BRONCHOPNEUMONIA IN CATTLE

by

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Bronchopneumonia is one of the most serious causes of economic loss in the cattle industry. It is commonly seen in domestic animals, but is most prevalent in cattle. The condition is not a definite entity, but should be regarded as a general type of pulmonary reaction to a number of different causes such as bacteria, viruses, fungi, parasites and other types of foreign matter.

The outbreaks of respiratory infections have reached serious proportions in some areas of the world where intensive systems of feeding and management have been introduced. Seasonal effects are important contributors to the outbreaks of respiratory infections. The disease occurs chiefly in low—lying regions with extensive pasture land. It is prevalent in feed—lot calves after they have been collected from small individual farms and put together in the large feed—lots. The age and breed of animals may influence the outbreaks, such as shipping fever occurs commonly in young beef cattle.

Bronchopneumonia is a name given to an inflammation of the lungs and bronchioles. The condition will primarily affect the terminal bronchioles which become filled with tenaceous purulent mucus and surrounded by areas of consolidation. The disease may be primary or secondary, but is usually secondary in character, following infections of the upper respiratory tract. It usually begins as a tracheobronchitis and extends to a few or many lobules. Consolidation may be gradual or rapid.

Etiology.

The etiology of bronchopneumonia in cattle is very complex. The intensive management conditions, transportation of animals and physical stresses are predisposing factors which lower the animal's resistance and allow a number of infectious agents to precipitate the disease (Mckercher, 1964). Inhalation of smoke and fire, high concentration of ammonia, and increased humidity of air (Kovaleva et al., 1961) and aspiration of liquid material give rise to bronchopneumonia which is then proceeded by infectious agents. Bacteria, fungi, and parasites seem to be the main causes of bronchopneumonia.

MICROORGANISMS

Bacteria.

Many species of pathogenic bacteria may cause bronchopneumonia in cattle. In calves, Pasteurella spp., especially P. multocida and Corynebacterium pyogenes are often found. Actinobacillus actinoides has been isolated from pneumonic calf lungs showing all types of lesions but is not believed to be a primary etiological agent (Levi & Cotchin, 1950; Blakemore, 1945). This organism can neither produce pneumonia nor contribute significantly to the severety of the disease produced by e. g. parainfluenza 3 virus. Staphylococci spp. have been isolated from the lungs of calves during an outbreak of pneumonia (Tutt, 1941) and from the lungs of a calf which was injected with a suspension of bronchopneumonic calf lungs (Jennings & Glover, 1952). Streptococci are considered as probable causative agents (Thorp & Hallman, 1939) and have been isolated from pneumonic calf lungs (Barócsai & Farkas, 1964). Diplococcus pneumoniae have been found in pneumonic calf lungs (Bratanovic et al., 1964). Carpenter & Giman (1921) found Pseudomonas aeruginosa associa-

ted with pneumonia. Neisseria catarrhalis has been isolated either in pure culture or in combination with other organisms from pneumonic calf lungs (Hunter & Harbourne, 1964). Salmonella dublin has been isolated from pneumonic lungs (Levi & Cotchin, 1950; Harbourne et al,. 1965). Examinations at Veterinary Investigation Centres in the U.K. during 1956–61 revealed that Salmonella was an organism causing pneumonia in calves (Maff, 1964). Haemophilus spp. were consistently isolated from the cases examined by several authors (Thorp et al., 1942; Lamont et kerr, 1939, Watt, 1952). Coliforms have been isolated from pneumonic calf lungs (Jennings & Clover, 1952; Thorp & Hallman, 1939; Barocsai & Farkas, 1964, Maff, 1964). E. coli was also found in mycotic pneumonia. (Mills & Hirth, 1967).

Corynebacterium pyogenes has been recovered from lungs of calves suffering from bronchopneumonia and shipping fever (Jennings & Glover, 1952; Levi & Cotchin, 1950; Carpenter & Gilman, 1921; Carter, 1954). This organism probably acts as a secondary agent which associates with unidentified viruses (Bratanovic et al., 1964)

Pasteurella multocida has been recovered from pneumonic calf lungs in Canada (Carter & Rowsell, 1958). P. multocida and P. hemolytica have been isolated from bronchopneumonic lungs by a number of workers (Carter, 1954; Bratanovic, 1964; Levi & Cotchin, 1950; Palotay & Newhall, 1958; Carter & McSherry, 1955). It has been proved that they can provoke bronchopneumonia although no clear-cut evidence as to their possibilities as primary agents has been found (Collier et at., 1960, Heddleston et al., 1962; Hetrick et al., 1963; Sorensen et al., 1964). There is some indication though that debilitating factors are necessary to provoke the disease (Sorensen et al., 1964; Carter 1956; Cody, 1965).

Viruses

The viruses have been observed as etiological agents causing pneumonia in cattle by many workers. Since Carpenter & Gilman (1921) and Thorp et al. (1942) reported that bacteria isolated from pneumonic calf lungs could not produce pneumonia, the interest of many workers has been directed against virus as a possible agent. The research into this problem has been done in the form of transmission experiments (Baker, 1943 and Watt, 1952), virus isolation (Jarrett, 1954, 1956) serological studies.

Parainfluenza type 3 (PI3 or MP3 virus).

Myxovirus parainfluenza 3, bovine type, was first isolated from cattle with shipping fever and classified as the SF4 strain by Reisinger et al. (1959). Since then the parainfluenza 3 virus has been isolated from cattle lungs in many parts of the world. In the U.S.A. (Abinanti & Huebner, 1959; Abinanti et al., 1960; Gale & King, 1961; wood et al 1964, Sorensen et al., 1964, Hamdy, 1965), in Canada (Jolly & Ditchfield, 1965), in the U.K. (Dawson & Cruikshank, 1963; Dawson et al., 1964; Betts et al., 1964; Omar, 1965) in Sweden (Bakos & Dinter, 1960; Dinter, & Bakos), in Yugoslavia (Klemenc et al., 1964), in France (Charton et al., 1965), in Germany (Bögel, 1961), in Japan (Inaba et al., 1963, 1964), in Italy (Scatozza, 1966) and in Denmark (Michelsen, 1964).

Electronmicroscopy has been used in identification of the morphological characters of the Myxovirus group (Berkaloff et al., 1965). Jurmanova et al. (1965) have shown that diploid bovine embryonic cell strains (BES) were suitable substrates for isolating viruses causing bronchopneumonia in cattle.

The wide geographic distribution of PI3 virus among cattle in many parts of the world is evident from serological studies. Significant titres of hemagglutination-

inhibiting antibodies against the virus have been found in a large proportion of animals suffering from respiratory diseases and healthy animals (Abinanti et al., 1961; Hoerlin et al.. 1959; Dawson & Darbyshire, 1964 ; Harbourne, 1966; Inaba et al., 1965). In Northern Sweden, the PI3 virus which associated with mucosal disease seemed to be introduced by cattle purchased from other parts of the country (Bakos & Dinter, 1960).

Various strains of PI3 virus have been tested in calves for their abilities to produce a respiratory disease (Woods et al., 1965; Inaba et al., 1964; Omar et al., 1966; Betts et al., 1964). In studies of the combined effect of PI3 virus and Pasteurella spp. in shipping fever, a severe disease resembling more or less the natural infection was produced only when the virus was inoculated with those organisms (Hetrick et al., 1963; Heddleston et al., 1962, Sorensen et al., 1964. and Hamdy, 1965). Calves which received a high initial level of passively transferred antibodies to PI 3 virus may not become susceptible to the infection until between 19 and 23 weeks of age (Dawson, 1966), and when vaccinated against this virus, antibodies were present 21 days after vaccination (Cvetnic et al., 1966).

Infectious Bovine Rhinotracheitis (IBR) virus.

The IBR virus is commonly found in respiratory infections. This virus was first isolated in the U.S.A. by Madin et al. (1956). The disease caused by the virus in cattle in the U.S.A. was first described by Schroeder & Moys (1954) and Mckercher et al. (1955), since then by other workers (McKercher et al., 1957; Gillespie et al., 1957). The virus has been isolated from lungs, nasal discharge or faeces of cattle with respiratory infections in many parts or the world. In Italy, (Moretti et al., 1964), in Germany (Grunder et al., 1960), in New Zealand (Webster & Manktelow,

1959), in Canada, (Studdert et al., 1961), in South Africa (Mare & Van Rendsburg, 1961), In Hungary (Barocsai & Farkas, 1964), in Rumania, (Coman, 1964) and in the U.K. (Darbyshire et al., 1962). Different strains of the IBR virus have been isolated from affected calves by Darbyshire & Shanks (1963).

The virus has been proved for its ability to provoke a respiratory infection in calves (Baker et al., 1960; Collier et al., 1960) and intranuclear inclusion bodies have been found in the epithelial cells of the respiratory tract (Crandell et al., 1959).

The IBR virus seems to be the primary etiological agent of the respiratory infection (McKercher, 1964). Although in studies of the combined effect of IBR virus and P hemolytica on cattle, Colleir et al. (1960) reported that the duration of the disease was greater than in those given only one agent, the evidence of serum antibodies against IBR virus has been found in sick and healthy animals (Dawson & Darbyshire, 1964; Harbourne, 1966).

Adenovirus.

The bovine adenoviruses were Previously isolated by Klein et al. (1959) from the faeces of normal cattle and from an apparently normal calf (Klein et al., 1960). Different strains of the virus have been isolated from pneumonic calf lungs (Bartha & Aldasy, 1964; Cole, 1967) and from calves with pneumo-enteritis (Aldasy et al., 1964) in Hungary.

The adenoviruses have been proved to produce respiratory syndromes by various routes of inoculation (Mohanty & Lillie, 1965; Darbyshire et al., 1966, 1965). Darbyshire et al. (1966, 1965) reported that the virus was recovered from conjunctiva, nose and faeces for up to 11 days after inoculation.

The presence of serum antibodies against adenoviruses has been revealed in cattle suffering from respiratory infections (Darbyshire & Pereira, 1964; Harbourne, 1966). Precipitation reactions have been shown to occur with adenovirus systems by means of gel diffusion test (Darbyshire & Pereira, 1964).

Reovirus.

The reovirus type 3 was first isolated from the faeces of normal calves in Maryland by Rosen & Abinanti (1960). Fifty-nine strains of all the three serotypes have been isolated from the faeces of cattle in Maryland, and all except type 2 have proved to be able to produce pulmonary infections (Rosen et al., 1963).

In the U.K. serological evidence indicates that these viruses are involved in the outbreaks of respiratory diseases (Dawson et al., 1966). They have been tested in calves for their abilities to produce respiratory disease, but they failed to elicit the disease. Nevertheless, the animals developed antibodies against these viruses (Lamont et al., 1968; Rosen & Abinanti, 1960). In studies of the combined effect of reovirus type 1 and P. multocida in calves by various routes of inoculation, the animals developed a mild respiratory disease (Trainor et al., 1966). At present there are no reports about the isolation of reoviruses from other parts of the world.

Other viruses.

Many other viruses have been isolated from the lungs or nasal discharge of calves and cattle suffering from respiratory infections. In Hungary, Shope's virus has been isolated from cattle with bronchopneumonia (Romvary et al., 1962). In Germany, rhinitis virus has been isolated from calf with respiratory diseases (Böhm, Bögel 1962). Enteroviruses have been isolated from cattle and calves with pneumo-enteritis (Huck & Cartwright, 1964) Cvetnic et al., 1964; Pritchard et al., 1956; Burki & Germann, 1964).

Psittacosis—Lymphogranuloma Venerum (PLV) Group of Organisms.

Virus of the PLV group in cattle was first isolated in the U.S.A. by York & Baker (1951) from portions of intestine and faeces of apparently healthy calves and named Miyagawanella bovis. Since then these organisms have been isolated from lungs and nasal discharge of cattle suffering from respiratory diseases in many parts of the world. In Germany (Semordjiev et al. 1964), in Hungary (Romvary, 1964), in Japan (Matumoto et al., 1955; Omori et al., 1960), in Rumania (Popovici, 1964; Sarateanu et al., 1961), and in the U.S.A. (Palotay & Christensen, 1959).

Different strains of PLV organisms have been isolated from calves with respiratory infection in Rumania (Popovici, 1964).

The PLV organisms have been tested for their abilities to produce respiratory disease in cattle by various routes of inoculation, and the diseases provoked can be characterized as typical types of pneumonia (Palotay & Christensen, 1959; Polony et al., 1960, Omori, 1950, Popovici, 1964). In addition to causing respiratory infection, PLV organisms may produce sporadic bovine encephalitis (McNutt & Waller, 1940; French & Snowdon, 1960) and epizootic bovine abortion (Störz & McKercher, 1962).

Pleuropneumonia like Organisms (PPLO).

PPLO other than Mycoplasma mycoides have been isolated from calves with respiratory infections. The organisms have been isolated from bronchopneumonic calf lungs and nasal swabs of calves with respiratory disease in a few countries. In Canada (Carter, 1954; a, b, and Carter & McSherry, 1955), in the U.S.A. (Hamdy et al., 1958). in Australia (Johnston, 1963) and in the U.K. (Harbourne et al., 1965; Dawson et al., 1966).

Eighteen strains of Mycoplasma have been isolated from trachea, and from healthy and pneumonic calf lungs (Davies, 1967). Many strains of PPLO have been proved to be non—pathogenic for calves (Hamdy et al., 1958). Nasri (1966) reported that a culture of a virulent M. mycoides strain 121 contained both virulent and nonvirulent agents.

Fungi

Pneumonia caused by fungi is commonly seen in birds, but it seems to be more uncommon in cattle. It has been detected though in recent years in some fatal infections. The primary respiratory infection seems to be started by inhalation of spores from mouldy litter and feeds, especially hay and grain which have been damp and heated during storage. This disease usually occurs in summer (Rensberg & Every, 1961), September and rainfall (Hugh—Jones & Austwick, 1967). A nocardia-like organism has been isolated in pure culture from the affected organs of a calf that died after developing the disease. The lung showed severe inflammation and hepatization (Van der wall, 1964).Candida albicans has been isolated from pneumonic calf lungs (Mills & Hirth, 1967).

Coccidioidomycosis, a disease caused by coccidioides immitis, occurred enzootically in the U.S.A. (Ainsworth & Austwick, 1959; Jubb & Kennedy, 1963). Scholz & Meyer (1965) have isolated Mortierella polycephala from pneumonic lung of a cow. Rhodotorula mucilaginosa has been isolated from the lungs of calves suffering from bronchopneumonia (Galati, 1964). Mucor spp. and Aspergillus spp., especially A. fumigatus, have been commonly found in mycotic pneumonia in cattle (König et al., 1967; Cordes et al., 1964; Eggert and Romberg, 1960; Mollello & William Busey, 1963; Jubb & Kennedy, 1963; Austwick, 1962; Ainsworth, 1948; Ainsworth & Austwick, 1959; and King et al, 1965), and the disease is usually associated with myotic abortion

Transmission

The transmission of infection may occur by direct or indirect contacts. The direct contact happens by inhalation of infected droplets coughed up or exhaled by infected animals. In mycotic pneumonia the animals get the infection by inhalation of the spores from mouldy litter aud feeds. The indirect contact occurs by the ingestion of contaminated material. The disease may spread rapidly and affect a high proportion af the herd when the conditions are optimum as e.g. when feedlot cattle are maintained together in dam barns, while the spread may be slower in animals at pasture.

Pathogenesis.

A decrease in the resistance of the mucous membranes of the respiratory tract is most important for the possibilities of the infectious agents to propagate. The different agents seem to have different predilections as to sites, but spread from any part of the lung occurs easily.

In the acute stages following infection, the mucosa will be hyperaemic and oedematous, while in the chronic stages more progressive and to a certain extent irrepairable changes are seen. Exudation to the lumen of the lung is a very common finding.

In mycotic pneumonia, the hypae will penetrate the mucosa and the surrounding lung tissue and produce granulomatous lesions.

The bronchopneumonia will typically affect the ventral parts of all lobes, especially the spical, cardiac and intermediate lobes.

Clinical findings.

Attention will first be drawn to the animal by dullness, dry or moist cough and fast breathing. The animals stand with their heads extended, are stiff in their

movements, and later become recumbent still with the head advanced. The animals show depression, respiratory distress characterized by dilated nostrils and mouthbreath ing, rhinitis, conjunctivitis, pyrexia, diarrhoea and anorexia (Inaba, 1964; Dawson et al. 1965; Reisinger, 1959). In mycotic pneumonia bloody and foetid diarrhoea was observed by Rensburg & Every (1961) and Ainsworth and Austwick (1959). The nasal secretion varies from serous to mucupurulent, Conjunctivitis may be pronounced. Cordes et al. (1964) reported that expiration is reinforced by abdominal muscle-contraction. There is generally a rise in body temperature, reaching 40-41°C. In dairy cattle, an abrupt cessation of milk flow may be one of the first indications. There will be increased respiratory and heart rates. On auscultation of the chest the major abnormalities can be detected over the anterior and ventral parts of the lungs, bronchial sounds transmitted through consolidated lung, vesicular murmur, loud rales and a pleuritic friction rub may be heard. Palpation of the thorax will not reveal any particular abnormality. The heart beat may be audible more clearly than usual. Percussion may induce cough, cause pain and abnormal dullness is often found at the anterior and ventral parts of the lungs.

Clinical pathology.

Haematological findings, total red blood cell count, haemoglobin and haematocrit will mostly be normal. The leucocyte count usually shows leucocytosis and neutrophilia. In viral infection leucopenia is often seen (Schalm, 1965). Johnston (1963) reported that in PPLO infections, the animals showed leucopenia, eosinopenia, the lowest leucocyte count being 1100.

Diagnosis

The diagnosis is based on the history of the affected animals. Important factors are season and predisposing causes. The disease is usually recognizable by

one or more of the clinical symptoms found on examination of the respiratory system. Especially important are increased rate of respiration, cough, rales and dullness on auscultation and percussion over the chest. Allergic tests may be used in some cases. The isolation of organisms, hematological and serological findings provide specific means of diagnosis.

PARASITES

The most common parasites causing parasitic bronchitis and bronchopneumonia are Nematodes of the family Metastrongylidae, genus Dietyocaulus. Other species of parasites appear to be the cause of pneumonia only by accidental migration of the larvae through the lung. For instance, the migratory larval stage of A. vitulorum may cause pneumonia (Cameron, 1951 and Lapage, 1962). An acute pneumonia has been reported in 17 yearling cattle lo days after being put into a pen contaminated with A. lumbricoides ova (Allen, 1962). The post mortem findings revealed a pneumonia and the fourth stage larvae of A. lumbricoides have been found from bronchial scrapings. Anon (1964) reported a case of chronic bronchopneumonia possibly induced by pulmonary infection with liver flukes and Corynebacterium. Dictycauliasis or lungworm disease is an enzootic bronchitis and bronchopneumonia due to the presence of worms in the bronchi of cattle reported as long ago as in 1744 (Ruysch). In 1756 (Nicholls) the presence of bovine lungworms was correlated with the distinct disease. The disease occurs in all countries, but chiefly in low—lying regions with extensive pasture land, in boggy districts and with a wide variation depending on climatic conditions and season.

Usually all lungworm species are host—specific (Blood & Henderson, 1963). Lungworm disease in cattle is almost exclusively caused by Dictycaulus, viviparus

Enigk & Hildebrandt (1964) and Parfitt (1963). infected calves with 3.000–12.500 D. filaria of which a few larvae developed to the fifth stage, but none obtained sexual maturity. Animals of all all ages susceptible to this disease, but especially calves 4 to 10 months of age, The animals are affected during grazing on pastures in which the lungworm larvae are found.

The climate is an important factor governing the incidence of parasitic bronchitis (Rose, 1960), the highest incidence seems to occur in warm, wet summers.

Frick (1964) reported that the faecal counts of D. viviparus larvae in cattle reached a maximum in August, During a survey over two years of lungworm in cattle, Henriksen (1967) found that the percentage of positive samples was highest (over 40%) in July and thereafter gradually fell to about 20% in October.

Life cycle.

The mature worms live in the larger air passages of the lungs where their eggs are laid. The eggs may hatch in the lungs but are usually coughed up and swallowed and hatch while they pass through the alimentary tract of the host to produce first–stage larvae develop in the internal environment first into the second stage then into the third and infective stage the process taking less than seven days under optimum conditions. The infection of the host occurs by ingestion of larvae. They penetrate the wall of the small intestine and migrate through the mesenteric lymph nodes to the lymph vessels. From here they pass eventually to the venous blood stream, through the heart and reach the lungs. They penetrate the lung capillaries into the alveoli. and gradually ascend into the bronchioles passing through the fourth larval stage. After migration up to the bronchi they are mature.

Jarrett et al. (1957) stated that it was the fourth–stage larvae which left the lymph nodes to migrate to the lungs and that the pulmonary lesions were noticed

five days after initial infection. On the other hand, Douvres & Lucker (1958) working with guinea pigs recovered third-stage larvae from the lung 18 hours after administration. In experimental calves, Poynter et al. (1960) reported that the larvae reached the lung in twentyfour hours after infection.

Infectivity.

Michel (1959) reported that of the larvae passed in the faeces about 98 % will die out in the first 14 days. Climatic conditions were considered as the most important influencing the development and survival of the lungworm larvae from first-stage through to the infective stage (Rose, 1956, 1960). The rate of development is decreased in autumn and winter and increased in spring and summer. Michel & parfitt (1955) and Michel & Shand (1955) observed a survival period, in the autumn and early winter, of 4 months, and they expressed the opinion that overwintring of the larvae seemed unlikely to occur. Popov. et al. (1965) reported that the larvae survived up to 8-15 days in summer, up to 15-30 days in spring and up to 113 days in winter. Enigk & Düwel (1961) showed that the larvae could survive in winter and still remain infective. In studies of the epidemiology of parasitic bronchitis Jarrett at al. (1955) reported that the larvae of D. viviparus could survive and remain infective for one year on pasture.

The infective larvae are not very active and rarely migrate from the faeces to the herbage, and as the animals usually avoid to graze near the faeces, some method of dissemination of the larvae is necessary. Semifluid faeces thus has the ability to spread and disperse more larvae on the herbage than the normal faeces (Rose & Michel, 1957).

In studies on the role of fungus Pilobolus in the spread of the infective larvae of D. viviparus Robinson (1962) and Robinson et al. (1962) found that growth of sporangiphores of Pilobolus usually occurred on the faeces. A characteristic of this mould is the violent discharge of the whole sporangium, containing the ripe spores, on to the neighbouring herbage reaching a height of 6 feet and a distance of $8\frac{1}{2}$ feet in still air. The larvae appeared to be stimulated to climb up the sporangiophores of the fungus, coming to rest on the upper surface of the sporangium.

Immunology.

Many workers have reported that calves' which had been infected by D. viviparus acquired strong resistance to reinfection of D. viviparus (Taylor, 1951; Gregoire, 1951; Jarrett et al., 1955a; Swanson et al., 1956 and Michel, 1955). Jarrett et al. (1959) recovered a great variation in immunity after exposure to D.viviparus. In resistant animals, the larvae may fail to become established in the lung, or may be inhibited in development or eliminated or destroyed before attaining maturity (Michel, 1956). The duration of resistance seemed to decline very rapidly in the absence of reinfection (Rubin & Lucker, 1956; Weber, 1958; Jarrett et al., 1955b).

Inspired by these findings (immunity) a lot of promising research is going on in the line of vaccination (Wade et al., 1962; Jarrett et al , 1960a, Jarrett, 1958a, Engelbrecht, 1961; Edds, 1963; Olsen, 1962; Vercruysse et al., 1963; Pierre et al., 1961; Enigk g Düwel, 1963a,b Düwel, 1963; Cornwell, 1962a; Downey, 1965; Lucker & Vegors, 1960; Nelson, 1964, and Lucker et al., 1964),

Pathogenesis.

The susceptible animals are infected by ingestion of contaminated grass, recently cut wet roughage or contaminated water. The response of the lungs depends

primarily on the number of larvae which are ingested, but the nutritional status, the age of the animal and debilitating factors may be involved. In studies on the development or lungworm disease, Jarrett et al (1960 b) divided it into a penetration, a prepatent, a patent and a postpatent phase. Poynter (1963) reported that during the penetration phase. (1–7 days) petechial haemorrhages were caused by the penetration of larvae from the capillaries into the alveoli. During the prepatent phase (7–25 days), there is an intense eosinophilic exudate into the lungs which block small bronchi and bronchioles. These blockages lead the collapse of parts of the lungs. In the patent phase (25 to 55 days), there is a lung reaction in which marcophages and giant cells attempt to engulf the nematode material. This produces consolidation of lung lobules. The postpatent phase (55 to 70 days) of the disease is often one of gradual recovery. There is alveolar epithelialization which may spread to involve the whole lobe of the lung. Jarrett et al (1960b) listed several complications which might be seen; e.g. pulmonary oedema, emphysema, bronchiectasia and pulmonary fibrosis. Jarrett & Sharp reported that the eosinophilic response appeared more marked. According to the severity of clinical diseases it is generally divided into acute and chronic forms.

In the acute form there are areas of collapse of all lobes, septal oedema, interstitial emphysema and accumulation of eosinophils. These lesions are followed by alveo—epithelialization, oedema and formation of a hyaline membrane. Severe dyspnoea and cough are commonly seen. Frothy fluid containing larvae deposits in the bronchi and the animals often die before the mature worms have developed (Jarrett et al., 1959)

In the chronic form the adult worms and larvae can be found in bronchi which are also filled with pus and mucus. The worms live in the small bronchi

where they suck blood and irritate the mucosa, roducing a catarrhal bronchitis. The inflammatory process spreads to the surrounding peribronchial tissues, and exudate usually passes back into the bronchioles and alveoli, causing atelectasis, and pneumonia may be set up by secondary invaded bacteria. The irritation of the mucosa by the penetrating larvae may cause diarrhoea.

Clinical findings.

The acute form of lungworm infestation is usually found in adult cattle. Clinical signs are not seen in the first week after infection. The first clinical sign may be diarrhoea followed by the onset of respiratory signs (Djafar et al., 1960), other clinical signs may develop by the end of the Second week or during 7 to 25 days. There will often be a rise in temperature 104 to 105°F, dry cough slight nasal discharge, shallow bronchial breathing at the rate of up to 100 per minute (Blood & Henderson,1963). On auscultation, the lung sounds will show an increased vesicular murmur and bronchial tones over the whole of the lung field. In severe cases, mouth breathing, a violent respiratory heave and grunt. cyanosis and recumbency may be found. The evidence of consolidation can be detected by loud bronchial sounds and absence of vesicular murmur, moist rales may be heard over the bronchial tree and cracking of interstitial emphysema often appears over the dorsal two-thirds of the lung.

The chronic form is more common in calves than the acute form. Diarrhoea again may be the first sign of the onset followed by slightly elevated temperature, dry sough, an increase in the respiratory rate of about 60 to 80 per minute (Fisher & McIntyre, 1960). In severe vases the respiratory rate may reach 1lo to 140 per minute

(Parker & Vallely, 1960). In later stages shallow bronchial breathing accompanied by an expiratory grunt is observed. The head will often be extended, the tongue protruded and expiration vigorous with the whole body concerted in the effort. On auscultation there will be consolidation and bronchitis ventrally, and emphysema dorsally. The course of the disease is quite long, it my be 3 to 4 weeks or 7 to 8 weeks. The animals are susceptible to secondary bacterial bronchopneumonia and many animals die, the lung lesions showing proliferative bronchopneumonia.

In both forms, emaciation, loss of appetite, dehydration, congestion of mucus membranes, loss of weight and reduction in growth are usual findings.

Clinical pathology.

The larvae are present in faeces 12 days after the development of the clinical signs or 24 days after infestation. The faecal examination can be performed by the Baerman method or by the improved method of Henriksen (1965); The faecal sample is placed on a double layer of gauze suspended on the rim of a conical glass vessel with water. After 24 hours a drop of fluid is removed from the bottom of the vessel by pipette and examined under microscope. By this method twice as many cases of lungworm infestation can be detected as by the Baerman method.

Haematologically, calves suffering from D. viviparus infestation shows eosinophils in the circulating blood (Weber & Rubin, 1958; Djafar et al., 1960, Cornwell, 1962b, and Downey, 1965). Djafar et al. (1960) found that no significant changes were recorded in the total number of erythrocytes, haemoglobin, haematocrit values, immature neutrophils, basophils, monocytes or alpha globulin. In studies of the duration of acquired immunity to D. viviparus infection in calves, Michel & Mackenzie (1965) reported that the general blood picture in resistant and control animals

were alike except for the differentila count. The circualting blood eosinophils showed a characteristic primary response in animals given 2 successive doses of normal larvae and a mild transient rise in thoes that were vaccinated. The blood eosinophils was paralleled by dense infiltration of tissues by eosinophils.

Many workers have studied the serum antibody formation against D. viviparus by complement—fixation test, but it cannot so far be relied upon to reflect the immune status of agiven calf (Jarrett et al., 1959; Weber, 1958; Michel & Cornwell, 1959; Cornwell & Michel, 1960; Poynter et al., 1960; Cornwell, 1960a,b, 1961. 1963a). Michel, Mackenzie et al. (1965) reported that complement fixation antibodies were not found in animals after vaccination. They also performed precipitin tests in which it was shown that several precipitins were produced in response to infection, though not to vaccination.

In studies of the estimation of complement—fixing antibodies in bovine dictyaulosis by means of an antigen prepared from A. suum, Tomanek & Prochazka (1967) reported that the c. f. titres increased within 3 weeks after infection.

In studies of the allergic diagnosis of dictyaulosis in cattle, Pagirys et al. (1966) claimed that specific reactions were obtained in 409 infected cattle (aged 4—9 months) following i/d injection of 0.2 ml of allergen, either 1:1000 dilution Bivin extract or a 1:100 dilution of extract prepared by Ershov's method. Positive reactions were first obtained 10—12 days after infection with D. viviparus larvae.

Biopsy of the lungs has been used to determine the presence of larvae (Campbell & wetherill, 1957).

Diagnosis.

The diagnosis may be based on a history of exposure to pasture previously grazed by animals of the same species, the presence of the disease in the area, failure

to treatment for bacterial and viral pneumonia and the time of the outbreak. The presence of fever, cough, consolidation of the lungs and interstitial amphysema are undependable criteria because these signs are found in bacterial and viral pneumonia as well. One clinical sign which may be of value in differentiation this disease form other types of pneumonia is a harsh and dry cough, The presence of larvae in the faeces, the increased respiratory rate and eosinophils in circulating blood and lung biopsy support the diagnosis.

Summary

Bronchopneumonia is commonly seen in cattle. The disease is mainly caused by bacteria, viruses, fungi aad parasites. The intensive husbandry, physical stresses, pasture management, grazing behaviour and climatic conditions seem to be of major importance. The infections are characterized by a specific respiratory syndrome. The specific diagnosis of infection caused by organisms is based on the isolation of organisms and serological findings. In parasitic infection haematological findings and the presence of larvae in the faeces provide specific means of diagnosis.

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สาระสรุป

Bronchopneumonia เป็นโรคที่พบบ่อยในโค สาเหตุของโรคนี้ส่วนใหญ่เกิดจากแบคทีเรีย, ไวรัส, เชื้อรา และพาราไซต์ การจัดให้สัตว์อยู่รวมกันอย่างหนาแน่น ความเครียดทางสรีระ การจัดแปลงหญ้าสำหรับสัตว์, ลักษณะการเล็มหญ้าของสัตว์ และสภาพของดินฟ้าอากาศ ก็เป็นเรื่องสำคัญเหมือนกัน ลักษณะของโรคจะพบว่ามีกลุ่มอาการโดยเฉพาะเกี่ยวกับระบบหายใจ การวิเคราะห์โดยเฉพาะของโรคที่มีสาเหตุจากเชื้อจุลินทรีย์กระทำโดยการแยกเชื้อ และการตรวจหาทางเซรุ่ม ในกรณีที่โรคนี้เกิดจากพาราไซต์ การตรวจก็ใช้วิธีการตรวจโลหิต และการตรวจหาตัวอ่อนในอุจจาระของสัตว์