Studies On
The Respiratory Diseases of Bovines
with Special Reference to
Pneumonia

Thesis
Submitted to the
RAJENDRA AGRICULTURAL UNIVERSITY BIHAR
in partial fulfillment of the requirements
for the degree of
MASTER OF SCIENCE (VETERINARY)

BY
Naundra Bahada Singh
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Post Graduate Department of Pathology
BIHAR VETERINARY COLLEGE
PATNA,
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I certify that this Thesis has been prepared  
under my supervision and guidance, by Sri Narendra  
Bahadur Singh, a candidate for the degree of Master  
of Science (Vet.) with PATHOLOGY as major subject,  
and that it incorporates the results of his independent study.

( C.D.N. SINGH)
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(NARENDRA BAHADUR SINGH)
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CHAPTER - I

INTRODUCTION
INTRODUCTION

Animal Husbandry has been realised to play an important role in a nation's economy.

As a result of population explosion, today the world is facing great food shortage. Mal-nutrition and starvation pose the greatest concern in major world population. Protein, a vital nutrient and other ingredients of human food are mostly provided by animals. In view of the present acute shortage of animal protein much emphasis has been given for the production of better quality and quantity of animal products.

The crux of the problem lies initially in improving the quality of livestock and protect them against various diseases and disorders affecting the farm economy.

In many parts of the world, by constant efforts the productive efficiency of the livestock has been satisfactorily improved. In developing countries, livestock improvement is gaining more attention.

In pace of changing standard of domestication, veterinary profession is gaining more importance because of challenges due to various diseases to the farm enterprises. The devastating animal diseases such as rinderpest has been successfully controlled in most part of the world. Threat to livestock by several other diseases still poses great economic discrepancy to livestock industry. In the domain of livestock farm enterprise, genetic improvement and protection from diseases are equally important. The search of the disease is not a very simple affair, coordinated
efforts of many specialists such as, pathologist, epidemiologist, bacteriologist, parasitologist etc. are needed for scientific approach to the problem. It is only after the correct knowledge of the disease, the suitable control methods could be suggested.

Involvement of digestive and respiratory system causes more casualties and economic losses than any other condition in most parts of the world. In many livestock farms of India and Nepal, respiratory diseases primarily or in a complex form with digestive disturbances are the major killer of animals, especially young calves. Respiratory disorders affect the economy of farm from several angles viz., death of productive animals, death in calvehood, retardation in growth, reduction of productivity, latent infections or carrier states of the animal endanger their own life. The latter acts as a nucleus for the spread of disease in epizootic form, as and when natural or innate resistance of the animals breaks down due to some predisposing factors. Several zoonotics such as pulmonary tuberculosis and viral diseases like influenza and many other with aerosal nature of dispersal are constant hazards to human being. Some authors have reported recovery of PI3 virus in milk of cow.

Viruses, bacteria, parasites and fungi etc. have been studied in connection with respiratory lesions. Stress, is now believed to act in predisposing the animals to the disease conditions involving the respiratory tract.

Virus appears to be the key to the problem of many respiratory infections, because they are believed to be the primary infectious elements in the chain of events which result in depression and death of animals. Respiratory viruses are known to be capable of entering and damaging the intact healthy respiratory
epithelium. Bacteria such as *pasteurella* appears to lack this capacity and depends for their means of entry on lesions already present. Cellular debris and effusion provide a suitable medium for the multiplication and extension of the pathogenic effects (Collier, 1968).

Stress of long duration would be necessary to produce a depletion of general immunological defence. However, research on epidemiology of common cold in humans, Lidwell *et al.* (1965) demonstrated a significant correlation between the incidence of colds and the mean daily temperature and humidity four days prior to the onset of symptoms which suggests that the influencing stresses need only be of short duration. Perhaps, therefore, stress exerts its most important effects on the epithelial surfaces, particularly those of the respiratory and intestinal tracts. Rapid change of temperature and humidity could adversely depress the metabolism of epithelia and their associated immunologically competent tissue to such an extent that production of surface active antibodies resembling IgA of human could be insufficient to repel the infective challenge.

Stress of various types in relation to respiratory syndrom in field condition and experimentally infected animals have been intensively studied in recent years by many workers.

Enzootic pneumonia of calves and adult cattle is gaining importance in many countries. Both bacteria and virus have been studied to be responsible for this condition. Many workers have studied that shipping fever, a major cattle disease in U.S.A., is due to infection with PI$_3$ virus and *Pasteurella* sp. At least five important virus infections in bovines have been thoroughly studied by several workers. Infectious rhinotracheitis (IBR) virus,
Parainfluenza-3, Adenovirus, Reoviruses, Psittacosis-lymphogranuloma-venerum. Other viruses such as Rhinoviruses, Bovine viral diarrhoea-mucosal disease viruses etc. have been isolated from calves with respiratory affections. Many of these viruses have been found responsible for causing more than one condition. It is suspected that P.I.V. which causes abortion and encephalitis are identical with those causing respiratory diseases.

Most of the viruses cause mild form of respiratory illness which is later exaggerated by secondary invasion by bacteria. Diversity of bacterial isolates has been reported from the respiratory tract in normal as well as diseased bovines, such as Cocc, Escherichia, Pasteurella, Salmonella, Corynebacterium, Actinobacillus, Haemophiles, Neisseria, Proteus and Pseudomonas etc. Among the bacterial flora, Pasteurella sp. is associated with the more important condition in bovines "Shipping fever" in U.S.A. and other parts of the world and haemorrhagic septicaemia in Asian countries like India, Nepal etc. Great loss in bovines due to haemorrhagic septicaemia (Pectoral form) have been experienced in these countries. Both cattle and buffaloes are equally infected with this condition, as in shipping fever there seems some correlation between stress and pathogenesis of haemorrhagic septicaemia. This disease (H.S.) has generally a seasonal occurrence. In my experience of the field, the epidemic form of pasteurellosis has been noticed during the late rains. It is quite possible that stress in one or many farms may predispose the animals to infections. In August and September, a number of buffaloes are carried on foot or on trucks from distant Tarai to Kathmandu valley during Durgapuja celebration. After arrival of these animals in the hilly localities, a disease which
definite diagnosis. Bacterial isolation and transmission studies are made to support the pathological findings. Haematological picture to study the effects of the aetiological factors was carried out in some sick animals.

The survey of incidence of pulmonary diseases was carried out in certain districts of Nepal, Government Cattle Farm, Patna and local slaughter houses in Bihar with the object of elucidating the maladies affecting the bovine respiratory tract.

The present investigation has been aimed to throw some light on the various pathological conditions which are deliterious to livestock and also menace the livestock enterprise. The study of gross and microscopic changes in the lung tissue represent the correct picture of pulmonary disease conditions. These findings may serve as basis for further research on the various aspects of the disease condition of bovine respiratory tract. Attempts had also been made to isolate bacteria or other aetiological factors and transmission experiments were also conducted to correlate the pathological findings with the aetiological factors. Since the knowledge on the cause and pathological details of mycotic pneumonitis in cattle and buffaloes is very much imperfect. It has been my object in present study on the "respiratory diseases" especially to illustrate the natural progression of anatomic changes in the mycotic diseases of bovines.

Further, this work may help in understanding the correct disease position, which may also be of help in devising suitable control measures and in planning irredication schemes of the respiratory group of diseases prevalent in bovines. The present study may guide the veterinarians to adopt correct remedial measures in undertaking the treatment of above conditions in animals.
CHAPTER - II

REVIEW OF LITERATURES
REVIEW OF LITERATURES

A. DISEASES OF RESPIRATORY SYSTEM

Inflammation of nasal mucosa (Rhinitis):

The nasal mucosa is exposed to external environment. Atmospheric changes directly affect the nasal mucous membrane. This part of the body is always in contact with the various environmental factors such as viruses, bacteria, fungi, parasites, chemical elements, allergens, etc. The protection mechanism which consists of mucus secreted by goblet cells and the cilia protects the delicate mucous lining of the upper respiratory tract.

The inflammation of the nasal mucosa (rhinitis) may be differentiated according to its course, as acute or chronic or as serous, catarrhal, purulent, pseudomembranous or granulomatous depending upon the changes. The acute form starts as serous inflammation and later changes to other forms. The chronic or granulomatous form is manifested either by polypos swelling on the mucous membrane or by atrophy of the mucosa.

In acute rhinitis the epithelial cells show hydropic degeneration and loss of cilia alongwith hyperaemia and infiltration of inflammatory cells in the lamina propria. Acute form often progresses to catarrhal, purulent or pseudomembranous type depending upon the nature of aetiological factor and duration of inflammation. Granulomatous reaction in the mucous membrane often takes a longer course. Constant mild irritation produced by a variety of aetiological factors produces this condition. Atrophic rhinitis is a separate type of reaction.
The nasal mucous membrane gets involved in some systemic diseases and through the haematogenous route more often as accompaniment of several diseases.

Granulomatous reaction due to higher bacteria, fungus or some metazoan parasites have been studied by several workers in past. Nasal schistosomiasis is one of the most frequent conditions in some countries. Rao (1933) and Malkani (1953) reported about nasal schistosomiasis in Indian cattle.

Roberts et al. (1963) studied three cases of maduromycosis of bovine nasal mucosa. Histology revealed eosinophilic granulomatous proliferation of nasal mucosa. Granule formation was also seen. Fungal elements composed of chlamydospores and hyphae were observed.

Martin (1969) reported a condition resembling porcine atrophic rhinitis in young cattle. Inflammatory changes in the nasal mucosa, increased formation of mucopurulent exudate. Pathologic changes in the nasal cavity of the cattle were similar to those found in porcine atrophic rhinitis.

Inflammation of other parts of the upper respiratory tract:

Schroeder and Moys (1954) reported acute upper respiratory infection of dairy cattle with rise in temperature. Virus or a virus-like organism was suspected to be the causative factor.

McIntyre (1954) described an outbreak of acute respiratory disease with temperature 104-106°F. Lesions included severe haemorrhagic tracheitis, bronchitis and numerous small areas of necrosis in larynx and pharynx. On inoculation of blood or sputum of diseased animals to calves by subcutaneous intra-nasal or conjunctival routes
the calves became sick. Calves in contact with infected calves also picked up infection. The lesions in the infected calves were hepatization of cardiac lobes, moderate scattered petechiation in trachea. Congestion of sinuses and nasal passage, petechiae and echymosis on epiglottis were also present.

In some instances, the infection starts at the nostrils and spreads gradually along the pharyngial, laryngial, tracheal mucous membranes and later the infection even involved the lungs.

**DISEASES OF LOWER RESPIRATORY TRACT**

There are a very few reports available on the congenital diseases in the bovine lungs.

**Pathology and aetiology of pneumonia in bovine:**

Pneumonia, pneumoenteritis and pneumonia associated with some other conditions constitute serious disease problems in bovines. Such conditions are among the diseases responsible for severe calf and adult mortality. McKercher (1968) categorised pneumonia as caused by viruses only (IBR, BVD-MD etc.) those which are apparently triggered by virus or viruses and in which secondary bacterial infections are responsible for severity of condition - e.g. infections due to adeno, rio and rhinoviruses. The last type is caused solely by bacteria.

Fatal pneumonia nearly always results from secondary bacterial infections. Histological descriptions by earlier workers have been very similar to severe exudative bronchopneumonia. Distinguishing lesions present, however, must have been obscured by reaction to secondary bacteria.
Various attempts to classify pneumonia in both human and animals were made in past. The classification has been based on anatomical distribution of the lesions such as lobar, lobular and interstitial pneumonia, on the gross appearance of the exudate - catarrhal, purulent, haemorrhagic and fibrinous, the route of entry of infection viz. aerogenous, haematogenous; duration of the disease process as acute, subacute and chronic, and on the basis of aetiological agents like viral, bacterial, fungal parasitic, etc.

Omar (1966) recently suggested the classification of pneumonia as exudative and proliferative (atypical) based on the predominant histological lesions; in addition to this, pneumonia caused by viruses has been taken as separate intity.

In the present enquiry, Omar’s classification (1966) has been followed for surveying the review of literature. The classification of pneumonia given by him is as follows:-

(1) **Exudative pneumonia** :-
   
   (a) Catarrhal, and/or purulent (*C. pyogenes* as important causative agent).

   (b) Fibrinous (*Pasteurella sp.* as important causative agent).

   (c) Embolic pneumonia (accompanied with septicaemic state).

(2) **Proliferative pneumonia (Atypical)** :-

   (a) (i) Epithelializing pneumonia

   (ii) Giant cell pneumonia (dealt in viral type).

   (b) Cuffing pneumonia

   (c) Granulomatous pneumonia
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2. **Proliferative pneumonia (Atypical) :-**
   
   - (a) (i) Epithelializing pneumonia
     (ii) Giant cell pneumonia (dealt in viral type).
   - (b) Cuffing pneumonia
   - (c) Granulomatous pneumonia
(3) **Specific virus pneumonia:**

(a) Due to infectious bovine rhinotracheitis (IBR) virus.

(b) Due to adenovirus.

(c) Due to parainfluenza-3 virus.

(d) Pneumonia due to Psittacosis-lymphogranuloma venerum (PLV) organisms.

1. **Exudative pneumonia:**

Smith (1921) grouped cases of bronchopneumonia into acute, chronic and intermediate types. In young calves, it was associated with *B. actinoides*, *B. pyogenes*, *B. bovissepticus* and staphylococci, streptococci in the later stage.

Carpenter and Gillman (1921) described three types of pneumonia in calves such as congestive, catarrhal and purulent forms. The congestion started from the anterior lobe and spread posteriorly in congestive pneumonia. The lesions described for catarrhal form were dependent on the type and duration of infection. In purulent form one or more lobes were seen with tiny abscesses ranging from 1 mm. to 1 cm. in diameter and pleuritis was often seen in the last two forms. Micrococci, *C. pyogenes*, *E. coli* and streptococci were isolated.

Schmid (1933) studied an out-break of calf pneumonia and recovered *C. pyogenes* from the internal organs of most of the cases.

Shirlaw (1935) reported in calves areas of hepatization in middle and anterior lobes of the lungs and observed serofibrinous exudate distending the pleural sac. Histological picture in acute cases simulated acute form of pasteurellosis. In the terminal phase of the infection, bronchitis followed by localised catarrhal
pneumonia terminating in central necrosis and abscess formation were seen. Large number of lymphoid nodule formation was seen around the bronchioles in chronic cases.

Smith (1935) studied cases of pneumonia caused by Brucella abortus as causative or predisposing factor for secondary bacterial infection in foetus and calves.

Lovell and Hughes (1935) observed 12 percent of cases of consolidated lungs with areas of necrosis and suppuration and recovered C. pyogenes from these calves. In 6 percent of cases, Haemolytic coccobacillus was isolated.

Weischer (1935) described infectious bronchitis in cattle. Lesions of catarrhal bronchopneumonia, pleurisy, oedema, atelectasis and interstitial emphysema were observed at autopsy.

Lebedev (1937) described pneumatic lesions from which C. pyogenes and staphylococci were recovered.

Thorp and Hallman (1939) studied 40 cases of acute pneumonia in calves with serofibrinous exudate in the alveoli. Extensive consolidation superimposed on chronic bronchopneumonia and lesions of abscess formation and necrosis. B- streptococci, Bact communior, and Staphylococcus aureus were isolated from 26 cases of pneumatic lungs.

Lovell (1939) reported lesions of pneumonia along with whitescour in calves. Consolidation, suppuration and necrosis were observed in the lungs. Haemolytic coccobacillary organisms and C. pyogenes and Brucella abortus were isolated.

Lamont and Kerr (1939) reported lesions of pneumonia
caused by *H. influenzae* and isolated *C. pyogenes* from the chronic cases. 

Sandars (1939) studied enzootic form of bronchopneumonia in calves. Its incidence took place mostly in warm months in over crowded and insanitarily housed calves. *E. coli* and staphylococci *pasteurella sp.* were recovered from lung lesions.

Thorpe et al. (1942) described histological lesions of bronchopneumonia in calf and *H. suis* like organisms were constantly isolated. No conclusive result was obtained in experimental infection.

Langham et al. (1942) studied bronchopneumonia in 9 cattle, 12 sheep and 10 pigs. Lesions were found mostly in apical, cardiac, intermediate and anteroventral portions of diaphragmatic lobes. Histological examination revealed, acute, subacute and chronic types of lesion. Acute type had purulent bronchitis and respiratory bronchioles plugged with exudate containing numerous polymorphs and few monocytes. Alveolus had various degrees of inflammation. Haemorrhagic serofibrinous exudate containing some polymorphonuclear leucocytes was present. In subacute forms, there was proliferation of fibroblasts and fibrosis of various structures was present in addition to the lesions of acute pneumonia. Lung and pleura had generalised fibrosis in chronic cases. B-streptococci were isolated from 4 cases.

Shigley and Thorp (1943) observed in calves bronchopneumonia, pleuritis and abscesses in apical, cardiac and parts of diaphragmatic lobes. Microscopically the lesions were acute interstitial bronchopneumonia and bronchitis. *H. influenzae* was isolated from the lesions.

Langham et al. (1944) observed that 33.8 percent of bovines
examined at autopsy had pneumonic lesions and incidence was high in young animals.

Blakemore (1945), in two outbreaks of calf pneumonia, recovered organisms resembling *Bacillus actinoides* which induced pneumonic lesions in susceptible calves on intra-nasal inoculation.

Gunning (1946) reported infection of lungs with an organism resembling *Bacillus (Actinobacillus actinoides)* in an outbreak of parasitic bronchitis in calves.

Levi and Cotchin (1950) observed bronchopneumonia affecting the anterior and ventral portions of the lobes of lungs. Abscess formation was seen in subacute and chronic stages. Much lobular fibrosis with adhesion of pleura in the lungs was also noticed. Organisms resembling *B. actinoides* in the initial stage or *C. pyogenes* in the later stage were seen.

Jennings and Glover (1952) reported enzootic pneumonia of calves with lesions of suppurative bronchopneumonia from which *C. pyogenes* was isolated.

Watt (1952) isolated organisms of haemophilus group in two outbreaks of calf pneumonia with lesions of acute necrotizing bronchopneumonia and bronchiolitis with infiltration of polymorphs.

Harbourne et al. (1965) studied haemolytic and non-haemolytic *E. coli, Salmonella dublin, C. pyogenes* and *Proteus vulgaris* from bovine lungs.

Lesions simulating pasteurellosis with the recovery of *pasteurella sp.* from pneumonic bovine lungs has been described by Carpenter and Gillman (1921), Smith (1921), Tweed and Edington (1930), Lovell and Hughes (1935), Sanders (1939), Lovell (1939), and

Willems (1931) gave a detailed account of contagious bronchopneumonia of bovines (pectoral form of pasteurellosis). According to him the nature of infection had been sometimes spontaneous in a farm and sometimes outbreaks from outside sources following the introduction of an affected bovine. Long journey, fatigue and debilitating conditions were regarded as predisposing factors and causative organisms normally as saprophytes of respiratory or intestinal tract. Some unknown factor was thought to exalt the virulence giving rise to "spontaneous infection" in cattle. He further noted that organisms showed marked preference for the species as well as certain age group of the animals. In one instance, pigs were affected but no bovines. Some strains attacked only calves. He also studied strain of organisms adapted to different species of host. The pathology described was yellow or reddish serofibrinous exudate in thoracic cavity, petechie in pleura, various stages of hepatization, oedematous thickening of intra-lobular tissue (marbled appearance).

Grahm (1953) observed in majority of cases of "shipping fever" bilateral fibrinous pneumonia with bronchitis. Pneumonic portions showed red and gray hepatization with oedema of interlobular septa.

Carter (1954) in an outbreak of "shipping fever" described lesions of acute bilateral bronchopneumonia from which Past. haemolytica and Past. multocida were isolated.

Carter and McSherry (1955) carried out bacteriological examination of cattle suffering from "shipping fever" and isolated
Past. haemolytica from majority of cases.

Carter (1956a) induced pneumonia in calves by intra-nasal injection with young culture of Past. haemolytica.

Carter (1956b) postulated that cattle with chronic pulmonary infection were more susceptible to Past. multocida and Past. haemolytica.

Palotay and Newhall (1958) described an outbreak of pneumonia in calves and recovered Past. haemolytica.

Carter and Rowsell (1958) isolated Past. multocida from the lung of veal calves having bronchitis infiltrated with fibrin cellular debris and polymorphus in outbreaks of enzootic calf pneumonia.

Pneumonia with infiltration of fibrinous exudate in the lung is believed to be caused by pasteurella or mycoplasma organisms (Jubb and Kennedy, 1963, and Omar, 1966).

Trapp et al. (1966) exposed 34 calves to various combinations of physical stress, Pasteurella sp., Myxo virus parainfluenza-3 and PPLO. Thirteen had gross lesions of pneumonia and 5 were doubtful. Histological picture revealed pneumonia in 17 calves and one had pneumonitis.

The group receiving PI-3 virus, Past. sp. and stress had lesions simulating field case of "shipping fever".

2. Proliferative pneumonia:

According to Jubb and Kennedy (1963), bovine atypical interstitial pneumonia (BAIP) being a distinct clinical and pathological syndrome, is not in terms of one specific cause or a specific disease & as postulated, there is more than one beginning to the
Jarrett (1954-56) described two types of pneumonia in calves namely epithelializing and cuffing pneumonia. The former was characterized by widespread cuboidal epithelialization, and giant cell formation as salient features. Extensive oedema and hyaline membrane formation were also noticed.

Jarrett (1957) reported hyaline membrane in the respiratory passage of cattle dying with severe respiratory distress especially in parasitic bronchitis.

Runnel et al. (1960) suggested that majority of interstitial pneumonias are caused by viruses.

Luginbuls (1960) reported chronic interstitial pneumonia and adenomatosis in cattle inoculated with bacteria free filtrate.

Van Rensburg and Every (1961) described enzootic pneumonia in calves prevalent in wet summer months in calves 1-4 months lesions were thickening of alveolar walls with peribronchial cuffing.

Schiefer (1962) reported chronic interstitial pneumonia in cattle with transformation of the alveolar epithelium.

Blood (1962) while describing interstitial pneumonia, pulmonary adenomatosis, emphysema, and oedema of cattle incriminated allergy, and dust etc. as probable cause for them. Jubb and Kennedy (1963) attributed interstitial pneumonia to allergic condition.

Rathore and Singh (1965) described the interstitial pneumonia in buffaloes as thickening of alveolar septa formation of hyaline membrane, oedema in the alveoli and alveolar ducts and proliferation of alveolar epithelial cells. Seventythree percent of the calves showed interstitial pneumonia.
In majority of cases, lesions were confined to antero-ventral lobes of the lung. Lungs failed to collapse, had imprint of ribs and some lobes were pale while others were hyperaemic and red. Intermingling of normal, atelectatic and emphysematous lobules and thickening of interlobular septa were noticed.

Arndt (1965) studied hyaline membrane in the lungs of 19 cattle out of 214 pneumonic cases.

Chemical agents as the cause of interstitial pneumonia have been supported by Omar (1966).

Omar and Kinch (1966) described atypical interstitial pneumonia, a condition resembling fog fever in young calves (2-6 months of age) 13 times since the beginning of 1965 in outbreaks in 11 farms. Death took place mostly from October to February with mortality of 38 percent. Lesions were those of emphysema consolidation, septal oedema, frothy exudate in trachea and bronchioles with several lung conditions, towards the final stage. Bronchiolar and alveolar, cuboidal epithelialization, septal cell proliferation and reticular hyperplasia with resulting fibrosis were noticed.

Conway (1969) reported atypical pneumonia in grazing calves on two farms. In view of the presence of mast cells and eosinophils in the interlobular tissue, author considered this condition to be due to hypersensitivity.

Sastry (1969) described interstitial pneumonia, a condition in which the alveolar septa is affected. Though some exudate may be found in the alveoli, by far the most of the changes (Proliferative) are found in alveolar septa. The following are the main characteristics of interstitial pneumonia:
(1) Thickening of alveolar septa by -  
   (a) Exudate - serous or fibrinous  
   (b) Infiltration by leucocytes  
   (c) Formation of new connective tissue  

(2) Proliferation of epithelial cells of the alveoli  

(3) Formation of hyaline membrane in the alveoli and over the alveolar ducts  

(4) Almost complete absence of neutrophils in the exudate.  

Smith (1929), Blakemore (1945), Levi and Cotchin (1950), Jenning and Glover (1952), and Van Rensburg and Every (1961) described lesions of peribronchial cuffing by lymphocytes in cases of pneumonia.

Jarrett et al. (1953) described cuffing pneumonia in cattle of all age groups (most prevalent in 3-12 months age group). They suggested close resemblance of these diseases to gray lung disease of mice, spontaneous pneumonitis of guineapigs and virus pneumonia of pigs. Lesions of peribronchial lymphoid hyperplasia had been coined by authors as "cuffing pneumonia". Virus as probable cause was indicated.

Jubb and Kennedy (1963) described cuffing pneumonia in cases of enzootic calf pneumonia and PLV as probable cause.

Stevenson (1967) reported during the investigation of 27 animals with lesions resembling cuffing pneumonia. The possibility that the condition was a sequel to previous bronchopneumonia was discussed.

Cole (1970) studied 13 outbreaks of calf pneumonia and noted that parts of the lungs were slightly congested. Histological
lesions of mild peribronchial cuffing and lymphoid infiltration of the bronchiolar submucosa were noticed. Evidence of recent alveolar involvement was the presence of pseudoepithelialization, free alveolar macrophages and other inflammatory cells.

(3) **Specific virus pneumonia:**

This group of pneumonia is gaining more attention in the recent years.

(a) **Myxovirus:** Kromayer (1889) described giant cell pneumonia in children associated with measles and pertussis. Since that time, many workers have studied giant cell pneumonia with cytoplasmic and intra-nuclear inclusions in giant cells, alveolar and bronchial lining cells. Some earlier workers believed this condition to be due to vitamin A deficiency.

[W. Adams (1941) studied a case of primary virus pneumonitis with evidence of cytoplasmic inclusion bodies and their association with measles.

[P. Pinkerton et al. (1945) studied 5 cases of giant cell pneumonia in infants and found eosinophilic cytoplasmic or nuclear inclusions in 4 cases and in the 5th only cytoplasmic inclusions.

[C. Cordy (1949) and Watrach (1957) described giant cell pneumonia associated with distempering in dogs.

[A. Abnanti et al. (1960) reported 15 recoveries of PI-3 virus from 7 outbreaks of respiratory diseases in calves and adult cattle and 3 from the nasal secretions of apparently normal calves. They cited a report prior to recovery of PI-3 virus from cattle about an antigenically indistinguishable virus to be common source of
infection in man and responsible for respiratory illness in infants and young children.

Saunders (1961) studied aetiology and pathogenesis of "shipping fever" and experimentally supported the role of PI-3 in this condition.

Vardaman et al. (1962) studied the response of calves to PI-3, *Past. haemolytica* and *Past. multocida* and observed its transmission in test calves. Heddleston et al. (1962) studied that *Past. haemolytica*, *Past. multocida* and PI-3 were capable of transmission by contact under field condition from calves with shipping fever to the susceptible. Schipper (1963) studied the effect of shipping fever vaccine (*Past. multocida*, *Past. haemolytica* and PI-3) and achieved encouraging results. Hamdy et al. (1964), with transmission, studied that disease syndrome was not induced in calves given virus alone.

Nikolaev and Nikolaeva (1964) isolated and studied cytopathogenic agent from calves with respiratory disease. Acidophilic, cytoplasmic inclusion bodies in the cylindrical cells of bronchial mucosa were observed.

Hamdy et al. (1965) attempted immunizing cattle against shipping fever with different combinations of vaccine and concluded that combination of *Past. sp.*, PI-3, IBR, or PLV agents and physical stress were capable of producing disease symptoms.

Omar (1965) studied cytopathological effect of PI-3 in tissue culture and reported nuclear inclusions.

Dawson et al. (1966) reported serological evidence of PI-3 virus in the purchased calves although no virus could be isolated.
Blanco Loizier (1966) associated the disease complex involving respiratory and/or digestive system with PI-3 virus.

Singh and Baz (1966) isolated PI-3 virus from Egyptian water buffaloes. Singh and Baz (1967) studied PI-3 virus from buffaloes and found eosinophilic inclusions (both cytoplasmic and nuclear) in calf buffaloes and cammel kidney cells and only cytoplasmic inclusions in M.S. cells.

Burroughs (1967) recovered PI-3 virus from newly arrived cattle (4.5% during summer and 13.5% in winter). From the cattle with respiratory signs, virus was isolated 15.8 percent in summer and 66.6 percent in winter. The ratio of IBR and PI-3 was 1:1.55. James (1967) isolated PI-3 and other viruses from intestinal and respiratory tract of bovines.

Stevenson (1967) studied pathology of pneumonia in intensively reared calves. Morphologically, two distinct types of pneumonia were observed in 9 cases of which 6 had lesions resembling inclusion body pneumonias which were thought to be due to PI-3 virus.

Campbell and Martin (1968) noticed in a bullock with pneumonia marked interstitial oedema and emphysema. Cytoplasmic inclusions were seen in bronchial and alveolar epithelium and PI-3 virus was also isolated.

Balan and Iyer (1968) studied 6 cases of pneumonia with cytoplasmic and nuclear inclusion bodies in adult buffaloes. It was suggestive of PI-3 infection.

Phillip (1970) reported on the basis of serum survey 20-25 percent of outbreaks of respiratory disease to be due to PI-3 virus.
Arora (1971) reported pathology of giant cell pneumonia enteritis syndrome in an outbreak in Government Livestock Farm, Hisar among buffalo calves aged 3-8 months during the month of December, 1970 and March, 1971. The histological picture consisted syncytial giant cells which were either lying free in the lumen or still attached to the alveolar walls. Septal cell proliferation at places causing discontinues lining of the alveolar septa (Pseudoepithelialization). Moderate to heavy peribronchial and perivascular lymphoid cell infiltration was present in some areas. Stained with Giemsa's method, a few acidophilic pleomorphic cytoplasmic inclusions simulating viral inclusions were detected in some alveolar and bronchiolar macrophages. Possibility of giant cell pneumonia enteritis associated with Myxovirus infection was suspected.

(b) Infectious bovine rhinotracheitis (IBR) virus:- Schroeder (1945) reported acute respiratory infection of cattle in sporadic form. He discussed its resemblance to the infectious bronchitis described in Europe and Japan earlier. The disease could be reproduced experimentally. P.M. lesions included necrotic lesions in upper respiratory tract and hepatization and emphysema in the lungs.

Madin et al. (1956) isolated an infectious agent (IBR) from nasal washings and scrapings and subsequently the virus was isolated from lungs, nasal discharge and faeces of calves with respiratory illness.

McKercher et al. (1957) described clinical and epidemiological picture of bovine rhinotracheitis virus disease of cattle. Formation of pseudomembrane over the tracheal epithelium and erosion were described.
Gillepsic et al. (1957) observed symptoms produced by intra-nasal injection of virus (IBR) in calves. This virus was believed to be one of the exiting agents of "shipping fever" in U.S.A. (McKercher, 1964).

Tylar and Ramsey (1965) noticed isolates from bovine mucosal disease viral diarrhoeas showed similarities in clinical, haematological and pathological responses produced by IBR.

Moretti et al. (1964) studied clinical cases of respiratory illness in 120 calves aged 8 days to 4 months and isolated IBR agent evidenced by cytopathic effect and intranuclear inclusions. The virus was confirmed to be of herpes group. Blanco Loizelier (1966) studied pathogenesis associated with pneumoenteritis and pointed out IBR as one of the causative factors. Bureoughs (1967) found IBR to be most frequent among viruses in bovines.

Barthe et al. (1969) studied encephalitis and conjunctivitis in association with respiratory involvement in a herd of 300 calves. The affected calves were under 60 days. IBR virus was isolated. Some cases of central nervous symptoms without respiratory tract involvement were reviewed.

Mohanty et al. (1969) found that in an experimental study involving both intratracheal and intranasal inoculations with some tissue culture of IBR virus, animals showed clinical signs of disease. Those given orally and nasally and controls, remained clinically normal. All the male calves of 6-8 week old were used. Calves with clinical signs had pneumonic lesions both macroscopically and microscopically.

Phillip (1970) described IBR virus to be of world wide
distribution and correlated the virus with conditions as vulvovagi-
nitis and balanoprophritis, encephalitis and epizootic abortion, 
only one serotype was thought to be involved. Pathogenicity differed 
from strain to strain.

(c) Adenovirus :- Klein et al. (1959) first isolated 
adenovirus from the faeces of normal adult cow and further this 
virus was recovered from the normal calf (Klein et al., 1960).

Darbyshire et al. (1965) isolated adenoviruses from the 
eye of normal cow in the course of longitudinal survey of viruses 
associated with respiratory diseases in cattle. It was found to 
have similar properties as adenovirus-type-5.

According to Aldasy et al. (1965) high incidence of pneu-
enteritis has been observed in Hungary amongst calves (2 weeks to 
4 months). Losses were upto 60 percent out of 214 samples examined, 
78 strains of adenoviruses were isolated. Adenovirus is suggested 
to be primary cause of pneumoenteritis.

Darbyshire et al. (1965) gave detailed description of this 
pneumonia in calves, the salient features were proliferative bronch-
iolitis followed by necrosis and bronchiolar occlusion which result-
ed in extensive alveolar collapse. Peribronchiolar and perivascular 
accumulation of lymphocytes and plasma cells occurred with marked 
cuffing. Nuclear inclusions were present in the bronchiolar epithe-
lium, in septal cells and cells of the bronchial lymph nodes. Early 
inclusions were stated to be eosinophilic and Feulgen-negative 
granules within enlarged nuclei. After the collection of the granu-
les, large intensively basophilic and Feulgen positive mature inclu-
sions were described. According to Omar (1966), 17.6 percent of 125
outbreaks showed pneumonia characterised by proliferative and necrotizing bronchiolitis and by the presence of inclusions, he classed them as adenovirus type pneumonia.

Cole (1970) isolated 11 strains of adenovirus representing two serotypes in calves in Australia. On the basis of serum survey, Phillip (1970) reported 20–25 percent outbreaks of respiratory diseases due to adenovirus. Lesions described by him were areas of consolidation, collapse and emphysema. The histological picture presents proliferative bronchiolitis with necrosis and bronchiolar occlusion resulting in alveolar collapse. Nuclear inclusions were found in bronchiolar and septal cells.

(d) Reoviruses:— Rosen and Abinanti (1960) isolated first bovine reovirus from the faeces of normal calf which was identical serologically to reovirus type-3. Rosen et al. (1963) observed that inapparent infection was widespread in cattle when tested serologically.

Dawson et al. (1966) reported the association of reovirus with outbreak of respiratory disease in calves in U.K.

Lamont et al. (1966ab) reported development of lesions of pneumonia without clinical manifestation in clostrum deprived calves. The lesions described were proliferative reaction with minimal exudation. Involvement of both entodermal and mesodermal elements giving rise to proliferative bronchiolitis, alveolar epithelialization, pseudoepithelialization and interseptal cell hyperplasia and inclusions were not observed. Peribronchiolar and perivascular lymphoid hyperplasia were noticed.

Phillip (1970) described 20–25 percent of calf respiratory
disease outbreaks which were diagnosed to be due to reoviruses.

(e) Psittacosis-lymphogranuloma-venerum (PLV) group of organisms:— Bovine strain of this organism was first isolated by York and Baker (1951) from the faeces of apparently normal calves. Subsequently, Motumoto et al. (1955) studied bovine pneumonia in relation to PLV group of organism in Japan. Lesions were in apical, cardiac and intermediate lobes. Bronchitis, peribronchiolitis, intra-alveolitis and cytoplasmic inclusions were observed microscopically. The term "bovine pneumonitis" was proposed for bovine respiratory infection caused by PLV group.

Paloty and Christensen (1959) reported respiratory infections in bovines associated with PLV group. Death in them occurred from bronchopneumonia.

Omori et al. (1960) reported PLV organisms associated with encephalomyelitis and pointed out that they also produced respiratory and intestinal syndromes in cattle.

White (1965) studied in experimentally infected calves at slaughter with extensive red consolidation of apical and cardiac lobes and a tracheobronchitis with copious mucopurulent exudate. Elementary bodies were demonstrated in smears from respiratory tract.

Foggie et al. (1965) found out PLV in calves dying in an outbreak of respiratory disease. Dymel (1965) isolated organisms of PLV group from calves with acute bronchopneumonia. There were fibrous pleuritis, bronchopneumonia and haemorrhagic lymphadenitis. Inclusions and elementary bodies were demonstrated in smears of lungs.
Omar (1966) showed that histopathological picture in psittacosis-lymphogranuloma-venerum pneumonia was that of an interstitial bronchopneumonia. Blanco Loizelier (1966) indicated the association of PLV as among the causative agents of pneumoenteritis of cattle.

Other viruses:

James (1967) discussed the significance of viruses isolated from intestinal and respiratory tract. Other viruses isolated from lungs or nasal secretions of calves with respiratory illness include - influenza viruses (Shope strain) (Romvary et al., 1962). Omar (1966) isolated from pneumonic lungs of calves four agents which produced cytopathic effect typical of entero-viruses. The pathogenesis and aetiological importance of these viruses are yet to be investigated.

Granulomatous pneumonia:

Pneumonia in bovines may further be considered on the basis of different aetiological factors elucidating chronic proliferative cellular responses. Granulomatous pneumonia has been reported in cattle by Ligniers and Spitz, 1902; Mangrulkar, 1938, and Shahan and Davis, 1942.

Smith and Jones (1966) grouped various aetiological agents (higher bacteria or fungi) known to provoke tissue reactions somewhat similar in nature. The pathological condition was designated as "Infectious granuloma". The infection granuloma was considered to be of importance from diagnostic point of view.
The infectious nature of tuberculosis and its transmissible nature were experimentally proved through inoculation of tuberculous material from both man and cattle in the guineapigs in the middle of 19th century.

Koch (1882) for the first time actually demonstrated the causative organisms for tuberculosis.

Friedberger and Frohners (1903) described the anatomical changes in bovine tuberculosis mostly in lung (Phthisis) and serous membrane (grape disease).

Mitter (1910) noticed one case of tuberculosis in buffalo at Calcutta with only lungs lesions and he remarked that this condition may also be common in other parts of India.

Eber (1917) observed tuberculous lesions in 18 percent buffaloes at Budapest.

Soparkar (1925) studied a case of tuberculosis in a male buffalo at postmortem at Mukteswar.

Soparkar and Dhillon (1931) reported that 23.6 percent buffaloes at Lahore slaughter house showed either tuberculous lesions or reacted to tuberculin. Maik (1932) described lesions in both lungs and mediastinal or bronchial lymphnodes of she buffaloes. Carpano (1934) studied tuberculosis in buffaloes in Egypt abattoir survey at Cairo showed 60 percent older buffaloes and 9 percent calves slaughtered suffer from tuberculosis.

Datta (1935) described the prevalence of tuberculosis
among cattle in India and discussed the possibility of human infection through milk of cows and buffaloes.

Anthony (1935) surveyed the distribution of human tuberculosis in Wales and evidence indicating the proportion of cases of bovine origin.

Panisset and Jalabert (1936) observed in 30 sets of adult bovine tuberculous lungs that 3 cases showed lesions in one lobule only, in 9 cases lesions were scattered throughout the lungs, in 18 of the 30 cases no lesion was observed in apical lobe and all the others were affected. They noted that tuberculous lesions are usually irregularly spread throughout the lungs.

Nicolaus (1936), while studying the forms and genesis of tuberculosis, found that in late generalisation the lymphnodes were often not involved and in early generalisation tuberculous changes were invariably noticed.

Dobberstein and Wilmes (1937) studied pathogenesis and histogenesis of pectoral tuberculosis in calves.

Weers (1939) pointed out that 44 percent slaughtered cattle had primary lung lesions, and chronic pulmonary tuberculosis was the common form.

Innes (1940) described pathogenesis and comparative pathology of tuberculosis in animals. Three main stages were recognised in human being, viz. primary complex, postprimary disseminations and reinfection with production of isolated tubercles. He pointed out that pathogenesis in animals was analogous to three stages of human. He noted the disease of late generalisation not only in lungs but also in other organs.
Medlar (1940) studied 520 pairs of tuberculous lungs from abattoir. The lesions were cut and he classified them as soft caseous, fibro-caseous, calcified and cavitating types. More foci were on the dorsal aspect of the lung than ventral, particularly on caudal portion of diaphragmatic lobes. The haphazard distribution of lesions in the lungs was suggested to be due to aerosol infection and in some chronic progressive diseases, cavitation was also seen.

Iyer (1944) noted 5 percent tuberculosis out of 500 animal examined. According to him most of the lesions were confined to thoracic cavity either in lymphnodes alone or in the lung tissue suggested aerosal mode of infection.

Stamp (1944) said that spread of tuberculosis was by enlargement of initial lung lesions and subsequently by bronchial spread. The primary lung lesions of adult bovines were responsible for progressive pulmonary tuberculosis. In four cases, lesions healed with fibrosis, calcification and ossification, but frequent caseation, liquefaction and subsequent spread gave rise to progressive pulmonary tuberculosis. Others were groups of caseous nodules, caseous lesions with cavitations and small haematogenous scattered lesions of miliary tuberculosis.

Lall (1946) suggested that the lesions in buffaloes were different from cattle. In buffaloes creamy white foci of caseation with concentric layers of calcification was less frequent with marked tendency towards encapsulation.

Stamp (1948) reviewed the anatomical consideration of bovine thoracic tuberculosis and the occurrence and distribution of primary lung lesions in 100 bovine thoracic tuberculosis, with anatomical relationship of lung and lymphnodes. The pathway of infection
the dissemination from primary infection and pathogenesis of bovine tuberculosis were discussed. He concluded that no portion of lung failed to show lesions but more frequent the lesions were in diaphragmatic lobes commonly at dorso-caudal aspects. Both lungs were equally involved. Endogenous spread was mostly by bronchogenic route. The portal of entry for primary infection was inhalation.

Plaske (1953) discussed the clinical significance of different stages in mycobacterium tuberculosis infections and of different pathological types of the disease in domestic animals.

Hagan and Bruner (1957) described the lesions of caseous-calcareous mass located in the anterior lobes of lungs. "Pearl disease" was described as a form of tuberculous pleuritis or peritonitis.

Dwivedi and Singh (1966) studied pulmonary tuberculosis in buffaloes at Gorakhpur, Mathura and Kanpur abattoirs. The lesions were mostly nodular and confined to diaphragmatic lobes.

Kuppuswamy and Singh (1968) reported incidence of tuberculosis in the Tharparkar breed of cattle and found increasing inciden-
ce of T.B. in the older animals.

Cassidy et al. (1968) described Mycobacterium avium infec-
tion in cattle. The organisms were isolated from normal and tuberculo-
sous tissues.

Lall et al. (1969) reported incidence of T.B. 1.93 % among cattle and 6.39 % among buffaloes.

Singh and Prasad (1971) reported high incidence of T.B. in Tharparkar cattle and observed well marked lymphocytosis in the tuberculin positive cattle.
PULMONARY MYCOSIS

There seems to be no published information on mycotic pneumonia in Nepal.

It is said that mycotic diseases occur often as secondary invaders after bacterial infection and antibiotics are largely responsible for the increase in the fungal diseases in animals. Antibiotics cause indigestion especially in ruminants, dysentery and mycosis are frequently reported due to the restrain on beneficial bacteria antagonist to fungi.

According to Davis and Anderson (1955), the infection by mycotic elements can take place either by way of digestive or respiratory tracts. They isolated branching nonseptate (mucor species) which proved pathogenic for white mice. Histopathological changes were presence of necrotic foci, dense zone of inflammatory cells, lymphocytes and plasma cells, eosinophils, epitheloid cells, many giant cells of Langhan's type and nonseptate hyphae. Rosette formation in two out of 11 cases was reported.

McCarty (1956) described a widespread disease in cattle showing clinical symptoms like "chronic pneumonia" which was referred as "lungers". It was observed that symptoms got intensified after antibiotic therapy. After treatment, symptoms of pneumonia without febrile reactions were persistent. The disease was debilitating and frequently diarrhoea was noted. Postmortem revealed consolidation of the lung with nodules (pin point to 20 mm. in diameter). Scar formation was not predominant. Candida albicans was isolated from these foci. In terminal moniliasis lumen from bronchi to terminal
bronchus was filled with brown foam, which yielded yeast bud forms. The author has described two of the fungal isolates (i.e. Monilea and Mucor). These were found to be pathogenic in nature.

Gitter and Austwick (1959) described mucor mycosis and Moniliasis in a litter of suckling pigs.

Anasworth and Austwick (1959) reviewed earlier literature on pulmonary aspergillosis in bovines. Eggert and Rombery (1960) reported pulmonary aspergillosis in a calf.

Galati (1962-64) reported mycotic bronchopneumonia in 12 calves in Italy. Molello and Busey (1963) observed granulomatous pneumonia with septate hyphae and aspergillus type of conidiospores in a cow. Schotz and Meyer (1965) isolated Mortrulla sp. in Germany.

Narayana et al. (1964) reported a case of pulmonary aspergillosis in a Murrah buffalo. They observed numerous scattered nodules, 5 mm. to 5 cm. composed of semisolid caseous material. The parenchyma of the lung around these nodules was fibrosed. Histologically central area of necrosis encased by fibrous connective tissue was present. Fungal hyphae were visible in sections stained with toluidene blue.

Cordes et al. (1964) described fatal fulminating mycotic pneumonia in 13 dairy cows of which 8 cases followed by mycotic placentitis. The authors have described the lesions of extensive fibrosis, bronchopneumonia, large areas of necrosis, pronounced neutrophilic infiltration, large number of macrophages. Prominent and widespread thrombosis and necrosis of blood vessels, abundant hyphae at many areas of lungs, within and around blood vessels and alveolar walls were noticed. Culture from some cases indicated
Mucor sp. King et al. (1965) worked on mycotic pneumonia and abortion in cattle in Tasmania. The pathological picture as described by them consisted of presence of small (upto 5 mm.) granulomata containing "Asteroid bodies". Identical bodies were also noted by Austwick (1962) in bovine pulmonary aspergillosis. These were most numerous in older lesions in which granulomata were composed mainly of epithelial cells and a few neutrophils surrounded by fibrous capsule. The other main accompaniments were bronchopneumonia and organising fibrous alveolitis. Earlier cases had extensive alveolar epithelialization and interstitial oedema but no significant granulomatous reaction and central asteroid bodies showing varying degrees of phagocytosis by giant cells was present within the granulomata. They remarked the marked contrast in lesions described by Cordes et al. (1964) in New Zealand cases with their observation to be due to an acute infection of an unsensitized organ in cases reported by Cordes et al. (1964) whereas the pathology they had studied was more typical of an immune response to an agent with which the organ had previous contact.

According to Smith and Jones (1966), in mammals aspergillosis occurs in two forms i.e. diffuse pneumatic form and nodular pulmonary form. Short mycelia appear to be almost spherical, but true spores were not found. On surfaces exposed to air such as those of air sacs and lining of external orifices of trachea, the organism may produce long aerial mycelia which project into the lumen. The mycelial growth resembles the growth on artificial media.

Cordes and Shortridge (1968) described systemic phycomycosis and aspergillosis in 116 cattle. Out of 116 cattle, 9 neonatal calves had disseminated phycomycosis, mycotic placentitis
and/or acute mycotic pneumonia in 76 cows and 21 cattle had acute, subacute and chronic lesions in a variety of organs including brain, lungs, trachea, bronchi, spleen, kidney and heart etc. Chronic pneumonia was noticed in 5 cows. Out of these five, one had retained foetal membranes. At postmortem there were white masses (about 1 cm. in diameter) in lung, spleen and kidney. A case of metastatic carcinoma with a purely demarcated area in the lungs (about 8 cm. in diameter) was present. Three cases of heifer with fever, retained placental membrane and dispnoea which failed to respond to oxytetracycline therapy. One case with mycotic hepatitis and another case of mycotic encephalitis were also found. Histological lesions comprised areas of mixed caseation and coagulation necrosis and large part of the lobule of lung was involved. Necrosis and large number of caseated foci involving small alveoli with abundant hyphae and no evidence of in capsulation were seen. Inflammatory cells and hyphae were found in peribronchiolar alveoli.

Dwivedi (1968) studied 48 buffalo lungs pieces of which he reported 7 cases of mixed granulomatous pneumonia associated with mycotic elements resembling aspergillus or Mucor sp.

Buxter (1968) emphasised the growing veterinary interest in veterinary mycology in New Zealand and supported his views by giving in tabulated form the progressively increasing number of articles appearing in New Zealand Veterinary journal and also in the journal of International society of Human and Animal Mycology "Sabouraudia". In "Sabouraudia" (1963-1966), there appeared 107 publications on general article about fungal agents, 31 articles of human fungal disease, 51 articles were published on fungal diseases of animals.
Harcourt and Thompson (1969) reported mycotic pneumonia and mycotic abortion in a cow. The cow had temperature, vaginal discharge and retained placenta. The antibiotic treatment was futile. Postmortem revealed metritis and consolidation of the anterior half of both lungs with many small whitish yellow granulomatous lesions. A fungus was isolated from the lungs and from stomach contents of the foetus. The fungus was believed to be Mortierella sp.

Collins and Edwards (1970) demonstrated blastomyces dermitidis in mouse lungs. They described dense clusters or packets of budding yeasts as well as filamentous forms. Rarely multiple budding yeasts were also seen. Cells displaying dense bodies in cytoplasm resembling nuclei, clusters of rod and filamentous form of fungi were surrounded by tissue cells generally disrupted or degenerating and in many parts by polymorphonuclear leucocytes.

Rathore and Singh (1970) reported isolation of aspergillus sp. in buffalo lungs showing changes of proliferative pneumonia.

Damodaran (1971) reported in the lung of a Kangayam breeding bull of 7 years, grossly gray white discrete abscesses 0.1 cm to 1 cm diameter, and described histological lesion characteristic of pulmonary mycosis and noted the presence of mycotic elements.
PARASITIC DISEASE OF LUNG

Many helminth parasites pass through the lung in their larval phases. During this transient migrating phase the helminth produce some damage which is generally of little significance. There are, however, exceptions, and quite severe, and even fatal, pulmonary lesions may develop if the migrating parasites happen to be large in number or in size, or the host has some previous experience of exposure to the parasite, thereby developing a state of hypersensitivity. Some aberrant form of parasites growing in the lung cause considerable damage to the lung parenchyma. Morrow (1968) reported acute interstitial pneumonia in cattle due to Ascaris larvae.

Verminous pneumonia:

Strongylous species (lung worms) or other parasites have lungs as their final habitat. In bovines, Dictyocaulus viviparus infection is a serious parasitic disease.

Garger (1910, 1915) reported D. viviparus in different provinces of India. Mohan (1948) investigated D. viviparus as primary cause in an outbreak of pneumonia in cattle with typical lesions of verminous pneumonia.

Ramanujachari and Alwar (1954), and Awadhiya and Mehta (1960) reported that incidence of lung worm in bovine.

Michell and Shand (1955) studied 17 outbreaks of husk in cattle. Outbreaks were mostly endemic in the farm and also generally in subclinical form. They noted that gradual exposure to infection was less dangerous as compared to sudden exposure. Longer the
animals remained unexposed to infection, the more susceptible they became. The source of infection suggested was small number of larvae disseminated in the faeces of carrier animals and not those which were persistent on the ground throughout the winter. Larvae do not persist on ground even as long as five months. The larvae disseminated by these carrier animals turn into dangerous level of infestation by two ways viz., by intervention of calves who become lightly infected but pass sufficient larvae in faeces to raise the level of infestation of pasteure and directly when the conditions are particularly favourable for the larvae. Lush pasteure was noted to be a favourable vehicle for survival and transmission of infection.

Patnaik (1960) noticed verminous pneumonia fairly common among grown up cattle and prevalent in imported cattle. He reported bronchopneumonia in cattle and buffaloes due to D. viviparus.

Jarrett et al. (1960) described the lung worm disease of cattle. The progress of the disease was divided into prepotent, patent and postpatent phages according to the stages of parasitic life cycle. The lesions described mainly were small bronchi completely blocked with eosinophilic exudate, foreign body reaction to eggs or first stage larvae, alveolar epithelialisation, bronchial epithelial proliferation in response to dead larvae and bronchiolar damage and lymphoreticular reaction caused by dying larvae. In some calves lungs showed wide spread emphysema in diaphragmatic lobe. The complications caused by them were pulmonary oedema, pulmonary emphysema and secondary bacterial infection in course of disease. Bronchiectasis and pulmonary fibrosis were noticed, as sequalae. They also discussed the symptomology of parasitic bronchitis (husk)
and cuffing pneumonia which were identical.

Malaki (1961) reported lung worm infection in cattle in a farm of Bangalore. He described epizootiology of the disease. Warmth, moisture and plentiful supply of oxygen as prerequisites for incidence of lung worm disease.

Djafar et al. (1960) and Cornwell (1962b) found peripheral eosinophilia associated with *D. viviparum* infection.

Jubb and Kennedy (1963) described the changes produced in lung due to experimental infection with *D. viviparum*. The syndromes were described to be due to either massive invasion of the lungs by larval nematodes, or due to an indirect effect of the nature of a hypersensitivity reaction similar to the "self cure" phenomenon with release of histamin that develops with some forms of intestinal helminthiasis.

Poynter and Selway (1966) reviewed the work on diseases caused by lung worms.

Kuppuswamy and Singh (1969) reported for the first time lung worm disease in cattle in Bihar. Pathologic changes were described into affected lungs.

**Hydatidosis in lungs:**

Hippocrates, Aretaeus and Galen were clinically familiar with hydatid cysts, Redi, 1684; Hartmann, 1685; and Tyson, 1691 - (cited by Craig and Faust, 1943) suspected the animal nature of the cyst. Pallas (1766) mentioned similarities of hydatids in man and animals. Hartmann (1695) and later Rudolphi (1808) first studied the adult worms in dogs intestine.
Ganguly (1922) stated that 80% bovines in northern India harbour hydatids of taenia. He found hydatids in liver, lungs, spleen, kidney etc. Bhalerao (1935) listed the hosts of hydatids and observed that these cysts were mostly located in the lungs, liver, spleen and peritoneum. Sami (1936) pointed out that 88% of cow-buffaloes were infected with hydatidosis. Maqsood (1946) examined 340 buffaloes at Lahore and noticed that hydatid cysts were present in 234 animals (68.8%). The high incidence was attributed to a large number of stray dogs.

Craig and Faust (1949) observed that the cysts were surrounded by endothelial cells, giant cells and eosinophils and by well developed granulation tissue.

Thapar (1956) reported hydatids from lungs and liver of buffaloes in U.P., Bihar and Bengal. According to Smith and Jones (1963), the cysts grow slowly in man and were often encapsulated by thick connective tissue depending upon the organ parasitized.

Gill and Rao (1967) studied the incidence of fertility rate of hydatid cysts in Indian buffaloes. Out of 605 buffalo-carcases, 198 (33%) showed hydatid cysts. 88 percent of the cysts were fertile. Out of 376 cysts, 75 percent were found in lungs.

HAEMATOLOGY

It will be in fitness of thing to mention that there is no published information available on the cases of acute immature amphistomiasis accompanied with pneumonic lesions.
CHAPTER III

MATERIALS AND METHODS
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The materials concerned in this study consisted of pieces of lungs from cattle and buffaloes of all age groups and sex showing pneumatic or other changes. Pertinent data of autopsies available at Bihar Veterinary College, Patna and figures of mortality in calves and adults of Tharparkar breed of cattle belonging to the Government Cattle Farm, Patna, were incorporated in the present study. Blood samples were collected from healthy and sick cattle of the Government Cattle Farm, Patna with respiratory distress or dyspnoea etc. for haematological studies.

Altogether 300 bovine lungs showing consolidation and other observable abnormalities were examined. Out of 300 lung specimens collected, 85 lung specimens belonged to cattle slaughtered at various slaughter houses of Patna. These pieces of lungs were collected during the month of July, August and September, 1971. 106 buffalo lung specimens were obtained from buffaloes slaughtered at various places in the Bagmati zone of Nepal in October, 1971. Tissues were preserved in 10% formalin and brought in tightly covered specimen jars. 109 pieces of lungs, 15 pieces of brains, 10 pieces of lymph nodes and 4 pieces of heart muscles were obtained at autopsy of bovine cadavers. One specimen of nasal polyp from a cattle and another of congenital cyst from the bovine lung were obtained from the Department of Pathology, Bihar Veterinary College, Patna.

In all the above cases, a visual examination was done for various pathological abnormalities at autopsy, especially of bovine
lungs with pneumatic or other changes. The apical, cardiac, intermediate and diaphragmatic lobes were examined for change in colour and consistency. The lungs were placed with the dorsal side upwards and the caudal portion facing the examiner to detect the gross lesions. Lungs were palpated and examined to detect the pneumatic changes, abscesses, cysts, nodules, and other abnormalities.

Impression smears were made from the portion showing typical abscess or lesions suggestive of pulmonary tuberculosis. Smears were heat fixed and stained appropriately by PAS, Gram's and/or Ziehl-Neelsen's stain to study the morphological features of the microorganisms, fungi etc.

Tissue pieces or materials from the lesions were collected in a few cases in sterilized petri's dishes and swabs were prepared for bacteriological study and transmission tests.

Portions of lungs of suitable size (about 1 cm. cube) representative of diseased conditions were collected in 10 percent formalin for histopathological study. As far as possible only fresh lung tissues were collected.

Lungs (all lobes or portions of them) showing gross changes and presence of abscesses, cysts etc. were collected. Size, shape, colour, consistency, appearance of cut surfaces and relation to surroundings of the lesions were taken into consideration for preparing the statement of their gross anatomical changes.

Isolation procedure: - To illustrate the bacterial flora associated with the pathological condition, attempts were made to isolate the causative bacteria. The materials from the representative lesions were first studied in smears stained appropriately.
Thereafter, in interesting cases, plated in suitable media and colony characters were studied after incubating for a desired period. Microscopical studies of the suitably stained preparation of the culture were made. In a few, the virulence and pathogenicity were tested to confirm the aetiological significance.

In one case, isolates simulating *Mycobacterium tuberculosis* were inoculated intraperitoneally after proper dilution in guineapig, which was later destroyed and pulmonary lesions developed within 21 days, were studied with the recovery of similar bacteria.

The following media were used in this study:

(i) Nutrient agar - incubated at $37^\circ$C for 24 hours to 36 hours.

(ii) Blood agar - -do-- -do--

(iii) Dorset's egg medium - incubated for 4 weeks at $37^\circ$C.

(iv) Sabouraud's media - incubated at $37^\circ$C.

Transmission experiment for viral agents:-- To rule out the possibility of viral involvement in the bovine respiratory conditions, the following procedures were adopted.

24 healthy white mice were obtained from animal genetics section of the Bihar Veterinary College, Patna. These mice were divided into three groups on the basis of random assortment. Each group comprised of 8 mice.

Group I:-- Lung specimens of calves and adult cattle died with pneumatic lesions in the lung were collected separately and were kept in the freezing chamber of the refrigerator. Some pieces of lungs were pooled in the mortar and macerated to five consistency with the pestle. Normal saline with 100 I.U. of Penicillin, and 100 microgram of Streptomycin/ml. were used in proportion
of 1:25 for dilution. The whole was filtered employing Seitz filter connected to a vacuum pump.

The filtrate was ready. Before inoculating it into mice, the filtrate was plated in a plate of blood agar to test for the bacterial contaminants. With a sterile Pasteur pipette, the filtrate containing antibiotics were inoculated intranasally (two drops in each nostril) in three mice and then they were kept in one cage. Another group of three mice was administered intranasally two drops of cell free filtrate in each nostril and was kept in a second cage. The remaining two mice were left in the third cage. All mice were kept under observation for three days. Temperature were recorded before inoculation and every day during the observation period. They were destroyed after three days and the lungs were examined for any change. The lungs of mice were preserved for histopathological study in 10% formalin.

**Group II** :- Mice in this group were treated in the similar manner as in group I. Phosphate buffer saline (PBS) pH 7.2 was used as diluent instead of saline. Mice were killed after three days and lungs were preserved for study.

**Group III** :- Mice lungs with gross pneumonic changes from the infected mice of group II were used instead of bovine lung in the third series of test. Dilution of the filtrate used was 1:50 PBS with antibiotics in the same strength. The pneumonic lungs were preserved for histopathological study.

**Statistical study** :- Data obtained from P.M. records and mortality figures were statistically analysed. The incidence of pneumonia in relation to the different months of the year and its
incidence in different age groups were evaluated. In order to confirm the importance of the respiratory and other diseases under natural conditions, percentage of mortality due to different respiratory conditions in comparison with others were worked out.

Haematological studies of few cases of animals (Tharparkar cattle) belonging to the Government Cattle Farm, Patna, suffering from diarrhoea accompanied with clinical pneumonic syndrome were carried out. Blood samples were collected in vials containing Wintrobe’s anticoagulant for estimating the following blood values:

(a) Haemoglobin percentage (b) Differential leucocytic count.
(c) Total leucocytic count (d) Total erythrocytic count.

The blood values of the animals showing pneumonic symptoms were compared with that of normal animal.

Histopathological procedures:

The tissues were collected immediately after examination of the lung. After proper fixation in formaline, they were washed over night under tapwater, dehydrated in ascending grades of ethyl alcohol, cleaned in cedar wood oil and embedded in paraffin (58–60°C). Sections were cut at 5–7 microns thickness and were stained as routine by Haematoxylin and Eosin (Lillie, 1954).

The following staining procedures were also adopted to demonstrate special elements to confirm the lesions observed in routine H & E stained sections:

**Connective tissue**:

(a) Van Gieson's picrofuchsin method (Lillie, 1954):

(i) Deparaffinise the sections and bring them to water.
(ii) Wash in water

(iii) Stain for 5 minutes in haematoxylin (Weigert's acid iron chloride).

(iv) Wash in water

(v) Stain 5 minutes in picrofuchsian mixture

(vi) Dehydrate and differentiate with two changes each of 95% and 100% alcohol.

(vii) Clean with a mixture of 100% alcohol and xylene followed by two changes of xylene. Mount in DPX.

(viii) PAS positive factors - Periodic acid-schiff method (Culling, 1957):

(i) Bring sections to water

(ii) Oxidise for 5-10 minutes in 1% aqueous periodic acid.

(iii) Wash in running water for 5 minutes and rinse in distilled water.

(iv) Treat with schiff reagent 10-30 minutes

(v) Transfer directly to the first sulphite rinse one minute - second sulphite rinse for 2 minutes - transfer to third sulphite rinse for 2 minutes.

(vi) Wash for 10 minutes in water.

(vii) Counter stain with haemotoxylin

(viii) Dehydrate, clear and mount in DPX.

PAS positive substance bright red, Nuclei-blue, other tissue constituents yellow.

(c) Iron: Perl's Prussian blue reaction (for ferric salts) (Culling, 1957).

(i) Bring to distilled water

(ii) Transfer to fresh solution of equal parts of 2% aqueous potassium ferrocyanide and 2% HCl. for 30 mts.
(iii) Wash thoroughly in several changes of distilled water.
(iv) Counterstain with 1% eosin (alcoholic) stain.
(v) Dehydrate, clear and mount in DPX.
Ferric iron containing pigments (haemosiderin) blue, nuclei red.

(d) Gram Weigert technique for gram + bacteria (Culling, 1957).
(i) Bring section to water
(ii) Stain with 2.5% aqueous phloxine
(iii) Wash in water and stain with Gram's crystal violet for three minutes, blot with filter paper.
(iv) Treat with lugol's iodine for one minute, blot with filter paper.
(v) Decolourise in aniline oil and then treat with equal parts of aniline and xyline.
(vi) Clean in xyline and mount in DPX.
Gram + bacteria blue; other tissue constituents red.

(i) Bring sections to distilled water
(ii) Stain one hour in Giemsa's stain (Methyl alcohol 1.25 cc., 0.5% sodium carbonate solution 0.1 cc. (2 drops), Giemsa's stain 1 cc., distilled water 40 cc.).
(iii) Pour off and replace with two further changes of same mixture during the first hour and leave in the third change overnight.
(iv) Differentiate in 95% alcohol containing a few drops of 10% colophonium alcohol.
(v) Dehydrate with 100% alcohol, clear and mount in DPX.
Gram negative bacteria stains intense reddish purple.

**Acid fast bacteria:**

1. Bring the sections to water
2. Stain in hot carbol fuchsin for 10 minutes
3. Wash in water to remove excess stain
4. Differentiate in 3% acid alcohol
5. Wash in water
6. Counterstain lightly in 0.1% methylene blue for half a minute.
7. Wash in water
8. Dehydrate, clear and mount in DPX.

Acid fast bacilli - red, nuclei blue, other tissue constituents - pale blue.
CHAPTER IV

OBSERVATIONS
OBSERVATIONS

Incidence of Bovine Pulmonary Diseases in BVCH, GCF and other Materials:

The materials under the present study could be divided into three main parts viz. (1) Materials belonging to Tharparkar cattle of the Government Cattle Farm, Patna; (2) Materials collected from Bihar Veterinary College Hospital (BVCH) autopsy cases and (3) other category of materials including bovine lungs and other tissues from animals killed or dead in Patna and Bagmati zone of Nepal.

| TABLE - I |
| Composition of GCF materials |
| --- | --- | --- | --- | --- | --- |
| No. autopsied | Pneumonia | Other P. lesions | Tuberculosis | Digestive disorders (C., etc.) | Other conditions |
| 502 | 270 | 52 | 57 | 83 | 40 |

P. = Pulmonary; C. = Cotyphosphoriasis.

Table-I shows the distribution of the pertinent available data of the Government Cattle Farm, Patna, over a period of eight years along with various other pulmonary maladies observed postmortem. Pneumonic cases occupied the highest prevalence among the different diseases diagnosed in the autopsy cases.
TABLE - II
Incidence of Pneumonia in the different age groups of Tharparkar cattle

<table>
<thead>
<tr>
<th>Total no. of cases</th>
<th>No. autopsied</th>
<th>Age group of animals</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>362</td>
<td>102</td>
<td>0 to 6 months</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>72</td>
<td>6 months to 1 year</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>91</td>
<td>1 year to 3 years</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>53</td>
<td>3 years to 8 years</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>44</td>
<td>Above 8 years.</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
</tbody>
</table>

Incidence of pneumonia in Government Cattle Farm material among the different age groups of Tharparkar cattle is shown in table above. The incidence is highest among 0 to 6 months age group and 1 year to 3 years age group.

TABLE - III
Seasonal distribution of pneumonia in Tharparkar cattle of GCF.

<table>
<thead>
<tr>
<th>Year</th>
<th>No. autopsied</th>
<th>No. cases of Pn. &amp; others with lesions of Pn.</th>
<th>Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>1 2 3 4 5 6 7 8 9 10 11 12</td>
</tr>
<tr>
<td>1971</td>
<td>221</td>
<td>145</td>
<td>3 3 2 1 3 2 5 7 86 21 8 4</td>
</tr>
</tbody>
</table>

Pn. = Pneumonia.

The above table indicates seasonal distribution of incidence of pneumonia in GCF material. Its incidence is quite high during the late rains.
The crude death ratio (CDR) in Government Cattle Farm, Patna, cattle was found to be 0.25. With particular reference to pneumonia in Tharparkar cattle, cause specific death ratio (C-SDR), case fatality ratio (CFR) and age specific incidence ratio (A-SIR) in calves under six months were 7469.9, 28.57 and 0.205 respectively.

Incidence of pneumonic lesions in Bihar Veterinary College Hospital materials (Cadavers from BVCH and GCF, etc.).

**TABLE - IV**

Composition of BVCH materials

<table>
<thead>
<tr>
<th>No. autopsied</th>
<th>No. cases</th>
<th>No. cases of tuberculosis</th>
<th>No. cases of pneumonia</th>
<th>No. other cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>788</td>
<td>470</td>
<td>71</td>
<td>2</td>
<td>245</td>
</tr>
</tbody>
</table>

(Tuberculous pneumonia - 48)

Fn. = Pneumonic.

Table IV illustrates the frequency distribution of pulmonary lesions in Bihar Veterinary College Hospital materials.

**TABLE - V**

Seasonal distribution of pneumonic lesions in BVCH materials

<table>
<thead>
<tr>
<th>No. autopsied</th>
<th>No. cases of Fn &amp; others</th>
<th>Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>788</td>
<td>472</td>
<td>1 2 3 4 5 6 7 8 9 10 11 12</td>
</tr>
</tbody>
</table>

Fn. = Pneumonia; Pneumonic.

It is evident from the table above that the more incidence of pneumonic cases occurred during the late rains and
winter season of the year.

Fig. 1 indicates rather regular seasonal rise in incidence of pulmonary maladies in animals of both descript and non-descript types with its peak reaching during the month of September.

Incidence of pulmonary maladies in materials belonging to Bagmati zone of Nepal, are given below in table-VI, & VII.

**TABLE - VI**

The frequency of Pneumonia and other respiratory diseases in bovines during different seasons

<table>
<thead>
<tr>
<th>Years</th>
<th>Respiratory cases treated during the months of</th>
</tr>
</thead>
<tbody>
<tr>
<td>1967</td>
<td>8</td>
</tr>
<tr>
<td>1968</td>
<td>16</td>
</tr>
<tr>
<td>1969</td>
<td>21</td>
</tr>
<tr>
<td>1970</td>
<td>30</td>
</tr>
<tr>
<td>1971</td>
<td>39</td>
</tr>
<tr>
<td></td>
<td>Total</td>
</tr>
</tbody>
</table>

The data available from Central Veterinary Hospital, Tripureswar, Kathmandu, indicates the incidence of respiratory disorders which is quite low in July, but shows a sharp rise during August, September and October. There is a gradual decline during rest of the months in year.

Fig. 2 indicates sharp rise in incidence of respiratory diseases in bovines during the months of August, September and Oct. thereafter shows a gradual fall in rest of the months in year.
Frequency of different disease conditions in bovine lungs obtained from different sources for the present investigations, are presented in Table - VII below.

**Table - VII**

Composition of 300 specimens collected from bovine lungs

<table>
<thead>
<tr>
<th>Pathological diagnosis</th>
<th>BVGH autopsies (109)</th>
<th>S.H. material Patna (85)</th>
<th>Nepali buffalo materials (106)</th>
<th>Total</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydatid cyst</td>
<td>1</td>
<td>7</td>
<td>14</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td>Lung worm disease</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Interstitial pneumonia</td>
<td>8</td>
<td>20</td>
<td>26</td>
<td>9</td>
<td>28</td>
</tr>
<tr>
<td>Broncho-pneumonia</td>
<td>5</td>
<td>4</td>
<td>5</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Fibrinous pneumonia</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Mycotic pn.</td>
<td>2</td>
<td>8</td>
<td>5</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>Hyperaemia</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Oedema</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Oedema and hyperaemia</td>
<td>2</td>
<td>7</td>
<td>7</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Emphysema</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Pulmonary inf.</td>
<td>-</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Abscess</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>-</td>
<td>2</td>
<td>2</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>3</td>
<td>7</td>
<td>4</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Chronic cong.</td>
<td>-</td>
<td>4</td>
<td>2</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Virus Pn. like</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Adenomatosis</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Pn. = Pneumonia; Inf. = infarct; cong. = congestion.
The above table shows well marked incidence of interstitial pneumonia, hydatid cysts and mycotic pneumonia in cattle and buffaloes.

Incidence of pulmonary mycosis (PM) in Nepali buffaloes are shown in table 8 below.

**TABLE - VIII**

Depicts incidence of pulmonary mycosis in Nepali buffaloes.

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of slaughtered cases.</th>
<th>No. of P.M. cases</th>
<th>No. of P.M. cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Under 3 years</td>
<td>Over 3 years</td>
<td>Total</td>
</tr>
<tr>
<td>1971</td>
<td>600</td>
<td>2</td>
<td>14</td>
</tr>
</tbody>
</table>

It is evident from the above table that so called apparently healthy animals slaughtered for human consumption were found to be affected with pulmonary mycosis with its higher incidence in buffaloes over 3 years of age.

The geographical distribution of pulmonary mycosis in buffaloes in Bagmati zone of Nepal is presented in fig. 1. Figure indicates that the pulmonary mycosis cases were recovered from different places in Bagmati zone of Nepal under the present study.

Pathologic changes in respiratory diseases of bovines:

**Nasal polyp**: The material was received from Bihar Veterinary College Hospital, Patna. The nasal polyp was recovered from a seven year old cow with complaints of respiratory distress and snoring sound.
Macroscopic appearance: It was 10" long greyish white pendulated and somewhat cylindrical growth. It had 5" long hollow cavity filled with clear slimy fluid (fig.2). It was soft in consistency and tapering proximally. About 1/3rd of its distal portion was a solid structure.

Microscopic appearance: Histological sections manifested that it was lined with a layer of mucous membrane consisting of hyperplastic epithelial cells at places. The inner growth of the tissue showed loose mesenchymal tissue consisting of sparsely distributed stellate cells, collagenous fibrils, a few thin walled capillaries and some infiltrating round cells (fig.3).

Congenital cyst of lung:

The congenital cyst of lung was obtained from the department of Pathology, Bihar Veterinary College, Patna. It was recovered from an immature calf at autopsy.

Macroscopic appearance: Inter lobular septa of right lung were severely infiltrated with watery fluid which escaped copiously from the cut surfaces of the lungs. There were several pin point holes in the right diaphragmatic lobe. The left lung was hypoplastic and smaller than the normal.

Microscopic appearance: Several cystic cavities of different shapes and sizes were seen in the right diaphragmatic lobe. The cuboidal cells lining these cavities had proliferated at some places to form three or more layers of cells. The cystic wall consisted of lining cuboidal epithelium, surrounded by a
coat of fibrous tissue with no ingrowth of blood vessels. 
Vari-sized air spaces like alveoli or bronchioles were visible. 
The interlobular septa were distended and had fibrous elements 
well separated from each other by empty spaces. A few cyst 
like cavities were also present in the left lung.

**Exudative Pneumonia:**

**Bronchopneumonia:** Of the three hundred lung specimens studied under the present investigation (6.66%) showed pathologic changes of bronchopneumonia.

**Macroscopic appearance:** The lungs of the animals were reddened and consolidated mostly in the anteroventral portions (apical, cardiac and intermediate and diaphragmatic lobes). Greyish white emphysematous raised areas quite adjacent to these reddish patches were present above the level of the lung parenchyma. In some cases these patches were also greyish or yellow in appearance. The lungs were mostly dry when cut, but slightly bulged out at the cut surfaces. In many of these, bronchial mucosa were also reddened and covered with slightly frothy exudate. T

**Microscopic appearance:** In some specimens, bronchi and bronchioles contained exudate consisting of red cells, mononeuclear cells and other indistinguishable cellular detritus. There were also well marked consolidated areas in the lung parenchyma (fig.5). In some cases the lining of the bronchial epithelium had desquamated to lie into the bronchi and pink stained exudate a few inflammatory cells, cellular
cellular debris etc. (fig. 6) were present in the bronchi, the picture was suggestive of necrotizing bronchopneumonia. In few of the cases, there was slight increase of round cells in the peribronchial connective tissue. In some alveolar spaces were filled with oedematous fluid with or without the presence of degenerating and intact polymorphs or mononeculears or both (fig. 7). In two of the cases pictures were suggestive of peribronchial spread of the exudative lesions (fig. 8). In some of the cases, the focal area of suppuration with degenerating polymorphs were found. In a Frisien calf which was suffering from pneumonia and died, numerous small abscesses scattered throughout the lung parenchyma, were observed. Histopathological lesions were similar to that of bronchopneumonia with suppuration.

Sections of the above cases stained with Gram Weigert and Wolbach's Giemsa variant technique showed Gram positive and gram negative organisms in several cases.

In the smears made from lungs of twelve cases, gram positive pleomorphic rods were recovered in four cases. When plated on agar plates, they produced dew drop like minute colonies which became opaque on aging the culture. The morphology of the organism was suggestive of Coryne-bacterium species. In three case, the smears revealed gram positive spherical organisms arranged in clumps like bunches of grape or in clusters. On agar plates, the colonies were round, smooth glistening, opaque, low convex and amorphous. Their edges were entire golden in colour and butyrous in consistency. In two cases, chains of gram positive spherical organisms were recovered. When plated on agar they produced small
smooth, finely granular and glistening dew drop like colonies which on storing became opaque and with a raised central portions were observed. On blood agar, they produced beta type of haemolysis after incubating for 24 hours. In one case smears showed gram negative slender pleomorphic organism which varied from coccoid to long filamentous forms not dissimilar from Fusiformis sp. These organisms could not be isolated on the agar plate. In the last two cases no organisms could be isolated.

**Fibrinous pneumonia:** Of the three hundred specimen of lungs examined, fibrinous pneumonia was noticed in four cases (1.3%).

**Macroscopic appearance:** Red or dark brown areas of consolidation were seen in the lower halves of all the lobes of the lungs. The lungs showed characteristic marbled appearance due to the distension of pleura and interlobular septa with fluid and fibrinous strands (fig.9). There were also consolidated patches, emphysematous areas surrounded these patches. The exudate rich in fibrin were seen to have emphasized the inter lobular septa. Sero-fibrinous exudate was present in the thoracic cavity. Petichae or tiny haemorrhagic spots were noticed on the pleura. Severe pleuritis with strands of fibrin were seen in three cases. The consolidated lungs sunk into water.

**Microscopic appearance:** The lesions were mainly seen on pleura and in interlobular septa and alveolar walls. The pleura and interlobular septa were distended with serofibrinous exudate. Alveoli contained fibrinous threads or balls (fig.10). Mononuclear
cells were predominantly found infiltrating the interlobular septa. The alveolar walls were congested and the alveoli containing fibrinous exudate alongwith few cells were observed. The cells present were mostly polymorphs, macrophages etc. Thickened alveolar septa were also predominantly noticed.

Bipolar organisms resembling Pasteurella sp. were found in impression smears made from the diseased lung. These organisms were isolated on agar plates. The colonies were round, flat, fluorescent, whitish opaque, smooth and moist. These organisms were also isolated from cases of pasteurelosis that had broken out in epizootic form in Government Cattle Farm, Patna. Fresh culture of the isolate given subcutaneously killed the white mice with-in 24 hours. The above observation was indicative of pathogenic nature of these organisms.

Proliferative Pneumonia:-

Interstitial Pneumonia:- Of the three hundred specimens of lungs, 91 cases (30.3%) revealed features of interstitial pneumonia.

Macroscopic appearance:- The lungs were greyish white in colour, consolidated and firm in consistency. The lesions with increased fibrous tissue were mostly distributed on the ventral borders of the diaphragmatic lobes. Generally there were, in both lungs, emphysematous areas closed to the consolidated patches. The lungs were enlarged and did not collapse when thorax was opened. Sometimes lungs appeared oedematous and the cut surfaces looked greyish white. Slightly, blood tinged fluid escaped from the cut surfaces of the lungs. Inter lobular septa were widened and prominent due to the thickening. Only in few cases of interstitial
pneumonia, the other pulmonary conditions such as bronchopneumonia etc. were seen associated with it.

**Microscopic appearance:** Interalveolar septa and interlobular septa were distinctly thickened due to serous or serofibrinous exudate. Infiltration of inflammatory cells (mostly round cells) and proliferation 'Metaplasia' of the alveolar lining epithelium which assumed somewhat cuboidal shape, was seen in a few cases. Giant cells could be seen in only two cases. Round cell infiltration in the peribronchial spaces was noted in the sections of only few cases. In many cases, the parenchyma of the lungs at places was seen gradually replaced by newly formed fibrous tissue. In five cases, muscular hypertrophy of the arterioles with some thickening of the intima was seen. Empty alveolar spaces with thickened interalveolar septa were most commonly observed(fig.11).

No bacteria could be recovered on stained sections as well as in the impression smears made from these specimens.

**Pulmonary Tuberculosis:**

In 14, of the 300 lung specimens, (4.6%) pulmonary tuberculosis of varying intensities were observed.

**Macroscopic appearance:** A few to numerous multifocal lesions of different sizes and shapes with caseous areas at the centre were noticed. In some cases calcified nodules encased by capsule of fibrous tissue were present. In most of the lungs, pronounced lesions were seen in caudodorsal aspects of diaphragmatic lobes. There were areas of congestion and emphysema â€œclose
proximity of these lesions. In two cases only, distinct tubercle nodules bulging over the surface of the lungs giving an uneven nodular appearance to the organ, were seen. The nodules were compact and solid, when cut they were found to have greaty consistency owing to calcified materials in them.

**Microscopic appearance:** Replacement of the normal architecture of the lung parenchyma by caseous and calcified materials were observed (fig.12). The nodules from within outwards consisted areas of calcification surrounded by a wide zone of caseation. In the necrotic mass, some granular cellular debris which stained dark blue or black representing the nuclei of dead cells were seen.

This mass was found surrounded by epitheloid and mononuclear cells interspread with Langhan's type of giant cells (fig.13). Proliferated fibrous tissue capsule had been laid down around such lesions in many cases. At places, these lesions were seen coalescing with neighbouring lesions; thus formed larger lesions occupying greater portion of the lung parenchyma. The lining of bronchioles in contact with these lesions were disquamated and moderate lymphocytic infiltration in laminapropria were noticed.

Sections stained with Ziehl-Neelsen's method revealed several acid fast Basili resembling *Mycobacterium tuberculosis*. Foci containing epitheloid cells and some plasma cells were seen at places in the parenchyma of the lungs (fig.14). Smears from these lesions stained appropriately with acid fast method showed acid fast Basili not dissimilar from *Mycobacterium tuberculosis*. 
The organisms were isolated in pure culture on Dorsets media. Colonies were dry, rough and with crumbly growth which persisted even after a long incubation.

Guineapigs which were inoculated with the above materials developed a chronic debilitating disease. After observation period of six weeks, the guineapigs were killed and autopsied. Lesions were noticed all along the visceral organs including the lungs of guineapigs (fig.15). Acid fast Basili were present in stained smears from the affected organs.

**Pulmonary Mycosis (PM):**

Of the three hundred cattle and buffalo lung specimens, 32 (10.6%) showed pneumatic changes associated with mycotic elements.

**Macroscopic appearance:** In the lungs, there were numerous small (about .5 to 1 mm. diameter) grey areas scattered usually subpleurally in the lungs. Some of the lungs were pale or slightly oedematous. Others were consolidated and firm in consistency.

**Microscopic appearance:** Coagulative necrosis and caseation with infiltration of neutrophils and large number of macrophages around the cellular debris and necrotic tissue were present (fig.16). Thrombosis in the pulmonary blood vessels were present in many cases (fig.17). Varying degrees of degenerative and proliferative changes in the wall of the blood vessels profuse arthritis and thickening of the various layers of blood vessels and sometimes even completely occluded arterial lumen were observed(fig.18).
Some bronchi and bronchioles were seen containing the disquamatéd epithelial cells and mass of cellular debris. Branched hyphae of the fungi which were PAS positive were seen in the alveoli and also traversing through the bronchi (fig.19). Hyphae were also noticed passing through the walls of blood vessels. Hyphae were found even within some of the blood vessels (fig.20). PAS positive hyphae or mycelial threads were also present in the caseous areas. At places the epithelial cells in the vicinity of caseated mass or nodules assumed swollen cuboidal appearance. Alveolar tissue were found approximated to each other at few places. Several oval or round PAS positive bodies (spores) in the bronchi and alveoli were discernable. At places the caseous nodules showed epitheloid cell reaction and a tendency of incapsulation. Bronchi and alveoli at places contain tangled mass of mycelia alongwith alveolar thickening (fig.21). Changes of interstitial pneumonia with lesions of mycotic pneumonia were noticed in many histological preparations. Out of the specimens examined, only in four cases of Nepali buffaloes and two cases of cattle granulomatous reactions were noticed. In many of the specimens bronchial mucosa at places were markedly thickened and proliferated to form papillary infoldings projecting deep into the bronchial spaces (fig.22), and mycelial threads and thickened alveolar septa were present in several lung specimens of both cattle and buffaloes. There were several nodules consisting mostly of round cells and mononuclear cells in the lungs of buffalo and cattle (fig.23). Very conspicuous intimal and medial hyperplasia alongwith some round cell infiltration led to considerable thickening of the arteries to the extent of almost obliteration of the lumen were present in the lungs affected with
mycotic pneumonia (fig.24). PAS positive oval and round spores were present in a large numbers in the lumen of a thickened bronchiule (fig.25).

From 12 buffalo lungs showing small greyish white spots plating was done on the sabourauds dextrose agar media with antibiotics. On incubation colonies of fungi developed. These colonies were of two morphological types - one has round white velvety growth with steel grey to black elevated central area (fig.26). The other was white velvety and its centre was elevated markedly (27). The central grey portion when picked up on the glass slide and examined under microscope revealed several conidia (spores). These were almost round seen in clusters and thickly dispersed in the field in the mounted preparation under microscope (fig.28). The white portion of the growth were the tufts of mycelial threads. A portion of the colony mounted on the glass slide revealed interlacing hyphae several conidia spores and numerous conidia (fig.29).

Pulmonary infection resembling calf pneumonia:

Calf pneumonia like conditions has been found causing heavy mortality in the Tharparker calves of Government Cattle Farm, Patna since long. The calf suffered from loss of appetite, high fever, rapid breathing and unthreptiness in this disease. There was anamnesis of quick death after a course of only a few days. Diarrhoea was also constantly reported among the calves, dying of this type of pneumonia.

Out of 300 specimens of bovine lungs, lugs from two
calves with pneumonia belonging to Government Cattle Farm, were studied under this group.

**Macroscopic appearance:** The anterior and ventral borders of both the lungs revealed reddish patches mottled with grey. The cut surfaces of the lung were moist and serous fluid escaped from the incised parts. There were greyish white raised areas above the level of the healthy lung parenchyma close to the firm and consolidated area.

**Microscopic appearance:** There was pink staining exudate in the alveoli with few desquamated epithelial cells etc. There were a slight increase in the number of round cells in the bronchiolar mucus membrane but there was a moderate number of round cells around the bronchi and blood vessels (fig. 30).

**Transmission experiments:** With a view to ascertain the presence of virus like elements if any in the suspected cases and whether pneumonia could be established in white mice with cell free filtrate of the lungs given intra-nasally, this experiment was carried out. In the intra-nasally infected first lot of white mice no clinical symptoms were observed. The mice were killed three days post infection. The lungs revealed some areas of focal congestion and consolidation. Microscopically there were pink stained exudate containing few round cells and cellular debris in some bronchioles and alveoli. The lungs of the mice of the second lot showed areas of congestion and consolidated zones, involving greater area of the lung parenchyma. Microscopically the lesions were a bit more intense than in the first lot.
They had some clinical symptoms also. The mice of the third lot developed more severe pneumonic symptoms with rise of temperature. The lesions in the lungs were more intense, in this group, almost entire lung was found consolidated. The alveoli and bronchi contained oedematous fluid, round cells and cellular debris (fig.31).

**Verminous Pneumonia:**

Of the 300 specimens of the lung, 5 (1.6%) showed the presence of lung worms in the bronchi and the alveolar spaces, showed pathological changes, indicative of verminous pneumonia.

**Macrosopic appearance:** Lungs were voluminous, consolidated, firm and pale. At places, atelectatic and emphysematous areas were noticed. The lungs did not collapse when the thorax was opened. They were tough and cut with more difficulty. Frothy exudate was squeezed out of the incised part. The trachea and large bronchioles also contained frothy exudate. In three cases, thread like worms were recovered from the bronchioles.

**Microscopic appearance:** Alveolar septa were thicken due to round cell infiltration and proliferation of the connective tissue elements in their walls. Some alveoli contained pink exudate. Some bronchiles contained indistinguishible cellular debris. Some multinucleated giant cells were visible in the alveoli of only one case. In three cases the alveolar epithelium tended to be cuboidal. Hyperplastic changes of bronchial muoosa giving rise to papillary growth which looked like as folds
of mucous membrane, were noticed in two cases. In some bronchi and alveoli fragments of the larvae of the lung worms were noticed, (fig.32). At places the embryonated eggs of the lung worms were detected. Round cells and neutrophils could be seen in some of the alveoli. In one case granuloma consisting of epitheloid cells was observed (fig.33).

**Hydatidosis in the lungs:**

Of the 300 specimens of lungs, 42 cases (14%) were found associated with varying sizes of hydatid cysts. These cysts were mainly of two types, viz. (i) cysts with intact cyst wall and (ii) those in which cyst wall was collapsed and chronic changes were evident.

**Cysts with intact cyst wall:**

**Macroscopic appearance:** Majority of the cysts were of this type. In few cases, one or more cysts were situated superficially in the lungs giving characteristic cystic appearance to the organ, whereas in others cysts were found situated deep inside the parenchyma. Diaphragmatic lobes were mostly the site of preference for the cysts. On palpation these cysts were soft and had a tendency to collapse when pressed. Some of the cysts were large and had many spherical elevations (fig.34). When incised these cysts were found to contain watery fluid. In many cases the cavity was lined by smooth delicate membrane, which could be separated easily (germinal layer).

**Microscopic appearance:** The germinal layer could not be detected in the stained sections although they were present
in the gross specimens. The cystic spaces contained an eosinophilic mass. The layers that could be seen were a well developed compact and concentrically laminated parasitic wall (external cuticular membrane) which was found disrupted and degenerated, at places with infiltration of round cells mostly in the cyst wall (fig. 35). A zone of epitheloid cells was also noticed around the necotic area. A dense connective tissue capsule was well recognisable in the section (fig. 35). The lung parenchyma external to the capsule was highly compressed almost atelectatic as a result of pressure exerted by the growing cyst. The alveolar spaces in the vicinity were filled with mononuclear cells, lymphocytes and other leucocytes. They were also seen infiltrating in the capsule.

Cyst with collapsed wall:

Macroscopic appearance: Collapsed hydatid cysts were yellowish in appearance, solid to touch. The surface manifested laminated appearance with small quantity of viscus fluid. The cysts were seen in the state of collapse.

Microscopic appearance: Dominant changes in the cyst mass were retrogressive in nature.

Alveolar Emphysema:

Out of 300 specimens, 10 cases (3.3%) showed emphysematous changes dominantly over the parenchyma.

Macroscopic appearance: In seven specimens, the
lesions were focal in nature, in the rest three the changes were of diffused character involving nearly the whole lungs. The focal changes were mostly noticed in the ventral borders of the diaphragmatic lobes of the lungs. In focal cases, changes were sharply defined as foci of emphysema involving the few lobules. The emphysematous area were well above the level of the normal parenchyma. Characteristic ballooning of the alveolar tissue could also be observed in gross lesions. These areas were paler than normal lung parenchyma which were also puffy in consistency.

In diffuse cases lung as a whole were excessively enlarged almost completely filling the thoracic cavity. The lungs were pale in appearance crepitating and spongy, on palpation, difficult to cut and cut surfaces were smooth and dry. Ballooned appearance of the lung were also due to bullae in the interlobular septa with air, without tendency to collapse were the features of this condition. In some impression of the ribs were visible.

Microscopic appearance:— Greatly enlarged alveoli and alveolar ducts were present, at several places in the lung parenchyma. Alveolar walls at places were ruptured forming giant alveoli (fig.36). The alveolar septa were thin and bloodless and torn. The torn ends had bulb like swellings. In few cases, hypertrophic changes in the musculature of the neighbouring bronchi were seen. In some, which may be considered acute emphysema spasmodically closed bronchioles were visible. The chronic cases manifested bronchiolitis with varying degree of obliteration and replacement with fibrous tissue.
Atelectasis:

Out of 300 specimens, 6 cases (2%) had distinct and considerable areas of atelectasis.

Macroscopic appearance:— The lungs were very much reduced in size. The affected part were clearly depressed below the level of surrounding parenchyma. It was firm inelastic, airless and of meaty consistency. The colour was steel grey or slat grey due to stasis of circulation. The cut pieces of the lungs sank into the water.

Microscopic appearance:— Alveolar walls were pressed together with narrowing to complete obliteration of the lumen (fig.37). The alveolar lining cells assumed more or less cuboidal appearance. The pleura were thick and wrinkled.

Oedema and Hyperaemia:

Out of 300 specimens, 19 cases (6.3%) showed characteristic combination of hyperaemia and oedema, 15 cases (5%) were predominantly hyperaemic and 13 cases (4.3%) were dominantly oedematous. For the sake of description these lesions are described under three separate headings — namely Hyperaemia and oedema, Hyperaemia, and Oedema.

Hyperaemia and Oedema:—

Macroscopic appearance:— The lungs were pale or slightly cyanotic, larger in volume, heavy and firm, and most often doughy. They showed marked pitting on pressure, When cut
and squeezed, fluid poured out from the cut surfaces and from the bronchi. In some cases, bronchus contained frothy fluid. The colour of the fluid depended on the presence or absence of congestion. Some fluid was occasionally present in the pleural cavity. Interlobular septa were widely distended by gelatinous fluid. Lesions accompanied by congestion, were uniformly deep red to reddish black in colour. Casts of clotted blood were visible in the bronchi. They were resilient on palpation.

Microscopic appearance:- In some cases, there was interstitial oedema. The fluid infiltrated and distended the alveolar septa which were preceded by filling the alveolar spaces with albuminous fluid. Some sections revealed spaces completely filled with pink to deep eosin stained homogeneous material. In a few cases, sharp holes were visible which represented the trapped air bubbles. The higher the content of protein in the oedema fluid, the more intensely it took the stain. The blood vessels in alveolar walls were dilated and filled with blood.

Hyperamia:

Macrosopic appearance:- Focal lesions were mostly present. In the more pronounced cases, lesions were mostly observed in the anteroventral border of the apical and cardiac lobes. These areas were swollen dark red in colour and when cut blood with frothy exudate escaped.

Microscopic appearance:- The interalveolar septa were widened due to distended capillaries which were engorged with blood. Few to numerous erythrocytes and leucocytes had escaped
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lobes. These areas were swollen dark red in colour and when cut
blood with frothy exudate escaped.

Microscopic appearance:– The interalveolar septa were
widdened due to distended capillaries which were engorged with
blood. Few to numerous erythrocytes and leucocytes had escaped
in the alveoli (fig.38). In some cases, the oedema fluid were also present in few alveoli. In some cases, bronchitis and bronchiolitis with some erythrocytes in the lumen were noticed at places. A mild peribronchial hyperplasia was noticed in them. Few peribronchial vessels were also dilated and engorged.

From two cases suspected as infected with pasteurellosis belonging to the Government Cattle Farm, Patna, bipolar organisms were recovered when plated on agar plates. The morphology of the organisms and its colonies were suggestive of *pasteurella* sp.

**Oedema:**

**Macroscopic appearance:** The lungs were pale, voluminous, heavy doughy, showed pitting on pressure and the cut surface when squeezed, frothy material oozed out. Such material was also present in bronchi. In some cases, fluid was also found in the pleural cavity. Interlobular septa were distended by gelatinous fluid which were distinctly visible in the gross specimens.

**Microscopic appearance:** In some cases there was interstitial oedema infiltrating and distending the alveolar septa. This type of changes were sometimes accompanied by alveoli filled with albuminous fluid which in the histological preparation revealed alveoli filled with pink to eosin stained homogenous material (fig.39).

**Chronic Venous Congestion:**

Out of 300 specimens, 9 cases (3%) showed the changes
of chronic venous congestion.

**Macroscopic appearance:** Lungs were voluminous, dark red in colour, firm in consistency and indurated. When cut, it looked dark red, swollen and moist.

**Microscopic appearance:** Alveolar vessels were widely dilated and tortuous. Few vessels were present in the alveoli. The characteristic feature of this condition was the presence of large phagocytic cells filled with brown pigment. These granules gave the characteristic prussian blue reaction of iron. There was marked increase in fibrous tissue in the interstitium of the lung parenchyma which was responsible for the induration. The pigment which was carried by macrophages was also present in the interalveolar tissue. The changes in the bronchial walls were those of dilatation of capillaries and thickening of the walls.

**Abscesses:**

Out of 300 specimens, 10 cases (3.3%) showed lesions of varying size of abscesses.

**Macroscopic appearance:** Large abscesses were upto the size of a tennis ball with prominent, raised nodular bulgins over the surface of the lung. Some of the abscesses contained thick cheesy pus and other few cases had greyish to reddish thin pus. In few cases, abscesses contained almost dried up cheesy material. A calf lung contained tiny multiple abscesses distributed all over the lung parenchyma. In most of the cases abscesses
had dense fibrous tissue capsule.

**Microscopic appearance:** The section from tiny abscesses revealed areas of caseation, necrosis, cellular accumulation and new fibrous tissue formation around the necrotic mass.

**Pulmonary infarcts:**

Out of 300 specimens, 2 (0.6%) showed pulmonary infarction.

**Macroscopic appearance:** The lungs had some depressed grey and dry areas.

**Microscopic appearance:** The lung parenchyma was seen undergoing degenerative changes from inside out, the changes were accumulation of cellular debris homogenous cellular mass (dead alveolar tissue with lost cellular detail). Areas of atelectasis leucocytic infiltration in the surrounding necrotic mass and connective tissue forming the capsule and isolating the dead areas were seen.

**Fibrosis:**

Out of 300 specimens of the lungs, 5 (1.6%) showed changes of pronounced fibrosis.

**Macroscopic appearance:** Lungs were firm and grey, and cut with great difficulty.

**Microscopic appearance:** Parenchyma at places was replaced by fibrous tissue. In the section stained with Vangisons Picrofuchsin method. The areas dominated by connective tissue
elements were distinctly visible. Areas of atelectasis were also seen.

Adenomatosis:

Out of 300 specimens, 1 (0.3%) showed changes simulating that of pulmonary adenomatosis in the lung specimen from local slaughter house. The material belonged to a old bullock.

Macroscopic appearance:— Nodules of varying sizes were observed in all the lobes of the lungs. The lungs looked bulky and heavy, emphysema were present at few places.

Microscopic appearance:— Changes were those of intense hyperplasia and hypertrophy of the alveolar epithelium. Alveolar lining epithelium had become cuboidal to round with big round nucleus. Papillary projections of the epithelium due to proliferated lining cells had almost completely filled the alveolar spaces (fig.40). These epithelial cells were found desquamated in many of the alveoli looking as free cells. The bronchial epithelium likewise showed papillary projections deep into the lumen of the bronchi and bronchioles.

**TABLE X**

<table>
<thead>
<tr>
<th>No. of cases</th>
<th>No. examined</th>
<th>Conditions of lung</th>
<th>Etiological agents recovered in smears and/or isolated.</th>
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<tr>
<td>20</td>
<td>12</td>
<td>Exudative pneumonia (Broncho pn.)</td>
<td>Coryne bacterium sp. - 4 Staphylococci sp. - 3 Streptococci sp. - 2 Fusiformis sp. - 1</td>
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<tr>
<td>4</td>
<td>4</td>
<td>(Fibrinous pn.)</td>
<td>Pasteurella sp. - 2</td>
</tr>
<tr>
<td>15</td>
<td>2</td>
<td>Hyperaemia</td>
<td>Pasteurella sp. - 2</td>
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<tr>
<td>14</td>
<td>10</td>
<td>Pulmonary T.B.</td>
<td>Mycobacterium tuber. - 10</td>
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<td>2</td>
<td>Calf pn.like</td>
<td>Suspected virus r - 2</td>
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<tr>
<td>32</td>
<td>12</td>
<td>Aspergillosis</td>
<td>Aspergillus sp. - 12</td>
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The above table shows that there was a marked decrease in the normal value of haemo-

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<th>3.00</th>
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<td>9.6</td>
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**Normal**

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**TABLE X**

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Graph showing incidence of pneumonic lesion at autopsy in bovines.

Scale:
1 small sq = 1 case

Number of Cadavers:
- Jan: 8
- Feb: 5
- Mar: 2
- Apr: 3
- May: 4
- Jun: 3
- Jul: 2
- Aug: 1
- Sep: 1
- Oct: 2
- Nov: 3
- Dec: 5
Graph Showing Incidence of Respiratory Diseases in Bovines

Scale
1 small sq = 2 cases

Number Cases Treated

Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec
Area surveyed for pulmonary diseases

Nepal
The aetiology of bovine respiratory infections is both very queer and complex. Several aetiological factors are associated with pathogenicity of respiratory diseases. The environmental factors play an extremely important role in the aetiology of respiratory diseases in bovines by providing direct stress and also by creating favourable ecological situations which may alter or modulate the pathogenic capacities of the various causative agents. It appears from the survey of the incidence of respiratory diseases in cattle and buffaloes that pulmonary group of diseases is general and pneumonia in particular had a very high incidence. Pneumonia is one of the main causes of great mortality in cattle and buffaloes, especially young stock are more prone to this group of diseases. Other major respiratory and enteritis have also been contagious factors (viruses and V.E. agents) present to which enormous death in calves.

It is evident from the tables 3 and 4 that more than 80% losses in cattle is attributed to one or the other factor in coccidiosis. As shown in table 8, the calf loses under 6 months of age is the highest. The causation appears to play an important role in the aetiology of bovine respiratory diseases. The effects of seasonal changes are clearly noted in this context. In the present survey, based on different materials, collected from diverse sources, as is shown in the tables 3, 5 and 6, the aetiology of the bovine respiratory disease has almost never a seasonal incidence which is noticed at first during the late
DISCUSSION

The epizootiology of bovine respiratory infections is both very queer and complex. Several etiological factors are associated with pathogenicity of respiratory diseases. The environmental factors play an extremely important role in the epizootiology of respiratory diseases in bovines by providing direct stress and also by making favourable ecological situations which may alter or exalt the pathogenic capacities of the various causative agents. It appears from the survey of the incidence of respiratory diseases in cattle and buffaloes that pulmonary group of diseases in general and pneumonia in particular had a very high incidence. Pneumonia is one of the main causes of great mortality in cattle and buffaloes, especially young stock are more prone to this group of disease. Poor natural immunity unthriftness and probably also some contagious factors (viruses and PLV agents) seem to cause enormous death in calves.

It is evident from the tables 1 and 4 that more than 50% losses in bovines is attributed, in some or the other form, to pneumonia. As shown in table 2, the calf loss under 6 months of age is the highest. The season appears to play an important role in the epizootiology of bovine respiratory diseases. The effects of seasonal changes are clearly noted in this context. In the present survey, based on different materials, collected from diverse sources, as is shown in the tables 3, 5 and 6, the epizootiology of the bovine respiratory disease has almost rather a seasonal incidence which is noticed at first during the late
rain. The diagram 1 and 2 show a rather abrupt rise in incidence of the disease during the months of September and October, and it gradually subsided during the rest of the months of a year. It is probable that due to some obscure reason, disease is picked up by several young animals during the months of September and October of a year, which are the months of gradual transition from summer to winter season. The incidence of pulmonary form of pasteurellosis has somewhat a seasonal occurrence. Almost every year, severe deaths in bovines occur due to pneumonic changes. The disease commences during the late rain and lasts for a few weeks. There is movement of animals from neighbouring Tarai area to Kathmandu valley and other hilly localities of Nepal. During the subsequent months just after rains, the incidence of pasteurellosis is frequently reported from the localities where the animals had access to reach or stay. Some of these animals in transit also pick up the infection. The animals travelled mostly on their own feet or are transported by trucks.

In 1971, the epizootic of pasteurellosis was found to result in heavy calf mortality in Government Cattle Farm, Patna. The continuous rain seemed to reduce the resistance of the cattle and more over, the cattle and buffaloes had been brought from flood affected areas and stationed near about the premises of Government Cattle Farm, Patna. It is not quite clear whether the disease broke out on the farm spontaneously among the susceptible calves or was carried through some other agencies.

With special reference to pneumonia in the animals of Government Cattle Farm, the cause specific death ratio (C-SDR)
was 17469.9. As shown in tables 8 and 9, there are several etiological factors which may be responsible for the pulmonary conditions in bovines. The high incidence of hydatid cyst in the material under study suggests that numerous stray dogs infected with tape worm, constantly contaminate the pasture with its faecal material. Animals pick up the infection by grazing such pasture.

An analysis of the composition of Government Cattle Farm material indicated that there is high incidence of pneumonia in cattle as compared with other diseases such as coryllophoriasis, pulmonary tuberculosis etc. in Tharparkar cattle. Tuberculosis is a very common observation affecting either lung or lymph nodes. The lesions contain usually caseous and calcified nodules, similar to that described by Naik (1932).

Incidence of pneumonia occupied the highest position among the G.C.F. cattle (0-3 yr. of age), however, its incidence was highest in calves under 6 months of the age. Pneumonia in Tharparkar cattle was quite high during the late rains. As indicated by the survey of the G.C.F., B.V.C.H. and non- G.C.F. and B.V.C.H. materials, more incidences of pneumonia occurred during the late rain or early winter. The month September of a year predisposed greatly the cattle and buffaloes (mostly calves) to pneumonia. In Nepal, the incidence of respiratory diseases was quite high during the months of August, September and October of a year.

It appears from the table 8, that mycotic elements have been responsible for cases of pneumonitis in several cattle and buffaloes and the pulmonary mycosis is now emerging as a new
bovine disease. Hitherto, pulmonary mycosis was not known either in Nepal or in Bihar.

Although inflammatory growth caused by schistosomiasis in nasal mucosa, were observed quite frequently, nasal polyp which was observed in a non-descript cattle seemed to have been caused by some factors other than that of schistosomes.

According to Jubb and Kennedy (1963), congenital pulmonary cysts are rare anomalies and have been observed in dogs and foals. The congenital pulmonary cyst as observed under the present studies, was noticed in the lung of a Tharparkar calf. There were several pin point holes in the right diaphragmatic lobe and the cystic cavities in the lung parenchyma were of different shape and sizes. This condition seems to be quite rare in bovines.

Exudative pneumonia, especially bronchopneumonia in cattle and buffaloes, was commonly observed and the lesions which were similar to those of catarrhal pneumonia as described by Carter and Gillman (1921). According to them, *corynebacterium pyogenes*, staphylococci etc. were isolated from the affected lungs. The lesions of necrosis and suppuration caused by *C. pyogenes* in the lungs of the calves are in accord with the description given by Lovell and Hughes (1935). Lovell and Hughes (1935), Lebedev (1937), Thorp and Hallman (1939), Lovell (1939) isolated *C. pyogenes* from the affected lungs of the cattle. The pattern of distribution of pneumatic lesions in the antero-ventral portions of the apical, cardiac, intermediate and diaphragmatic lobes was similar to that described by Langham et al. (1942)
Willems (1931) described the lesions of bronchopneumonia in bovines and found serofibrinous exudate in the thoracic cavity, petechiae, various stages of hepatization and thickening of interlobular septa. Fibrinous pneumonia is well known in human beings but incidences of this type of pneumonia are not a well marked condition in cattle and buffaloes. However, in the present studies, fibrinous exudate covering the pleura of the lungs and also infiltrating into the parenchyma, was noted. Some fibrinous flakes were also found in the pleural cavity. From the lungs of some of such cases organisms of *Pasteurella species* were isolated. There were 4% cases of fibrinous pneumonia in the specimens of the lungs included under the present observation. The pathogenicity of these organisms was also tested on white mice, which was found to be quite virulent causing death of the mice. Failure of isolating any *Pasteurella* or mycoplasma organisms from some of such cases indicate involvement of some other unknown factor. Jubb and Kennedy (1963) and Omar (1966) described pneumonia with infiltration of fibrinous exudate in the lung and believed it to be caused by *Pasteurella* or mycoplasma organisms.

The heavy mortality of Tharparkar calves might be due to some contagious nature of the etiological factor responsible for the death of the calves. Debilitating and predisposing factors such as age etc., also appeared to have certain role in incidence of such diseases. The lungs of most of the calves had gross pneumonic lesions. The histological pictures from these specimens showed lymphocytic cuffs in the peribronchial and perivascular spaces. White mice were inoculated intranasally with cell free filtrate from some of these lung specimens. The mice
developed rise in temperature above normal and pneumonic lesions were present in the lungs. There was catarrhal exudate in alveoli and bronchioles and few mononuclear cells were also visible in the sections of the mice lung. The histological pattern of lesions of bronchopneumonia in some calves indicated possibility of involvement of some viral elements or unknown factors. According to Jubb and Kennedy (1963), it appears adequately established, that one and perhaps the most common enzootic pneumonia of calves (calf pneumonia) is caused by a large virus of the PLV group. However, in the present study inclusion bodies could not be observed in the histological preparations, of pneumonic lungs. As based on the patterns of result obtained under the present studies, the probability of the involvement of the virus in the pneumonia of cattle opens the road for further research on this category of etiological factor in detail.

The pneumonic changes observed in majority of cases (30.3%) in cattle and buffaloes were predominantly proliferative in nature. It is difficult to establish any one specific cause as responsible for these conditions. Hyaline membrane in the cattle affected with parasitic bronchitis has been described by Jarrett (1957). Pink stained almost hyaline material was noticed in the alveoli and bronchioles of the lungs infected with Dictyocaulus viviparus in the present study. Runnels et al. (1960) incriminated virus as causative factor in interstitial pneumonia. Omar (1966) described PLV agent as causative for interstitial bronchopneumonia. Thickening of the alveolar walls in enzootic pneumonia of calves was described by Van Rensberg and Every (1961). In the present studies, such changes as
thickened alveolar septa, lymphocytic infiltration in the peribronchial and perivascular spaces in lungs of māy cattle and buffaloes were the dominant lesions, seen microscopically. Other lesions of interstitial pneumonia such as thickening of interalveolar septa with exudate or cellular infiltration, epithelialization of lining epithelium as well as in some cases a few giant cells in the alveoli were also noticed. Some of the changes observed in this study are, suggestive of virus or PLV agent as one of the causative factors responsible for the pathological lesions of at least some conditions in this group of disease in Indo-Nepal sub-continent. Lugnibuhl (1960) claimed to have produced chronic interstitial pneumonia or adenomatosis by injecting bacteria free filtrate. Allergy, dust etc. were believed to be the cause of adenomatosis (Blood, 1962). Mohiyudddeen et al. (1967) observed jaagziekte lesions in buffalo. In the present investigation, only one case of lung belonging to an old bullock from local slaughter house, was found to contain this lesion. Neither any etiological factor could be isolated from this specimen, nor any observable extraneous irritant was noted in the histological preparation. The lesions were diffusely scattered throughout the lungs. These were solid, heavy and the lung was pale in colour. Microscopic lesions were like those described by other workers, mostly hyperplasia and hypertrophy of the lining epithelial cells, which had become almost round.

Well marked lesions of tuberculosis were usually present close to the dorsal border of the diaphragmatic lobes in the majority of cases. However, the tuberculosaic nodules were also distributed throughout the lungs. Most of the lesions
were noticed either in the lungs or lymph nodes. This finding
is similar to the observation of Iyer (1944). No cases of tuber-
culosis in the lung of Nepali buffaloes could be found. This
does not rule out the possibility of presence of tuberculosis
in buffaloes in Nepal. Tuberculosis in buffaloes have been
reported in India and many other parts of the world by several
workers. Tuberculosis of lung were commonly found in the older
cattle than in the young. The present finding of the pattern of
lesions and more incidences in older cattle, is almost in accord
with the observations of Kuppuswamy and Singh (1968), Singh and
Prasad (1971).

Mycotic diseases have been reported in many
species of the domestic animals and human beings. Lesions of
pulmonary mycosis have been described by many workers in many
parts of the world, including Austwick (1962), Molatto and Busey
(1963) who described granulomatous pneumonia with septed hyphae
and Aspergillus type of conido spore in a cow. Narayana et al.
(1964) also recorded a case of mycotic pneumonia. In the present
investigations, 32 cases of mycotic pneumonia were noticed.
Lesions observed, are in full conformity with the findings of
Cordes et al. (1964), who described fatal fulminating mycotic
pneumonia in dairy cows. In the present study, the lesions con-
sisting of hyphae or fungi in different structures of the lungs
in cattle and buffaloes, were observed. Caseous areas almost
tending to be capsulated by fibrous tissue were seen. Nodules
comprising mostly of caseous material, macrophages, lymphocytes,
neutrophils etc. were present in some specimens only. Other
dominant changes were, thickening of alveolar septa, thrombosed
pulmonary blood vessels, chronic arteritis, conspicuous hyperplasia of the media and intima with infiltration of some round cells. Mycotic elements had been noticed even within blood vessels, alveoli, bronchioles and caseated mass. Epithelialization of the alveolar lining and at places thickened alveolar walls were also some of the features in this condition. Formation of giant cells seemed to be not of much importance in this disease. The pattern of lesions in some of the histological preparations were suggestive of the involvement of more than one fungal agent. From 12 cases of Nepali buffalo lungs, fungus were isolated in culture, which resemble morphologically, fungus belonging to Aspergillus group. The wide spread distribution of *A. fumigatus* in nature may probably be the cause of infection in bovine. Pulmonary mycosis appears to have debilitating effect in buffaloes in Nepal. Due to this, the production of milk and meat are decidedly affected. The animals which are sent for slaughter generally have poor nutritional condition with certain degree of respiratory distress. Animals affected with mycotic pneumonia may also manifest similar symptoms of respiratory distress.

Gaiger (1910), Mohan (1948), Ramanujachari and Alwar (1954), Awadhiya and Mehata (1961) described lung worm disease in bovine. Michel and Shand (1955) reported outbreaks of husk in cattle. In the present investigations, the lesions of verminous pneumonia are in agreement with the findings of Jarrett et al. (1960). The lesions as noted were mainly proliferative in nature. A few foreign body giant cells were seen in the affected lungs, when histological sections were examined. Adult parasites were recovered from bronchioles and bronchi. The larval fragments and
embryonated ova were noticed in the alveoli, in histological sections. Granulomatous reaction was also apparent in some of the sections.

In the lung infected with hydatidosis, there was a fibrous tissue capsule surrounding the cyst wall. Microscopically well developed granulomatous tissue consisting of fibroblasts, inflammatory cells, etc. were seen. Similar type of lesions were described by Craig and Faust (1949), Smith and Johnes (1963).

Lesions of emphysema were noted in 10 cases. They were present in two forms i.e. focal and diffused. Focal lesions were present in the lung having areas of consolidation, atelectasis and also around the tuberculous nodules as compensatory emphysema. In diffused cases, thoracic cavity was completely filled with emphysematous lung. Allergy has been regarded as one of the factors responsible for this emphysematous condition. Hyperaemia and oedema were noticed in 47 cases of the 300 lung examined. The lung of cattle that died of piroplasmosis and calves died of FMD had well marked oedema with some degree of hyperaemia. Hyperaemia was also noted in pneumonic cases and most of the lungs examined at autopsy had this condition. From two cases of hyperaemic lungs, pasteurella organisms were isolated. Hyperaemia seems to be the primary stage in the acute inflammation of the lungs. Atelectasis, infarction, abscess and fibrosis were also noticed in many of the lungs examined.

To the author's knowledge no haematological studies have so far been carried out in cases of bovine cotylophoriasis. Singh and Lakra (1971) described pathologic changes in cases of
corylophorum corylophorum infection in calves in course of their investigations into a natural outbreak of this condition in cattle. Cases of coryllophorasis with pneumonic changes in the lungs of Tharparkar calves were studied in the present investigation. Haematological study of such cases revealed marked decrease in haemoglobin percentage and erythrocytic count.
CHAPTER - VI

SUMMARY

Influenza changes in the different respiratory diseases of cocks and hens in Nepal and Bihar, have been investigated.

Chronic bronchitis and the highest prevalence in association with bowel diseases is diagnosed. The incidence of pneumonia was very high in the early fall and early winter months of the year.

Larval figures were recorded for the first time in Nepal and Bihar. To the degree of severity affected with pulmonary systems, including oral brood was occasionally usually subsporadically by retropharyngeal, submandibular, posterior, nasal, and tracheal infection by hypertrophic bronchitis, excessive frequency of nasopharyngeal spread. The erosion of the trachea, vesicles, pharynx, and lungs. In the advance state of the disease, tracheal lesions.

Infectious diseases similar to those of viral pneumoza were observed in association to Baccharis calves of government cattle diseases.

Epidemiological studies were infections with some cases of disease, the number of diseased cattle.

Serological studies were carried out in some
SUMMARY

Pathologic changes in the different respiratory diseases of the cattle and buffaloes in Nepal and Bihar, have been described.

Pneumonic cases had the highest prevalence in comparison to other diseases diagnosed. The incidence of pneumonia was very high in calves under 0-6 months of age. Epizootics were mostly observed during the late rains and early winter months of a year.

Pulmonary mycosis is reported for the first time in Nepal and Bihar. In the lungs of bovine affected with pulmonal mycosis, numerous small grey areas scattered usually subpleurally were noticed. Coagulative necrosis, caseation and infiltration of leucocytes, and large number of macrophages around the necrotic areas with tendency of capsulation, were seen. Degenerative changes, chronic arteritis and thrombosis in the arteries were the common histological lesions.

Pathologic changes almost similar to those of viral pneumonia in cattle were observed in Tharparkar calves of Government Cattle Farm, Patna.

Laboratory animals such as mice could be infected with cell free filtrate from the lungs of diseased calves.

Haematological studies were carried out in cases of cotylophoriasis accompanied by pneumonic changes. Decrease in haemoglobin percentage and red cell count were found.
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