

BACTERIAL DISEASES

Colibacillosis (Coliform infections)

Problems attributed to coliform infections are often caused by strains of the *Escherichia coli* organism. There is a marked variation in severity. Problems range from severe acute infections with sudden and high mortality to mild infections of a chronic nature with low morbidity and mortality. Infections may result in a respiratory disease from air sac infection, a septicemic (blood) disease from generalized infections, an enteritis from intestinal infection or a combination of any or all of these conditions. The disease may result from a coliform infection alone as in primary infection or in combination with other disease agents as a complicating or secondary infection. Secondary infections commonly occur as a part of the classic air sac disease syndrome as a complication with *Mycoplasma gallisepticum* infections.

All ages can be affected; however, the acute septicemia in young turkeys and airsacculitis in young chickens is more common in young growing birds. High, early mortality may occur as the result of navel infections.

The symptoms of this disease is caused by the *E. coli* bacteria and the toxins produced as they grow and multiply. There are many different strains or serological types within the group of *E. coli* bacteria. Many are normal inhabitants in intestinal tracts of chickens and turkeys and consequently are common organisms in the birds' environment.

A marked variation exists between different strains in their ability to cause disease. Some are severe and by themselves can cause disease while others are supposedly harmless. All degrees of pathogenicity exist between the two extremes.

The primary routes of invasion by the organism are the respiratory system and the gastrointestinal tract. Omphalitis and infections in young birds may result from entry through the unhealed navel or penetration of the egg shell prior to or during incubation.

The symptoms vary with the different types of infections. In the acute septicemic form, mortality may begin suddenly and progress rapidly. Morbidity may not be

apparent and birds in apparently good condition may die. However, in most cases birds are listless with ruffled feathers and indications of fever. Additional symptoms of labored breathing, occasional coughing and rales may be apparent. Diarrhea may be evident. Mortality may be high in recently hatched chicks and poults as a result of navel infection of coliforms.

Extremely acute septicemic infection may result in sudden death with very few, if any, lesions apparent. Common lesions include dehydration, swelling and congestion of the liver and spleen and kidneys and pinpoint hemorrhages in the viscera. Fibrinous to caseous exudate in the air sacs, heart sac and on the surface of the heart, liver and lungs is a characteristic lesion. The intestines may be thickened and inflamed and may contain excess mucus and areas of hemorrhage. Navel infections, similar to those described for omphalitis may be seen in young birds.

Diagnosis by laboratory means is necessary since coliform infection in its various forms may resemble and be easily confused with many other diseases. Isolation and identification of the organism by culture procedures can be accomplished relatively quickly; however, mere isolation is not sufficient to make a diagnosis. One must take into consideration the organ from which the organisms were isolated, the pathogenicity of the particular isolate and the presence of other disease agents.

Management and sanitation practices designed to reduce the number of these types of organisms in the birds' environment are necessary. In addition, reducing stress factors and other disease agents can enhance the ability of birds to defend against harmful infections. Providing adequate ventilation, good litter and range conditions, properly cleaned and disinfected equipment and facilities and high quality feed and water will improve the disease resisting status of the birds. The poultryman must always avoid overcrowding, environmental stresses like chilling or overheating and avoid vaccination or handling stress during periods when the birds are already subjected to stressful conditions. Proper egg handling, good hatchery management and implementing a good sanitation program is necessary to reduce early exposure of chicks or poults to disease organisms. It is always emphasized that problems due to one of the more pathogenic strains may occur even under ideal conditions.

The response of coliform infections to various medications is erratic and often difficult to evaluate. Under practical conditions, treatment is often disappointing. Drug sensitivity varies with the strain of *E. coli* causing the condition. Laboratory tests to determine the sensitivity to the various drugs are useful to select the most beneficial drugs. When practical, moving birds to a clean environment may be of more value than medication. For example, when outbreaks occur in growing turkeys in the brooder house, moving to range is often the best treatment.

Mycoplasmosis (CRD, Air sac, Sinusitis)

Organisms in the genus *Mycoplasma* are a significant cause of respiratory disease in poultry. Of the numerous species of *Mycoplasma* that have been isolated from domestic poultry, three are of known significance. *Mycoplasma gallisepticum* is associated with chronic respiratory disease (CRD)/air sac syndrome in chickens and turkeys and infectious sinusitis of turkeys; *Mycoplasma meleagridis* is associated with airsacculitis in turkeys; and *Mycoplasma synoviae* is the cause of infectious synovitis in chickens and turkeys.

Chronic respiratory disease (CRD), air sac syndrome and infectious sinusitis of turkeys have a common cause. CRD was first recognized as a chronic but mild respiratory disease of adult chickens. It reduced egg production but caused little or no mortality. Afterward, a condition known as "air sac disease" became a problem in young birds. It caused high mortality in some flocks. Many birds became stunted, feed efficiency was reduced, and many fowl were rejected as unfit for human consumption when processed.

Infectious sinusitis in turkeys produces a sinus swelling under the eye as well as an inflammation of respiratory organs. It is a chronic disease adversely affecting growth and feed conversion. It may also cause significant mortality in young poults.

A peculiar bacterial-like organism known as *Mycoplasma gallisepticum* (MG) is common to all three conditions. CRD and sinusitis in turkeys are caused by a pure MG infections while the air sac syndrome is caused by an infection of MG in combination with *E. coli*. These conditions are triggered by acute respiratory infections such as Newcastle disease or infectious bronchitis.

Mycoplasma gallisepticum is widespread and affects many species of birds. Eradication programs have reduced the incidence in recent years. It is primarily spread through the egg. Infected hens transmit organisms and the chick or poult is infected when it hatches. Organisms may also be transmitted by direct contact with infected or carrier birds.

The true CRD produces slight respiratory symptoms such as coughing, sneezing and a nasal discharge. In the air sac syndrome there is an extensive involvement of the entire respiratory system. The air sacs are often cloudy and contain large amounts of exudate. Affected birds become droopy, feed consumption decreases and there is a rapid loss of body weight.

Infectious sinusitis in turkeys occurs in two forms. When the "upper" form is present, there is only a swelling of the sinus under the eye. In the "lower" form, the lungs and air sacs are involved. The air sacs become cloudy and may contain large amounts of exudate. Both forms of the disease are usually present in the flock and frequently are present in the same bird.

Diagnosis of either condition must be based on flock history, symptoms and lesions. Blood tests are useful in determining whether a flock is infected.

The answer to the MG problem in both chickens and turkeys is eradication of the disease organisms. This goal has been achieved in commercial breeding flocks with voluntary programs conducted by the National Poultry Improvement Plan (NPIP) and National Turkey Improvement Plan (NTIP). The treatment of CRD, air sac syndrome and the lower form of infectious sinusitis is not considered satisfactory. Many antibiotics have been used with varying success. Whether to give treatment is a decision that must be made on each flock based on economic factors. If treatment is attempted, give high levels of one of the broad spectrum antibiotics (Tylosin, aureomycin, terramycin, gallimycin) either in feed, drinking water or by injections. The "upper"; form of infectious sinusitis can be treated with success by injecting antibiotics into the swollen sinus cavity.

Fowl Cholera

This disease occurs throughout the country wherever poultry is produced and in recent years has become the most hazardous infectious disease of turkeys. Host range is extensive and includes chickens, turkeys, pheasants, pigeons, waterfowl, sparrows and other free-flying birds.

The causative organism of fowl cholera is *Pasteurella multocida*. The organism can survive at least one month in droppings, three months in decaying carcasses and two to three months in soil. *Pasteurella* apparently enters tissues of the mouth and upper respiratory tract. The disease is not transmitted through the egg.

Major sources of infection include:

- Body excreta of diseased birds that contaminate soil, water, feed, etc.,
- Carcasses of birds that have died of the disease,
- Contaminated water supplies such as surface tanks, ponds, lakes and streams,
- Mechanical transmission by contaminated shoes or equipment.

Studies indicate that animals other than birds may serve as reservoirs of infection and actively spread the disease. These animals include raccoons, opossums, dogs, cats, pigs, and vermin.

The disease is seldom seen in chickens under four months of age, but is commonly seen in turkeys under this age. In the peracute form, symptoms may be absent; in the acute form some birds may die without showing symptoms, but many others are visibly ill before death. Characteristic symptoms include stupor, loss of appetite, rapid weight loss, lameness resulting from joint infection, swollen wattles, difficult breathing, watery yellowish or green diarrhea and cyanosis or darkening of the head and wattles.

Lesions may be lacking in birds dying during peracute outbreaks. When present, lesions may resemble those associated with any acute septicemic bacterial infection, often those of fowl typhoid. Typical lesions may include pinpoint hemorrhages in the mucous and serous membranes and/or abdominal fat; inflammation of the upper third of the small intestine; light, firm "parboiled" appearance of the liver; enlarged and congested spleen; creamy or solid collection of material in joints; and cheesy material in the internal ear and air spaces of the cranium of birds having twisted necks. Turkeys may have pneumonia with solidification of one or both lungs.

A tentative diagnosis may be made on flock history, symptoms and postmortem lesions. A definite diagnosis depends upon isolation and identification of the organism.

Properly administered bacterins are helpful in preventing fowl cholera, particularly in turkeys. Their use must be combined with a rigid program of sanitation. In general, as it applies to the use of bacterins in turkeys, complete protection is unrealistic. Follow the manufacturer's recommendations for use of the bacterin. Vaccination in conjunction with treatment is not recommended.

Sanitation practices that aid in preventing the disease are:

- Complete depopulation each year with definite breaks between older birds and their replacements,
- Implement a rodent control program,
- Dispose of dead birds properly,
- Provide safe, sanitary water,
- Clean and disinfect all houses and equipment after disposing of flock,
- Keep birds confined to the house and away from wild feral birds and animals,
- Allow contaminated ranges or yards to remain vacant for at least three months.

Although drugs usually alter the course of a fowl cholera outbreak, affected birds remain carriers and the disease has a tendency to recur when treatment is discontinued. This may necessitate prolonged treatment with drugs added to the feed and water. Sulfa drugs and broad spectrum antibiotics (Penicillin) usually control losses.

Necrotic Enteritis

Necrotic enteritis is an acute disease that produces a marked destruction of the intestinal lining of the digestive tract. Common field names (rot gut, crud and cauliflower gut) accurately describe the condition. The cause of the disease is *Clostridium perfringens*, a spore-forming, rod-shaped bacterium. Bacterial

organisms and their toxins are the primary cause but coccidiosis may be a contributing factor. Most of the damage to the intestinal lining apparently is due to toxins produced by the bacterial organisms.

Little is known about the spread of the disease but transmission is thought to occur by oral contact with the droppings from infected birds. Necrotic enteritis appears suddenly in the affected flock. Apparently healthy birds may become acutely depressed and die within hours. Mortality is usually between two and ten percent, but may be as high as thirty percent in severe outbreaks. Losses due to reduced growth and feed conversion may be more costly than flock mortality.

Lesions of the disease usually involve the lower half of the small intestine, but in some instances the entire length of the tract is involved. The intestine is dilated, contains dark offensive fluid and a diphtheritic cauliflower-like membrane that involves the mucosa. The lining of the intestine will have a coarse Turkish-towel appearance and portions of the lining may slough off and pass out with the intestinal contents. Diagnosis is based upon history, symptoms and findings of the characteristic lesions.

Bacitracin or virginiamycin are effective treatments administered in the feed. Bacitracin can also be given in the drinking water. Supportive vitamin treatment may enhance the effectiveness of the treatments. Preventive medication may be of value on premises where prior infections have been observed. Since coccidiosis may be a contributing factor, attention must be given to an effective coccidiosis control program.

Ulcerative Enteritis (Quail disease)

Ulcerative enteritis is an acute or chronic infection of game birds, chickens, turkeys and other domestic fowl. Death losses may be high for young quail or pullets being raised for egg production.

The cause of the disease is *Clostridium colinum*, a spore forming bacterial rod. The infection spreads by the droppings from sick or carrier birds to healthy birds. The disease organism is very resistant to disinfectants and will persist under varying environmental conditions.

Birds with the acute form may die suddenly while in good flesh, whereas more chronically affected birds become listless, have ruffled feathers, whitish watery diarrhea, and develop a humped-up posture. Such birds usually die in an extremely emaciated condition.

The dropping may be confused with those of birds with coccidiosis and the two diseases are often seen in the same bird. Droppings of birds with only ulcerative enteritis never contain blood.

The postmortem lesions are characteristic. The entire intestinal tract often has button-like ulcers but the lower portion is most often affected. These ulcers often perforate, resulting in local or generalized peritonitis.

Although the disease is characteristic in nature, anyone suspecting the infection should seek professional confirmation before treatment is started. Bacitracin and penicillin are the most effective drugs in the treatment and prevention of this disease. If bacitracin is used, it should be incorporated in the feed at levels up to 200 grams per ton of feed. Addition of bacitracin to the water at the rate of one teaspoon per gallon aids in controlling an outbreak of the disease. Either method of administering bacitracin will control the disease within two weeks, unless a bacitracin-resistant strain of the disease organism is present. Penicillin is also used to treat the disease if bacitracin is not effective.

Raising birds on wire is an effective preventative measure. Specific drugs (bacitracin or penicillin) fed at low levels, are effective for controlling the disease in operations where the use of wire flooring is impractical.

Pullorum Disease

Pullorum disease is an acute or chronic infectious, bacterial disease affecting primarily chickens and turkeys, but most domestic and wild fowl can be infected.

The cause is a bacterium named *Salmonella pullorum*. This organism is primarily egg transmitted, but transmission may occur by other means such as:

- Infected hen to egg, egg to chick, or chick to chick in incubator, chick box, brooder, or house. Survivors become infected breeders (cycle begins again),
- Mechanical transmission (carried around on clothes, shoes or equipment),
- Carrier birds (apparently healthy birds shed the disease organisms),
- Contaminated premises (from previous outbreaks).

Disease organisms may enter the bird through the respiratory (as in the incubator) or digestive systems. Most outbreaks of acute pullorum disease in chickens or turkeys result from infection while in the hatchery.

Pullorum disease is highly fatal to young chicks or poults, but mature birds are more resistant. Young birds may die soon after hatching without exhibiting any observable signs. Most acute outbreaks occur in birds that are under three weeks of age. Mortality in such outbreaks may approach ninety percent if untreated. Survivors are usually stunted and unthrifty. Infection in young birds may be indicated by droopiness, ruffled feathers, a chilled appearance with birds huddling near a source of heat, labored breathing, and presence of a white diarrhea with a "pasted-down" appearance around the vent. The white diarrhea symptom

instigated the term "bacillary white diarrhea" that was commonly associated with this disease at one time. Gross lesions may be lacking in some adult birds.

Diagnosis in young birds is made by isolating the causative organism in the laboratory. In older birds, blood testing may indicate an infection but a positive diagnosis depends upon isolation and identification of the organism by laboratory procedures.

Complete eradication is the only sound way to prevent pullorum disease. All hatchery supply flocks should be tested and only pullorum-free flocks used as a source of hatching eggs. Purchase chicks or poults from hatcheries that are officially recognized as "Pullorum Clean" by National Poultry Improvement Plan representatives in your state.

Treatment primarily is a salvage operation and does not prevent birds from becoming carriers. Consequently, do not keep recovered flocks for egg production. Among the drugs used to treat pullorum disease are furazolidone, gentamycin sulfate, and sulfa drugs (sulfadimethoxine, sulfamethazine, and sulfamerazine).

Fowl Typhoid

Fowl typhoid is an infectious, contagious bacterial disease that is usually acute but sometimes chronic. It affects most domestic and wild fowl including chickens, turkeys, ducks, pigeons, pheasants and other game birds. It must not be confused with typhoid fever in humans that is caused by a distinctly different organism.

The cause is the bacterium, *Salmonella gallinarum*. Methods of transmission are the same as for pullorum disease, including egg transmission. However, mechanical transmission is more prevalent with this disease than with pullorum disease.

Any age bird can be infected, but the disease primarily occurs in young adults (usually those older than twelve weeks of age). Mortality varies from less than one to about forty percent, but higher mortality has been observed. Signs include sudden or sporadic mortality, listlessness, green or yellow diarrhea (accompanied with pasting of the vent feathers), loss of appetite, increased thirst and a pale, anemic appearance of comb and wattles.

A tentative diagnosis may be made from the history, signs and lesions but final diagnosis must be based on isolation and identification of the causative organism. Lesions observed at necropsy help verify a diagnosis of fowl typhoid. The lesions include an enlarged and mottled spleen, enlarged liver (colored yellow or greenish brown), small pinpoint hemorrhages in muscles and fat surrounding internal organs, and a slimy inflammation of the front third of the small intestine. Small, white plaque-like areas visible through the walls of the intestine is suggestive of

fowl typhoid in turkeys. Blood tests used to detect pullorum reactors are also used to identify fowl typhoid birds.

Prevention and control depend heavily upon basic disease prevention practices including the hatching chicks from disease-free flocks (as determined by pullorum testing), practicing strict sanitation on the farm, providing clean feed and water, and proper disposal of all dead birds as approved by the state animal health agency. The causative organism can live outside the bird body for at least six months, thus requiring extra management precautions to break the disease cycle. Following an outbreak, thoroughly clean and disinfect the premises. When feasible, practice range rotation and other special precautions to prevent the carryover of infection to the following flock.

Drugs cannot be depended upon as a means of typhoid prevention and are not recommended for that purpose. Infected birds may be salvaged using the same drugs as used to salvage pullorum infected birds.

Botulism

Botulism is a disease caused by the ingestion of a toxin produced by the *Clostridium botulinum* bacterium. All domestic fowl and most wild birds are susceptible to the toxin's effects. Many human deaths have also been attributed to the consumption of food or water containing the toxin.

Botulism is not a bacterial infection, but a condition produced by a byproduct of the bacteria's growth. The organism is common in nature and is widely dispersed in soils. Ingestion of the organism is not harmful. It becomes dangerous only when conditions are favorable for its growth and subsequent toxin formation. The organism grows best under high humidity and relatively high temperature and in an environment containing decaying organic material (plant or animal). The organism requires an environment in which all atmospheric oxygen is eliminated. The organism cannot multiply in the presence of air. Stagnant pools or damp areas with buried decaying matter are danger areas for toxin development. Botulism results after the decaying animal or plant material containing the toxin is consumed. Decaying carcasses are a frequent source of the toxin, as are many insects feeding in the same tissue. The insects may contain enough toxin to cause the disease in any bird that ingests it. Since the toxin is water soluble, water sources may become contaminated and provide a reservoir for the disease.

The toxin is one of the most potent discovered by scientists. The toxin is relatively heat stable but may be destroyed by boiling. There are different types of the toxin; types A and C cause the disease in birds while type B frequently produces the disease in man.

Weakness is generally the first sign of the illness and is followed by progressive flaccid paralysis of the legs, wings and neck. When neck muscles are affected the head hangs limp, thus causing a condition referred to as "limberneck". Affected birds may have a peculiar trembling, loose feathers that are pulled out easily and dull partly closed eyes. Some birds (turkey) do not develop loose feathers or limberneck symptoms. Because of the paralysis, birds are unable to swallow and mucous accumulates in the mouth. Fatally affected birds may lie in a profound coma appearing lifeless for several hours before death. Significant lesions are not usually observed in affected birds. Examining digestive contents may reveal insects, decomposed animal or vegetable material or other matter suggesting that the birds have consumed the toxin.

A tentative diagnosis can be made from the history, symptoms and post-mortem findings. As an aid to diagnosis, sick birds may be given water into the crop, kept in a cool environment and treated intravenously with antitoxin. Recovery of a large percentage of the affected birds would substantiate diagnosis.

Prevention should be aimed at eliminating sources of toxin production and preventing access of birds to such materials. These practices include prompt removal of all dead animals from houses and pens, debeaking the birds, controlling fly and insect populations and avoiding access to decaying organic material. Contaminated water supplies are particularly dangerous.

If the disease strikes, locate and remove the source of the toxin and separate all visibly affected birds from the flock for treatment. Place sick birds in a cool shaded area and give fresh water into the crop, twice daily. Mild laxatives may be used for birds that have been exposed but do not show disease symptoms. Epsom salts (one pound per 100 birds) may be mixed into feed. Adding a level teaspoonful of Epsom salts in one ounce of water and placing in the crops of sick birds has been beneficial in many instances. Antitoxin therapy is indicated only in birds that have high individual value since the antitoxin is difficult to obtain and is expensive.

Infectious Coryza

Infectious coryza is a specific respiratory disease in chickens that occurs most often in semi-mature or adult birds. Infection may result in a slow-spreading, chronic disease that affects only a small number of birds at one time, or in a rapid spreading disease with a higher percentage of birds being affected. The occurrence of infectious coryza is not widespread and the incidence is relatively low.

The disease is caused by a bacterium known as *Hemophilus gallinarum*. Outbreaks usually result from the introduction of infected or carrier birds into a flock. Transmission of the infection occurs by direct contact, airborne infection by dust or respiratory discharge droplets and drinking water contaminated by infective nasal

exudate. Susceptible birds usually develop symptoms within three days after exposure to the disease. Recovered individuals may appear normal but remain carriers of the organism for long periods. Once a flock is infected, all birds must be considered as carriers.

The most characteristic symptoms of infectious coryza include edematous swelling of the face around the eyes and wattles, nasal discharge and swollen sinuses. Watery discharge from the eyes frequently results in the lids adhering together. Vision may be affected because of the swelling. The disease results in a decrease in feed and water consumption and an increase in the number of cull birds. An adverse effect on egg production usually occurs in proportion to the number of affected birds.

Diagnosis can be confirmed only by isolation and identification of the causative organism. The organism, *Hemophilus gallinarum*, is extremely fastidious and often difficult to isolate.

Prevention is the only sound approach in controlling infectious coryza. It usually can be prevented by management programs that eliminate contact between susceptible and infected birds. It requires only separating affected or carrier birds from the susceptible population. In order to prevent the infection, introduce started or adult birds only from sources known to be free of the infection. If infection occurs, complete depopulation followed by thorough cleaning/disinfecting is the only means for eliminating the disease.

A number of drugs are effective for treating the symptoms of the disease although the disease is never completely eliminated. Sulfadimethoxine or sulfathiazole in the feed or water or erythromycin administered in the drinking water can reduce the symptoms of this disease.

Omphalitis

Omphalitis may be defined technically as an inflammation of the navel. As commonly used, the term refers to improper closure of the navel with subsequent bacterial infection (navel ill; mushy chick disease).

Apparently, most problems result from mixed bacterial infections including the common coliforms and various species belonging to the genera *Staphylococcus*, *Streptococcus*, *Proteus*, and others. Omphalitis can usually be traced to faulty incubation, poor hatchery sanitation or chilling/overheating soon after hatching (such as in transit). The significance of isolating one of the bacterial species mentioned above is complicated in that many of the same species can be isolated from the yolks of supposedly normal birds immediately after hatching.

Omphalitis occurs during the first few days of life, so it cannot be considered transmissible from bird to bird. It is transmitted from unsanitary equipment in the hatchery to newly hatched birds having unhealed navels.

Affected chicks usually appear drowsy or droopy with the down being "puffed up". They also generally appear to be of inferior quality and show a lack of uniformity. Many individuals stand near the heat source and are indifferent to feed or water. Diarrhea sometimes occurs. Mortality usually begins within 24 hours and peaks by five to seven days.

Characteristic lesions are poorly healed navels, subcutaneous edema, bluish color of the abdominal muscles around the navel and unabsorbed yolk material that often has a putrid odor. Often yolks are ruptured and peritonitis is common.

A tentative diagnosis can be made on the basis of history and lesions. The presence of mixed bacterial infections and absence of any specific disease-producing agent is used for confirming the diagnosis.

Good management and sanitation procedures in the hatchery and during the first few days following hatching are the only sure ways to prevent omphalitis. Broad spectrum antibiotics help reduce mortality and stunting in affected groups, but they do not replace sanitation.

Erysipelas

Erysipelas is a bacterial disease caused by *Erysipelothrix insidiosa*. The disease affects several species of birds including chickens, ducks and geese, but the fowl in which it has been of primary importance is the turkey. Man is susceptible to infection and may contract the disease from infected turkeys. Since this organism is pathogenic for man, care should be taken when handling infected birds or tissues.

Erysipelas in turkeys occurs most often during the fall and winter months and usually affects birds that are four to seven months of age, although any age bird is susceptible. Incidence has often been reported to be higher in males than in females, possibly because fighting males receive numerous skin abrasions that serve as portals of entry for the bacteria. In some instances the incidence is higher in hens than toms because of artificial insemination techniques that provide a means of transmission.

The organism may survive for long periods in the soil and most outbreaks are thought to originate from contaminated soil or premises. Sheep, swine and rodents may be carriers of the disease organisms. Recurrence of the disease on a premise is common. Predisposing or aggravating factors include over-crowding damp or inclement weather and poor sanitation and range management.

The first indication of the disease may be the discovery of several dead birds. Usually several morbid birds can be found; however, most affected birds are visibly sick for only a short period before death. Symptoms are typical of a septicemic disease and include a general weakness, listlessness, lack of appetite and sometimes a yellowish or greenish diarrhea. Occasionally, the snood of toms may be turgid, swollen and purple. Some birds may be found lame with swollen leg joints due to localization of the infection. In breeding flocks, this disease occasionally is associated with decreased fertility and hatchability. Daily morbidity and mortality usually are low; however, in untreated flocks mortality may persist for some time and become excessive.

The most characteristic lesions are small or diffuse hemorrhages located in almost any tissue or organ. Such hemorrhages are commonly observed in the muscles, heart, liver, spleen, fat and other tissues of the body cavities. Hemorrhagic conditions of skin may result in purple blotches. The liver and spleen are usually enlarged, congested and occasionally contain necrotic foci. Enteritis or inflammation of the intestinal tract is commonly observed, as in most septicemic diseases.

Symptoms and lesions may resemble other diseases so closely that a reliable diagnosis can be made only through isolation and identification of the causative organism.

Good management practices that aid in preventing erysipelas include avoiding the use of ranges previously occupied by swine, sheep or turkeys where erysipelas is known to have existed. Debeaking, removal of the snoods of toms, measures that prevent injury from fighting, avoiding overcrowding and providing well drained ranges will aid in preventing this disease problem.

Bacterins are available and are useful on premises where history indicates that outbreaks may be a problem. The amount and duration of protection is relative to the amount of exposure and may not be sufficient for the entire laying period. Administer bacterins in accordance with the manufacturer's directions.

Move sick birds to a hospital pen for individual treatment and to prevent cannibalism. Moving unaffected birds to a clean range may aid in preventing the spread of the disease but may also contaminate an additional range.

Various antibiotics have shown efficacy in treating erysipelas; however, penicillin is best. Penicillin injections in the leg or breast muscles of visibly sick birds is effective in decreasing mortality. One injection is usually sufficient, but more may be given if necessary. Water and feed medication may be of value under certain conditions.
