence of pain.

*Toxemia & fever by examination of an aspired fluid sample.

*Other space occupying lesions of thorax including tumor are not characterized by accumulation of fluid unless if the tumor have been implanted on the pleura.

Treatment:

Hydrothorax:

Aspiration of the fluid from the pleural sac, however the fluid reaccumulate rapidly.

Hemothorax:

1-parental coagulants.

2-Blood transfusion.

Diaphragmatic Hernia

Diaphragmatic hernia is rare in farm animals except in cattle especially in association with traumatic reticulo-peritonitis, here the hernia is small & not cause any respiratory disturbance except there is abnormal sound on chest. Occasional hernia can be occurred by foreign body. Congenital hernia is recommended in all spp.

Clinical findings:

- *Colic & dyspnea in horse.
- *Presence of intestinal sound in the thorax.
- *There may be a small fold projection from the chest wall.

Treatment:- surgically.

Pleurisy

Definition:

It means an acute inflammation of the pleura causing pain during respiration movements & manifested clinically by shallow & rapid respiration and signs of pain.

Etiology:

- *Traumatic perforation of the thoracic wall primary pleurisy.
- *In the course of some specific disease contagious bovine pleuropneumonia, pleuropneumonia of sheep & goats, pneumonia by caused by P. multocida & T.B in cattle & infect. Equine pneumonia & Strangles of horse.
- *Perforation of diaphragm by sharp foreign body.
- *Primary peritonitis spread of infections infected pleurisy.
- *Extension of infection from the lung.
- *Complication of traumatic pericarditis.
- *Rupture of esophagus due to severe obstruction.
- *It is permanent lesion in contagious pleuropneumonia.

Classification:

- *Primary pleurisy:- “rare broken rib” is due to traumatic perforation of the thoracic wall.
- *Secondary pleurisy:- (infective) is due to ineffective agent T.B. or toxic agent.

Pathogenesis:

- *In early acute dry stage contact & movements between parietal & visceral pleura cause pain respiratory disturbance shallow & rapid respiration.
- *The 2nd stage of pleurisy is characterized by the production of serofibrinous inflammation exudates. Collection of the exudate in the pleural sacs collapse of the ventral parts of the lung reducing the vital capacity of the lung interfering with gaseous exchange clinical signs may be restricted to one side of the chest.
- *The 3rd stage of pleurisy the fluid is absorbed & development of adhesion. In bacterial pleuritis accumulation of large amounts of pus toxins are produced by bacteria development of toxemia.

Clinical findings:

- *Shallow, rapid & painful respiration.
- *Abdominal respiratory movements.
- *There may be pneumonia accompanied by rales & increased vesicular sound.
- *Dyspnea & toxemia may appear.
- *Pleurisy is unilateral, movements of the affected side is restricted as compared with the other side.
- *Pain on percussion.
- *Shallow & short painful cough.
- *Extension of the inflammation to the pericardium.
- *Death occurs due to combination of toxemia & anoxia.
- *Rupture of adhesion excitation may occur & causes fatal hemothorax.
- *Painful cough & increased pulse & temperature.
- *On percussion of the chest area of the animals, show pain.
- *Respiration is accelerated & is wholly abdominal.
- *The animal stands with abducted elbow, to relieve pressure from lungs & pleura.
- *Loss of appetite, dullness depression.
- *There are 3 stages of pleurisy:

a- Dry stage:

- Auscultation * frictional sound.
- Percussion * dull sound.

b- Exudative stage:

The fluid is accumulated in the ventral part of sac line of demarcation “or pleuritic line which is horizontal line on percussion on the line * dull sound”. & on auscultation above the line * normal, vesicular sound. & below the line absence of sound because of exudates.

c- Adherent stage:

- On percussion * dull sound.
- On auscultation * frictional sound.

Clinical pathology:

- *Thoracic puncture to obtain a sample of inflammatory fluid for bacterial examination.
- *Radiological examination.

P.M.:

- 1-In early acute pleurisy * between the lobes of the lung, are found edema & hyperemia of the pleura & vessels are engorged with blood & presence shreds of fibrin.
- 2-In exudation stage * the pleural cavity contains excessive quantities of fluid containing flakes & clots of fibrin.
 - Thickened pleura, dark red color & collapse of the ventral parts of the lungs.
 - Pneumonia is usually present.
 - There may be “associated pericarditis.
- 3-In the adherent, stage:- There is adhesion connecting visceral & parietal pleura.

Diagnosis:-

- *From history of the case.

- *From symptoms.
- *Aspiration of the fluids by needle puncture.
- *Radiological exam.

Diff. diagnosis:

1-It must be differentiated from pneumonia, emphysema, hydrothorax & heamothorax.

Treatment:

- *As pneumonia.
 - *Apply, broad spectrum antibiotics.
 - *Aspiration of fluids by paracentesis.
 - *To reduce pleural percussion of fluids give dexamethazone 0 .1 mg / k.g B.W.
- N.B :** Diuretics are unlikely to aid in the removal of this or any other inflammatory exudate.