Bienn. Symp. North. Wild Sheep
and Goat Counc. 3: 163-172

AN OVERVIEW OF THE CLINICAL SIGNS, GROSS AND HISTOLOGICAL LESIONS OF THE PNEUMONIA COMPLEX OF BIGHORN SHEEP

T. R. Spraker, Department of Pathology, Colorado State University, Fort Collins, Colorado

C. P. Hibler, Department of Pathology, Colorado State Univesity, Fort Collins, Colorado

ABSTRACT

Bronchopneumonia is a commonly diagnosed disease in both captive and free ranging bighorn sheep. Three categories of mortality are recognized in bighorn sheep: the classical all-age die-offs and two types of summer-lamb mortality. The all-age die-offs and the summer-lamb mortality following all-age die-offs appear to be induced by a variety of stress factors where as the verminous types of summer-lamb mortality appears to be induced by transplacental transmission of numerous parasites (Protostrongylus stilesi). Herein is described the clinical features, gross and histological lesions found in these types of bronchopneumonia in bighorn sheep.

INTRODUCTION

Die-offs in bighorn sheep have been documented from the late 1880's to the present. Some of the reasons for the decline in populations have included market hunting (Shields, 1890; Grinnell, 1928; Seton, 1929; Honess, 1942; Packard, 1946; Buechner, 1960), loss of winter range, (Honess, 1942; Packard, 1946;) and diseases (Baillie-Grohman, 1882; Marsh, 1938; Honess, 1942). Some of the specific diseases that have been diagnosed as causing die-offs include mange or scab (Baillie-Groham, 1882; Seton 1929; Bailey, 1936; Wright, 1933; Spencer, 1943; Jones, 1950; Lange, 1980), anthrax (Grinnel, 1904, 1928) and pneumonia (Rush, 1927; Marsh, 1938; Potts, 1937; Honess, 1942; Packard, 1946). At the present time a major cause of mortality in bighorn sheep populations appears to be pneumonia (Beuchner, 1960; Spraker, 1979; Foreyt, 1982).

Currently the authors recognize two different field manifestations of mortality of bighorn sheep. The first is the classical all-age die-off that usually occurs in winter but can occur at any time of the year and has been seen under range conditions as well as in captivity (Rush, 1927; Marsh, 1938; Honess, 1942; De Martini, 1977; Spraker, 1977; Foreyt, 1982). Generally this is a bacterial and/or verminous pneumonia. The second type of mortality is referred to as summer-lamb mortality. This summer-lamb mortality usually occurs in July-September and affects only lambs. There appers to be two different types of summer lamb mortality, one in which lungworm plays a primary role and the other where lungworm does not play a primary role (Spraker, 1979 and unpublished data). This second clinical type of lamb mortality appears to be stress related. The purpose of this overview is to describe these three basic types of pneumonia, hopefully, to help wildlife biologists better evaluate the causes of mortality in bighorn sheep herds.

SUMMER-LAMB MORTALITY

Mortality of lambs during the summer months was first noted by Norton in 1933 in the National Bison Range of western Montana and two of these lambs were examined at necropsy by Marsh (1938). Others have also observed a summer lamb mortality in which pneumonia was the cause of death (Honess, 1942; Packard, 1946; Honess, 1955; Buechner, 1960, Woodward, 1970). Research into the summer lamb mortality has shown two clinical types of pneumonia: verminous pneumonia and a stress related pneumonia which follows a previous all-age die-off (Spraker, 1977, 1979, unpublished data).

VERMINOUS TYPE

Verminous pneumonia of bighorn lambs is usually first observed in late July or early August and occurs in lambs 1 to 5 months of age. The clinical signs in these lambs include weight loss, loss of luster of their hair coat which progresses to a dull yellow, violent paroxysmal coughing, and lagging behind in the herd. Some of the lambs fail to shed their lamb pelage, whereas other lambs do shed this rough lamb pelage and have a fairly slick appearing hair coat when they die.

Gross pathological changes are found in the integumentary, cardiovascular, respiratory and lymphopoietic systems. Lambs 6 to 8 weeks of age have a subtle loss of sheen of their hair coat, which becomes more apparent in older lambs. Lambs 8 to 12 weeks of age usually have a dull, yellow, rough, hair coat. Some lambs with pneumonia shed this rough, yellow, pelage and die with a fairly normal appearing pelage. Some lambs are in good condition with an abundance of abdominal and subcutaneous fat, but most lambs are in poor condition when they die.

The mucosa of the nasal turbinates and septum, ethymoidal labyrinths, trachea, and main stem and primary bronchi are usually reddened and covered by small amounts of white mucoid exudate. The lungs partially collapse. Several large, gray-white, firm raised nodules (lungworm nodules) are

located in the posterior dorsal aspects of both diaphragmatic lobes of the lung. The cut surfaces of these nodules are gray-to-white and have an irregular appearance. The freshly cut edges slightly bulge. Usually a small amount of white, mucoid exudate can be expressed from cut bronchioles within these nodules.

The anteroventral aspects of the lungs are dark red to lavender and firm. The pleura covering the consolidated lobes is thin, most, and shiny in most cases. The entire anteroventral aspect of the lungs in some cases is covered with a thick (2mm) layer of yellow, friable material (fibrin). Adhesions to the diaphragm and the costal pleura are present between these consolidated lobes.

Usually there is a sharp line of demarcation between the consolidated areas and the normal lung. Emphysema near this line of demarcation often can be found. The consolidated areas are actually level with or slightly depressed from the normal lung tissue. The subpleural pulmonary parenchyma and the cut surface are dark red-to lavender and have a "cobble-stone" appearance. This is due to many small cloverleaf-shaped, gray, raised areas surrounded by red, collapsed parenchyma. This "cobblestone" appearnace is subtle in acute cases of pneumonia, but becomes more prominent in chronic cases. A small amount of a white mucoid exudate can be expressed from cut bronchioles in these consolidated areas. Necrosis is uncommonly found in the consolidated areas. Chronic cases can have small (.5cm to 1cm) abscesses in the consolidated, pulmonary parenchyma. Small, fibrous adhesions often occur between the consolidated lungs and the costal pleura in chronic cases. Few lambs have fibrinous adhesions between the pericardial sac and the medial aspects of the lungs (fibrinous pericarditis). Fibrinous epicarditis is found occasinally.

The spleen is normal. The retropharyngeal, hilar, and mediastinal lymph nodes are enlarged and the cut surfaces are wet. The cortical areas of the lymph nodes are hyperplastic. The thymus is of normal mass and extends from the base of the heart to the mid-cervical region.

The histological lesions are predominantly within the respiratory and lymphoid systems. The epithelium of the nasal cavity, trachea, and bronchi usually contain areas of hyperplasia and atrophy. Bronchioles and respiratory bronchioles are usually cuffed by a mild-to-marked number of lymphoid cells and have mild to marked hyperplastic and hypertrophied epthelium. The severity of these lesions usually correlates with chronicity. The lumens of these air passages usually contain exudate and, occasionally, a first-stage, protostrongylid larva. The alveoli usually are partially collapsed and contain a mixture of macrophages and neutrophils. A small amount of fibrin and edema can sometimes be found the alveoli. Alveoli nearest to the alveolar ducts usually contian more neutophils. Degenerate and live, first-stage, protostrongylid larvae can often be found surrounded by macrophages within alveoli of the anteroventral aspects of the lungs.

Often times the pleura of the lung is coated with fibrin and, in chronic cases, granulation tissue. Lungworm nodules located in the posterior dorsal aspects of the diaphragmatic lobes are characterlized by numerous viable parasites (Protostrongylus stilesi) surrounded by either lymphocytes, plasma cells, macrophages, with a few eosinophils and multinucleated giant cells. In older lambs the lungworm nodules are usually composed of predominantly multinucleated giant cells and dead parasites.

Microbiological agenta have been isolated from the upper and lower respiratory system of the lambs with pneumonia. Viral agents include Parainfluenze type 3; mycoplasma agents include Mycoplasma arginini; bacterial agents include Pasterurella multocida, P. hemolytica, Neisseria sp., Hemophilis ovis, Corynebacterium pyogenes, Streptococcus sp., Staphylococcus sp. and a Mima sp.; and parasitic agents include P. stilesi.

The pathogenesis of this "verminous" variety of bronchopneumonia has been studied. The progession of events leading to pneumonia in these lambs correlates well with the activity of a Protostrongylus larvae crossing the placenta of the adult ewe and entering the fetal liver in the latter stages of pregnancy. These third-stage larvae remain in the liver until parturition. Following birth of the lamb, the larvae then migrate to the lungs of the newborn. The third-stage larvae lodge in small pulmonary arterioles, enter the lung parenchyma, and migrate to the dorsal posterior aspects of the diaphragmatic lobes. Development of the parasites begins simultaneously with birth of the lamb; therefore, the activity and maturation of the parasites correlate well with the age of the lamb. The parasites mature when lambs are 3.5 to 4 weeks of age and begin to produce thousands of ova which mature to motile first-stage larvae in about one week. Numerous first-stage larvae are aspirated in to the anteroventral aspects of the lungs predisposing lambs to subacute to chronic suppurative bronchopneumonia complicated by viral, mycoplasmal, and bacterial agents of low pathogenicity. (Hibler 1972, 1974; Spraker 1977, 1979).

STRESS RELATED TYPE

This stress related type of pneumonia has been observed in 5 or 6 cases and have included lambs born in captivity, and in lambs born in the wild following a previous all-age die-off. The clinical signs, gross and histopatholobical changes of the lambs are similar to the verminous pneumonia except that the thymus is atrophied whereas it is normal in the verminous pneumonia lambs. The gross and histological lesions in this stress related type of pneumonia can be more acute than the verminous pneumonia. Fewer lambs have been studied in this group but of these studied the agents isolated include: Pasteurella multocida, P. hemolytica, Corynebacterium pyogenes, Neisseria sp., Hemophilis ovis, Streptococcus sp., Staphylococcus sp., Mima sp., Herella sp., and immature protostrongylid lungworms. Thus far, mycoplasma and viral agents have not been isolated from these lambs. The pathogenesis of this stress related pneumonia is believed to be similar to the pathogenesis of the pneumonia in the all-age die-offs.

ALL-AGE DIE-OFFS

All-age die-offs were first described by Rush and Fisher (Rush, 1927) and Marsh (1938) and has been documented by others (Buechner, 1960; Griner, 1974; Foreyt, 1982; Davison, 1982). This type of die-off has been documented in both captive (Spraker, 1977, DeMartini, 1977) and free ranging sheep. The pathogenesis of this disease process is believed to be induced by stress.

Clinical signs in sheep during an all-age die-off may vary from acute death to chronic pulmonary cripples. Animals demonstrate a moderate degree of coughing. Some lose weight and die in an emaciated condition, whereas others die acutely and are in an excellent body condition. This disease condition affects all ages and usually occurs in the winter months but can occur in the warmer months especially in captive herds.

The main gross lesion found in animals dying during an all-age die-off is a bronchopneumonia. Animals that die acutely will usually be in excellent body condition. The nasal cavity, trachea and bronchi are usually slightly reddened and contain a small degree of white mucoid exudate in acute cases. The anteroventral or sometimes only the diaphragmatic lobes of the lung are dark red, firm, and slightly raised from the interface with unaffected pulmonary parenchyma. The pneumonic lung is firm and a moderate degree of exudate can be expressed from severed bronchioles. Firm, yellow-grey, raised, lungworm nodules are located in the posterior, dorsal aspects of the diaphragmatic lobes and the lungworm burden varies from light to heavy. Usually the surface of the lung is coated with a thin coat of fibrin which allows the lung to be easily separated from the costal pleura. The mediastinal, tracheal, pharyngeal, and prescapular lymph nodes and the lymphoid tissue located in the posterior aspects of the nasal septum are mildly enlarged in these acute cases.

The animals with the subacute-to-chronic form of this pneumonia have similar gross lesions except they are of longer duration. There is mild-to-severe weight loss and, even, emaciation in some animals. The fibrinous adhesions are now fibrous. The parenchymal and subpleural surfaces have a classical "cobble-stone" appearance, with small, pale, cloverleaf-shaped foci surrounded by a slightly depressed, red-grey parenchyma. Small abscesses are common within the lung parenchyma. Lymphoid tissues are usually hyperplastic and the thymus is atrophied. The hair coat is usually dull and rough. A mild to moderate degree of adrenal cortical hyperplasia is often found.

The histopathological lesions found in this type of pneumonia are typical of a bronchopneumonia. Acute cases are characterized by mild rhinitis, tracheitis and bronchitis. There is a mild degree of hyperplasia of bronchiolr epithelium. Exudate is often found in bronchioles. Alveoli are usually open and filled with edema, neuthrophils, and fibrin admixed with a few macrophages. Fibrin is often on the surfac of the pleura. A

few lungworm larvae are often in the anteroventral aspects of the lungs. The lungworm nodules contain adult male and female parasites, eggs and larvae. These parasites usually are surrounded by lymphoid cells, macrophages, plasma cells with a few neutrophils and eosinophils in healthy sheep; however, with this acute pneumonia there is often a severe suppurative bronchitis and bronchiolitis within lungworm nodules. Thus, in some cases, the lungworm nodule histologically appears to be similar to the ventral consolidated portions.

Chronic cases are similar to acute cases except for more atrophy of tracheal epithelium, and hyperplasia of bronchiolar and alveolar duct epithelium. Air passages are often cuffed by lymphoid cells and plasma cells. Fibrosis surrounds bronchioles in some areas. Exudate (mostly neutrophils and desquamated epithelial cells) usually fills the lumen of bronchioles. Alveolar ducts are often filled with neutrophils. The alveoli are usually partially collapsed and contain macrophages with an admixture of neutrophils. A few degenerate or live lungworm protostrongylid larvae sometimes can be observed in the anteroventral aspects of the lungs. The pleura is often covered with granulation tissue and overlayed by fibrin admixed with neutrophils and macrophages. Fibrous tags and adhesions and micro-to-macro abscesses are often found. The cellular reactions within lungworm nodules are similar to the nodules described with the acute cases. Mediastinal and bronchial lymph nodes are often hyperplastic and sometimes contain first-stage lungworm larvae. The thymus is atrophied and the adrenal cortex is slightly-to-moderately hyperplastic.

Etiological agents thus far isolated from sheep with the acute to chronic, fibrinopurulent bronchopneumonia include Pasteurella multocida, P. hemolytica, Neisseria sp., Corynebacterium pyogenes, Streptococcus sp., Staphylococcus sp., Parainfluenza type-3 (isolated from one die-off of captive sheep (Parks, 1972;), Protostrongylus stilesi and P. rushi. No mycoplasma or chlamydia have been isolated to date.

The pathogenesis of this fibrinopurulent bronchopneumonia that occurs in the all-age die-offs appears to be stress induced. Animals normally carry bacterial agents of low pathogenicity in their upper respiratory system and have low to heavy lungworm burdens. These sheep become stressed in some means such as encroachment by man or his domestic animals, loss of range, crowding, change of range conditions, lack of water, inclement weather, deep snow, malnutrition, captivity, dust, etc. This "stress" leads to an adrenal cortical hyperfunction and elevated serum cortisol. Increased cortisol causes the animal to be less resistant to the microbiological agents of low pathogenicity already within the animal. This either allows the bacteria to proliferate and invade the depths of the lungs or inhibits the body's mechanisms of removing bacteria that are inhaled into the depths of the lungs. The bacteria then proliferate within small bronchioles and induce inflammation. If the bacteria multiply rapidly and are moderately pathogenic, such as with Pasteurella, the animal may die quickly, or if the bacteria multiply more slowly a subacute to chronic bronchopneumonia will develop. Heavy burdens of lungworm can

initiate this bronchopneumonia as with the verminous pneumonia of lambs, (Spraker, 1977, 1979) but in many cases the lungworms, as with the bacteria, are on of the low pathogenic agents that are allowed to proliferate under stress conditions.

SUMMARY

Bronchopneumonia is a common disease in bighorn sheep, even though there are several field manifestations of pneumonias, i.e., two types of summer-lamb mortality, and the classical pneumonia associated with all-age die-offs. The basic clinical signs, gross and histopathology are fairly similar in these three different field manifestations of pneumonias. The agents isolated from those cases are similar also: lungworms, bacteria and sometimes viruses and mycoplasma. A combination of stress factors appears to play a vital role in all-age die-offs, mortality in captive lambs, and summer-lamb mortality following a previous all-age die-off. Transplacental transmission of heavy lungworm burdens with maturation of the parasites when the lambs are 3.5 to 4.5 weeks of age appears to be the most important predisposing factor for the verminous type of summer-lamb mortality. Ultimately, and regardless of the predisposing factors, many bighorn sheep die due to an acute to chronic fibrinopurulent bronchopneumonia.

LITERATURE CITED

Bailey, V. 1936. The mammals and life zones of Oregon. N. Am. Fauna. 55:1-416, p 64-70.

Baillie-Grohman, Wm. A. 1882. Camps in the Rockies. Chas. Scribner's Sons. New York, N. Y. p 32.

Buechner, H. K. 1960. The bighorn sheep in the United States, its past, present and future. Wildl. Soc., Wildlife Monogr. 4. 1-174.

Davison, P. 1982. Personal communications.

DeMartini, J. C. and R. B. Davis. 1977. An Epizootic of pneumonia in captive bighorn sheep infected with Muellerius sp. J. Wildl. Dis. 13(2): 117-124.

Foreyt, W. J. and D. A. Jessup. 1982. Fatal pneumonia of bighorn sheep following association with domestic sheep. J. Wildl. Dis. 18(2): 163-168.

Grinnell, G. B. (ed). 1904. American Big gAme in its Haunts. New York. Forest and Stream Publishing Co. p 497.

Hibler, C. P., R. E. Lange and C. J. Metzger. 1972. Transplacental transmission of Protostrongylus spp. in bighorn sheep. J. Wildl. Dis. 8:389.

Hibler, C. P. and C. J. Metzger, T. R. Spraker, and R. E. Lange. 1974. Further observations on Protostrongylus sp. infection by transplacental transmission in bighorn sheep. J. Wildl. Dis. 10:39-41.

Honess, R. F. 1942. Lungworms of domestic sheep and bighorn sheep in Wyoming. Bull. No. 255. Agriculture Exp. Station, University of Wyoming. Laramie, Wyo. 1-24.

Jones, F. L. 1950. A survey of the Sierra Nevada Bighorn. Sierra Club Bull. 35(6):29-76.

Lange, R. E., A. V. Sandoval, and W. P. Meleney. 1980. Psoroptic scabies in bighorn sheep (Ovis canadensis mexicana) in New Mexico. J. Wildl. Dis. 16: 77-82.

Marsh, H. 1938. Pneumonia in Rocky Mountain Bighorn Sheep. J. Mammal. 19(2) 214-219.

Packard, F. M. 1946. An ecological study of the bighorn sheep in Rocky Mountain National Park, Colo. J. Mammal. 27(1): 3-28.

Parks, J. B., G. Post, E. T. Thorne. 1972. Isolation of Parainfluenza virus from Rocky Mountain Bighorn Sheep. JAVMA. 161(6): 669-672.

Potts, M. K. 1937. Hemmorrhagic septicemia in the bighorn of Rocky Mountain National Park. J. Mammal. 18:105-106.

Rush, W. M. 1927. Notes on disease in wild game mammals. J. Mammal. 8(2):163-165.

Seton, E. T. 1929. Lives of Game Animals. New York. Doubleday Page and Co. Vol. III. 513-573.

Shields, G. O. 1890. The Rocky Mountain Sheep. In: The Big Game of North America. ed. by G. O. Shields. Rand-McNally and Company. Chicago and New York. 363-389.

Spraker, T. R. and C. P. Hibler. 1977. Summer lamb mortality of Rocky Mountain bighorn sheep. Desert Bighorn Council Transactions. p 11-12.

Spraker, T. R. 1977. Fibrinous pneumonia of bighorn sheep. Desert Bighorn Council Tansactions. p 17-18.

Spraker, T. R. 1979. The pathogenesis of pulmonary protostrongylosis in bighorn lambs. Ph.D. thesis. Colorado State University, 1-233.

Spencer, C. C. 1943. Notes of the life history of Rocky Mountain bighorn sheep in the Tarryall Mountains of Colorado. J. Mammal. 24(1):1-11.

Wright, G. M., J. J. Dixon, and B. H. Thompson. 1933. A preliminary survey of faunal relations in National Parks. Washington: Government Printing Office. U. S. National Park Service, Fauna Series 1. p 157.

Q. I have some questions that deal with ecological patterns more than anything else. To begin with, where do you find this pneumonia occuring primarily and do you find it with low-elevations or high-elevation populations?

ANS. We've seen the pneumonia in low- and high-elevation populations in Colorado.

Q. May I suggest that these pneumonias will become increasingly less frequent as you go to higher altitudes or higher latitudes.

ANS. If you ask the question about evolution, you must get into some detail. The mountain sheep is basically a relatively recent immigrant to this continent. It has been here probably about 45,000 years, which is a pittance. In these latitudes it is even more recent. As far as altitudes are concerned, it probably hasn't been at the low altitudes for a very long time either. It's like the elk, like the grizzly, like the wolf: spread basically after the megafaunal extinctions. For this reason you'd expect that northern animals adapted to high altitudes and high latitudes, adapted to glacier environments, will have terrible troubles if they get into contact with other environments which are notoriously rich in their biota and notoriously rich in pathogens.

So the behavior of the bighorn sheep in the southern latitudes here in the United States is fully expected to be one of a troubled species in relation to what it is in the northern environment where it is highly adapted. You don't have to go to bighorn sheep, you can go to human beings. If you look at the North American Indian and how the American Indian does when they come into contact, particularly in South America, with really adapted people, people adapted to the tropics, the negros, the South American Indians do notoriously very poorly. They are full of disease. Their reproductive rates decline, native tribes go down the drain literally because the African, by in large, is very well, in a biological since, adapted to tropical environments and South American Indians are not. They are very recent immigrants to that land.

You should find for instance, that if you take an old emigrant, horses or burros, and put them into competition with bighorn sheep, it's hands down that sheep will lose in southern environments. The reason: the horse is an old American genus which still should carry, in its geno type messages from ages passed which make it perfectly adapted to the conditions it finds here. So if you approach the question of, have the bighorn sheep adapted to these many pathogens, remember it is facing very different environmental conditions today than it did 50,000, 200,000 or a million years ago.

ANS. This may be so, but some of the first die-offs of sheep were described from Montana and some of the most wide-spread and devastating die-offs occurred in Canada. I think that die-offs, occur at both low- and high elevations.