Bacteria live under all kinds of conditions and in all kinds of environments. Some, called anaerobic bacteria grow in the absence of oxygen; others, called aerobic bacteria require the presence of oxygen. Facultative bacteria grow in either condition.

Bacteria are found in and on most areas of a chicken’s body as well as throughout the poultry house environment. Chickens, as do other animals take in bacteria with every breath, morsel of food or drink of water that is consumed. Bacteria cause disease when present in numbers that overwhelm natural defenses or when the natural defenses are lowered. Some bacteria have features that permit them to cause disease even in low numbers.

Anaerobic bacteria are located in all areas of the body, surprisingly even in the lungs and frequently outnumber the more familiar aerobic bacteria by more than 100:1. Anaerobic bacteria, like other bacteria have specific requirements for growth and remain in balance with other bacteria, causing few problems. Disease may occur when this balance changes and the specific growth conditions develop that allow anaerobic bacterial multiplication. Many infections by anaerobic bacteria are endogenous in origin, meaning they result from bacteria already present in the body. Normally found in small numbers, under certain conditions the bacteria begin to proliferate and possibly cause disease.

Predisposing factors for endogenous anaerobic proliferation and infection include immunosuppression, trauma, insufficient blood supply, necrosis, malignancy and growth of aerobic or facultative bacteria. These factors provide localized environmental conditions (such as lowering the oxygen level) that are ideal for growth of the pathogenic anaerobes. The proliferating anaerobes, in turn, overwhelm beneficial bacteria furthering pathogen multiplication. Pathogenic bacteria can be active participants that exploit the situation by expressing virulence factors (toxins for example) which aid their spread.

Of the many anaerobic bacteria found in poultry, the group that is most important to poultry producers is the Clostridia. Found in every chicken house and most soils in the world the clostridia form a diverse group of bacteria often causing disease. One of the most distinguishing characteristics of these bacteria is their ability to form spores. Found in only a few types of bacteria, spores are small structures, highly resistant to environmental stresses and which consist of a tough hard coat that encapsulates the bacterial genetic material and proteins necessary for growth. Spores enable the bacteria to enter a dormant phase when environmental conditions are unfavorable and remain so for as long as necessary. Once present within a chicken house, clostridial spores can remain viable for literally centuries!

Clostridia are also capable of producing the most potent toxins known. The activity of these toxins is so powerful it would require less than a teaspoon of toxin to kill every broiler chicken in any of the top three poultry producing states in the US. The toxins are actually responsible for causing the disease. When released from the bacteria into the bird, they can have such widely varying effects as causing damage or killing tissues they contact. They also can destroy red blood cells, thereby reducing the oxygen carrying capacity of blood and in some cases prevent nerves from sending impulses to the heart and lungs.

Ulcerative Enteritis

Ulcerative enteritis primarily occurs in younger chickens (around 4 weeks of age), poults and gamebirds. Caused by the bacterium Clostridium colinum, the disease can be characterized by a sudden and rapidly accelerating mortality. It often accompanies or occurs after outbreaks of coccidiosis or other stresses that damage the intestinal integrity. It is frequently associated with immunosuppressive agents such as infectious bursal disease virus or chick anemia virus.

Transmission occurs with the ingestion of droppings, contaminated feed,
water or litter. Birds that die acutely will often appear normal, be well muscled, have feed in the crop and show few if any outward signs of disease. More chronically infected birds are listless and emaciated.

Internally, the intestines may be easily broken and the inside surfaces show deep ulcers which may penetrate through the wall of the intestine. A distinguishing characteristic is that the ulcerative lesions will be scattered throughout the entire intestine and ceca, whereas, coccidiosis affected birds will more likely exhibit a more generalized enteritis (without ulcerated lesions) that is more confined to one area of the intestinal tract. Liver lesions, consisting of circular yellow to yellow-green discolored regions are also a common feature in ulcerative enteritis cases.

It is imperative that the diagnosis be confirmed to differentiate the liver lesions, if present from those caused by histomoniasis (black head). Early or less severe cases of the disease may only show small yellow spots on the intestinal lining, sometimes surrounded by small areas of hemorrhage. Diagnosis is confirmed by the appearance of the gross lesions, histological verification and isolation and identification of the causative agent.

**Necrotic Enteritis**

Necrotic enteritis affects chickens, turkeys and quail and is caused by the bacterium *Clostridium perfringens*. Commonly found in broilers at 2-5 weeks of age, the disease is often associated with consumption of high levels of fishmeal or wheat and often accompanies or follows coccidiosis. A normal constituent of the gut, the bacteria cause few problems until some type of mucosal damage occurs.

Little is understood about the actual factors that are necessary for disease to develop. Necrotic enteritis is transmitted with the ingestion of droppings, contaminated soil, water, feed or litter.

Birds affected by the disease are severely depressed and may have diarrhea. These birds will also refuse to eat and move only if forced. The disease can also occur so acutely that few outward signs will be visible. Internally, the intestines are frequently distended with gas and easy to break. The mid- to lower portion of intestinal lining is discolored with a roughened yellow-green membrane. The liver may also be enlarged and have small dark areas.

Diagnosis is confirmed by the characteristic lesions as well as histopathological examination. Isolation of the causative agent is of less importance since it is a natural inhabitant of the intestinal tract.

**Gangrenous Dermatitis**

Gangrenous dermatitis is a disease of chickens and turkeys characterized by severe skin lesions and mortality. Caused by several anaerobic bacteria including *Clostridium septicum*, *Clostridium sordellii*, *Clostridium colinum*, and *Clostridium perfringens*, the disease can also be caused by the aerobic bacteria, *Staphylococcus aureus*, either alone or in combination with the clostridia. Generally occurring in broilers 4-8 weeks of age, gangrenous dermatitis is a frequent complication of immunosuppression caused by infectious bursal disease virus or chick infectious anemia virus.

Like the other clostridial diseases, it too can be transmitted by consumption of contaminated droppings, feed, water or litter. Staphylococcus organisms are common inhabitants of the skin and mucous membranes and therefore ubiquitous. Disease results when a combination of immune suppression occurs prior to a break in the skin integrity, through which the causative agents are introduced. Birds suffering from gangrenous dermatitis are depressed and will not eat. They may also exhibit incoordination and stagger.

![Figure 2](image) In this case of necrotic enteritis in Leghorn pullets, the stages of the disease can be seen progressing through early reddening (right) to severe destruction of the intestinal lining,
skin may be discolored red or purple but be intact. The lesions are often wet and accompanied by subcutaneous hemorrhages. The skin is occasionally ballooned and may crackle when pressed, due to subcutaneous gas production by the bacteria.

**Botulism**

Botulism is probably the most devastating of all of the clostridial diseases. Sometimes called “limberneck” botulism is caused by the bacterium *Clostridium botulinum*. The disease affects chickens and turkeys and has been responsible for the death of literally millions of waterfowl.

Unlike the other diseases described, botulism is usually caused by the ingestion of the preformed toxin rather than ingestion of the actual bacteria. Broilers are typically affected from 2-8 weeks of age and frequently as the result of cannibalism. Birds acquire the toxins by pecking the carcasses of birds that have already died from the disease. The disease can also be the result of birds eating toxin containing litter beetles, flies or maggots that have fed on decaying carcasses.

A unique feature of the disease is the mortality pattern. Usually originating in a single location, the disease spreads in a radial pattern of continually enlarging concentric rings as birds continue to cannibalize and die. The most striking indication of botulism is that affected birds will exhibit a flaccid or “limp rag” paralysis, particularly in the neck region. The degree of paralysis is dependent on the amount of toxin that has been ingested. Less affected birds may appear to be very reluctant to move from a sitting position or, if walking, will be uncoordinated in their gait. Frequently, the birds will appear asleep, with eyes shut. Some birds may also have diarrhea that tends to have more white urates than normal. Internally, lesions are rare, but the crop should always be examined for the presence of maggots, litter beetles or larvae, which may be a significant source of toxin in heavily infested houses.

**Preventing Clostridial Diseases**

The old saying “prevention is the best cure” is certainly true in the case of clostridial diseases. By their very nature, they are difficult to manage and once established in the house, the problem can become persistent due to the presence of spores. Disinfectants are of little value since bacterial spores are resistant to their activity and once in the litter spores will remain virtually forever.

Management strategies must target the buildup of the spores by regularly removing the litter completely. Cleanout scheduling is dependent upon the degree of challenge to the birds. If a flock has experienced any of the described diseases, the litter should automatically be cleaned out completely before any new flock is brought onto the premises. This cleanout should be designed to remove as much organic matter as possible thereby, reducing the overall number of spores that are present. Physical cleaning is the cheapest method of breaking the cycle of clostridial diseases. Down-time, litter treatments, heating the house and disinfectants are of little consequence in stopping clostridial diseases because they will not affect the bacterial spores.

A common preventative procedure is the use of salt on the chicken house floor. Although common, this method is ill-advised due to the environmental consequences that accompany treatment, such as ground water contamination when litter is spread on agricultural land. Significant damage can also occur to the housing (particularly steel-truss buildings) and equipment. Salt can also cause increased litter moisture problems, which can in turn cause problems with ammonia and foot damage. Salt can also alter the normal bacterial flora present in the dirt. This is important because some members of this bacterial flora can actually kill clostridia prior to spore production. In some cases, an altered flora can increase the number of clostridial spores that are present, perpetuating the problem.

Floor soil should be tested in houses that have experienced chronic prob-
lems with clostridial diseases. If the dirt in these houses is found to be alkaline, (often a consequence of previous litter or floor treatments such as lime) it should be treated to adjust the pH. Alkalinity encourages spore production. Adjustment to a more normal pH generally requires treatment with organic acids. Once adjusted, the normal, beneficial bacterial flora will quickly re-establish and help control the number of clostridia that are present.

Immunity maintenance is another important factor in preventing clostridial diseases, particularly gangrenous dermatitis, necrotic enteritis and ulcerative enteritis. The adverse effects of infectious bursal disease and chicken infectious anemia should be closely monitored. Many questions have recently arisen as to the effect the J-strain of the avian leukosis virus has on clostridial diseases, especially gangrenous dermatitis. Although, no research has specifically addressed this issue a general rule of thumb is that any stressed or immunosuppressed bird (regardless of the source) will be more susceptible to clostridial disease.

**Treatment of Clostridial Diseases**

Clostridial infections are difficult to control by antibiotic therapy and mortality may return within a flock when therapy is discontinued. In the United States, antibiotics can be administered in the feed only within the regulations established by the Food and Drug Administration. Administration of antibiotics in the drinking water may require prescription as well as a valid veterinary-client relationship. All drugs should be administered according to approved recommended doses. It is the responsibility of the user to know the regulatory requirements of the locale and to adhere to antibiotic withdrawal times prior to the processing.

Among the antibiotics with reported efficacy in treating clostridial diseases are penicillin, bacitracin, tylosin, virginiamycin, chlortetracycline, oxytetracycline and lincomycin. Others have shown efficacy, but are not approved for food animals. Bacitracin has very limited absorption from the digestive tract and should be used only for intestinal infections. Field reports indicate that treatment of gangrenous dermatitis with antibiotics in the feed is less effective than through the drinking water. Several non-antibiotic therapies are receiving attention. Increased concentrations of vitamin E in the feed are recognized as effective in prevention of gangrenous dermatitis. Vitamin E is thought to increase the effectiveness of the inflammatory response to the clostridial infection. Probiotics are cultures of beneficial bacteria that may reduce the incidence or severity of intestinal disease. During stressful situations that promote growth of clostridia, beneficial bacteria in the probiotic suspension help restore the microbial balance in the intestine.