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# A Manual of



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# POULTRY DISEASES

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# A MANUAL OF POULTRY DISEASES

Written and edited by members of the Poultry Disease Bulletin Committee



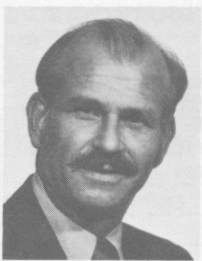
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## Section 1.

### GENERAL INFORMATION

**T**HE POULTRY INDUSTRY ranks fourth among agricultural commodities as a source of gross income in the United States and in Texas. The annual Texas gross value to the farm is about 225 million dollars. The production of poultry and eggs is highly commercialized and intensified and operates as large units.

Profit per bird is governed by fixed and variable factors. Variable factors usually determine the success or failure of an operation. A primary cost variable in poultry production is the disease level of the flock. Healthy birds are a prerequisite for profit.

Losses due to disease originate in many ways. Some are obvious, such as death, medication costs and condemnations. Others are sometimes less obvious, such as poor growth or rate of lay, poor feed conversion and downgrading.

This manual attempts to provide the professional Texas poultryman with a basic understanding of how infectious disease processes are established, ways to prevent the introduction of diseases, characteristics of more common infectious diseases of poultry and specific treatment or control of diseases.

#### POULTRY DISEASE PRINCIPLES

##### Nature and Cause of Disease

*Disease* is an alteration in the state of the body or any of the body organs which interrupts or disturbs the body's proper functions. Such disturbances often are recognized by detectable alterations of body functions.

*Etiology* is the study of disease causes. A disease state often results from a combination of two or more causes: (1) the indirect or predisposing factors which may lower the bird's resistance and (2) the direct or determining factors which produce the actual disease state.

Predisposing causes of disease are referred to frequently as "stress" factors. Stress factors may be chilling, poor ventilation, overcrowding, inadequate feeding and watering space, overmedication, etc. Disease itself may predispose another disease. For example, an outbreak of infectious bronchitis may predispose "air sac" infection.

Direct causes of disease are:

1. Bacteria
2. Viruses

3. Parasites
4. Fungi
5. Nutritional deficiencies
6. Chemical poisons
7. Unknown causes

Infectious diseases are the greatest threat to poultry health. They are caused by bacteria, viruses, rickettsia and fungi. Some protozoan diseases, such as coccidiosis and blackhead, behave much as infectious diseases and often are considered as such. More correctly, they should be classed with the other parasitic illnesses.

When living agents such as bacteria enter the body and multiply, they cause a disturbance of normal function and infection occurs. Disease is caused by the chemical toxins (poisons) produced by invading organisms. At one time some scientists believed that microorganisms caused disease by mechanical obstruction of vessels or tissue spaces. This theory has been disproved and it is now clear that damage is caused by chemical substances. But in some protozoan diseases, such as coccidiosis, mechanical damage to tissues is an important factor.

All contagious diseases are infectious, but all infectious diseases are not contagious. A *contagious* disease is one that is transmitted readily from one individual or flock to another. An *infectious* disease is one produced by living organisms. Most infectious diseases of poultry are contagious; however, a few such as aspergillosis are not.

The ability of an organism to cause disease in the particular host is known as its *virulence* or *pathogenicity*. Many microorganisms that are unable to cause disease under most conditions may cause disease under certain conditions and would, therefore, be considered pathogenic in that particular host under the existing conditions. On the other hand, some organisms almost always are pathogenic and produce disease when they enter the body of a susceptible host. Some will invade the body of only one species of birds or animals and are said to be specific for that particular species. For example, infectious bronchitis virus will cause disease only in the chicken. Other organisms affect a large number of species. For example, some of the *Salmonella* organisms affect a large variety of species including reptiles, rodents, domestic animals, poultry and man.

The ability of an organism to cause disease is not a fixed characteristic. It depends upon many



factors, such as ability to invade tissues and produce chemical toxin. Often pathogenicity can be altered intentionally. This characteristic has been used in developing some vaccines. Variation in pathogenicity of organisms also explains partially why the same disease may present different forms and degrees of severity.

#### How Infectious Diseases Are Spread

Some of the more common ways infectious diseases are introduced into and spread within the poultry flocks are:

1. Introduction of diseased birds.
2. Introduction of healthy birds that have recovered from disease but are still carriers.
3. Contact with inanimate objects (fomites) that are contaminated with disease organisms (poultry crates, feeders, waterers, etc.)
4. Carcasses of dead birds that have not been disposed of properly.
5. Impure water, such as surface drainage water.
6. Rodents and free-flying birds.
7. Insects — fowl pox transmitted by mosquitoes.
8. Shoes and clothing of men who move from flock to flock.
9. Feed or contaminated feed bags.
10. Contaminated premises through soil or old litter.
11. Airborne — organisms do not spread far through the air, but this source of infection can be very important in heavily populated poultry areas.
12. Egg transmission — a number of diseases such as pullorum and fowl typhoid are egg transmitted.

#### Body Defenses Against Disease

The body has a well-developed defense mechanism that must be understood and utilized in controlling infectious diseases. *Immunity* means the ability to resist infection; however, this ability can be overcome under certain conditions. Resistance is used interchangeably with immunity.

An animal has two types of protective mechanisms: (1) those that hinder or prevent invasion of organisms and (2) those that combat agents which invade the body.

Mechanisms which hinder or prevent invasion of organisms include the intact skin and mucous membranes which create a direct barrier, secretions

such as mucous which tend to dilute and wash out invading organisms and cilia (hair-like projections on some mucous membranes) which, with wavelike action, move foreign material out of such structures as the trachea (windpipe).

Mechanisms which combat agents that invade the body include the white blood cells and circulating antibodies.

Immunity or resistance is outlined as follows:

1. Innate or inherited.
  - a. Species
  - b. Racial (strain or breed)
  - c. Individual
2. Acquired
  - a. Active
    - (1.) Resulting from having the disease
    - (2.) Stimulated by vaccination with dead or living disease agents
  - b. Passive
    - (1.) Injection of antiserum
    - (2.) Transferred from dam to offspring

Inherited resistance may be complete or partial; for example, turkeys are not susceptible to laryngotracheitis, and although chickens are more resistant than turkeys to blackhead, they may become infected under certain conditions. Inherited resistance or susceptibility to lymphoid leukosis is well established, but no completely resistant breed or strain of chickens has been developed. Individual resistance is apparent in practically every disease outbreak in a poultry flock. Some birds, although exposed to the same chances for infection, fail to develop evidence of the disease.

While inherited immunity is important, acquired immunity is a more controllable reaction that can be used intentionally by the poultryman. Acquired immunity is the reaction we hope to stimulate by application of all vaccines. The purpose of vaccines is to stimulate an active production of *antibodies* by safe means. Active immunity depends upon the production of antibodies within the body of each individual. Antibodies are proteins associated with the globulin fraction of the blood serum. Antibody production is not understood completely, but antibodies are apparently produced by various organs such as the liver, spleen and bone marrow. In general, antibodies are specific for the organism which stimulated their production; thus, immunity to one disease ordinarily does not imply resistance to others.

*Passive immunity* is the transfer of antibodies from the individual in which they are produced



to another individual. This is done by the injection of serum from an immunized individual. Antibodies also are transferred from the dam to the offspring through the egg; thus, hens that have had Newcastle disease transfer antibodies through the yolk to their chicks. Such passive immunity is an important consideration in vaccination programs. Passive immunity is of short duration and there is usually a marked decline in the antibody level within 21 to 30 days. Passive protection against infection usually lasts no longer than 4 to 6 weeks.

### Manifestations of Disease

Detectable signs of disease are known as *symptoms*. Visible changes in the size, color, shape or structure of an organ are known as *lesions*. Loss of body weight, decreased egg production, reduced feed consumption, droopiness and lameness are some symptoms. An enlarged liver, tumor on the intestine, abscess in a lung or collection of exudate in an air sac are examples of lesions.

Many symptoms are *general*; they usually are seen in any diseased individual. Examples are droopiness, ruffled feathers, diarrhea and loss of appetite. Other symptoms are *specific*; they are seen only when certain diseases are present. Examples of such symptoms are the tremors associated with avian encephalomyelitis (epidemic tremors) and the flaccid paralysis associated with botulism.

Lesions likewise may be of general or specific nature. For example, enteritis is associated with many diseases, but the "gray eye" of ocular leukosis is specific.

### Flock Health Management

Sanitation is a much used, but poorly defined word. The usual implication is that sanitation is a universally understood practice that may be applied to prevent all diseases. This concept often leads to misunderstanding and disappointment. Good sanitation in relation to one disease actually may provide favorable conditions for the development of other diseases. Although many good sanitation measures always should be applied, others must be based on the nature of specific diseases. The ambiguity surrounding the term "sanitation" can be avoided by using the expression "management and sanitation for disease prevention." This phrase then would be defined as all practices, specific and nonspecific, that the poultryman applies to prevent disease or reduce severity and economic loss from diseases.

A standard disease prevention program that can apply on all poultry farms does not exist. But there are some basic principles that always should be observed.

Some practices that aid in disease prevention are:

1. Select a well-known, reliable source from which to purchase chicks, poults or hatching eggs — one that can supply healthy stock, inherently vigorous and developed for a specific purpose.
2. It is best to purchase only day-old chicks or hatching eggs. If it is necessary to purchase started birds, select the best possible source.
3. Keep birds separate according to source and age groups. To mix birds is an invitation to trouble.
4. Follow an "all in, all out" program.
5. Change litter and thoroughly clean and disinfect the house and equipment between each group of birds. While litter selection and management is a large subject, applying this recommendation as a general practice will prevent many disease and parasite problems.
6. Keep chickens and turkeys separate. Preferably, only chickens or only turkeys should be kept on the same premises.
7. Maintain hatchery supply flocks on separate premises from other birds.
8. Select a reliable commercial feed, or, if farm mixing is done, mix carefully according to a dependable formula.
9. Provide an adequate supply of wholesome water. Avoid watering from surface tanks, streams or ponds.
10. Make and carry out a precise vaccination schedule for each flock. Work out the vaccination program with poultry disease authorities in each state or local area.
11. Discourage persons other than the caretaker or essential personnel from visiting the poultry house or yard.
12. If a disease problem develops, obtain an early, reliable diagnosis and apply the best treatment, control and eradication measures for that specific disease.
13. Dispose of all dead birds by burning, deep burying or by a disposal pit. This phase of management often is overlooked.
14. Maintain good records relative to flock health. These should include vaccination history, disease problems and medication employed.

Many facts of disease prevention are acquired only through experience and a well-rounded grasp of modern poultry husbandry.

## Hatchery Management and Sanitation

The information contained in this section has been adapted from recommended procedures of the National Poultry Improvement Plan and National Turkey Improvement Plan.

*Hatching egg sanitation.* Collect hatching eggs from nests at frequent intervals and observe the following practices:

1. Use cleaned and disinfected containers to collect the eggs and take precautions to prevent contamination from organisms that may be on hands or clothing.

2. Maintain the identity of all eggs as to the breeder flocks of origin.

3. Do not use dirty eggs for hatching purposes. Collect them in a separate container from hatching eggs. Slightly soiled eggs may be dry cleaned by hand or by a motor-driven buffer.

4. As soon as possible after collection, fumigate the visibly clean eggs as described under the subheading "fumigation."

5. After fumigation, store eggs in a cool place. Store eggs for as short a period as possible before setting. Properly clean and disinfect racks used for storing eggs.

6. Use new or fumigated cases to transport eggs to the hatchery. Discard soiled egg case fillers.

*Hatchery sanitation.* An effective program for the prevention and control of Salmonella and other infections includes these practices:

1. Arrange the hatchery buildings so that separate rooms, with separate ventilation, are provided for each of the four operations; egg receiving, incubation and hatching, chick holding and disposal of offal and cleaning of trays. Place these rooms under isolation so that admission is granted only to specifically authorized personnel who have taken proper precautions to prevent introduction of diseases.

2. Thoroughly clean and disinfect frequently the hatchery rooms, tables, racks and other equipment in them. Burn all hatchery wastes and offal or otherwise properly dispose of them. Clean and sterilize containers used to remove such materials after each use.

3. Thoroughly clean and fumigate the hatching compartment of incubators, including the hatching trays after each hatch.

4. Use only clean eggs for hatching purposes. Fumigate all eggs set prior to setting or within 12 hours after they are placed in the incubator. Also fumigate them after transfer to the hatching compartment.

5. Use only new or clean fumigated egg cases for transportation of hatching eggs. Destroy soiled egg case fillers.

6. Distribute day-old chicks, poults or other newly hatched poultry in clean, new boxes or in disinfected plastic cartons. Clean and disinfect all crates and vehicles used for transporting started or adult birds after each use.

7. Maintain the identity of all chicks and poults as to the breeder flock of origin.

8. Do not mix the progeny of different breeder flocks if it can be avoided.

### *Cleaning and disinfecting.*

1. In poultry houses and hatchery rooms, cleaning and disinfecting should include these steps:

- a. Settle dust by spraying lightly with disinfectant.

- b. Remove all litter and droppings to an isolated area where there is no opportunity for dissemination of any infectious disease organisms that may be present.

- c. Scrub the walls, floors and equipment with a hot soapy water solution. Rinse to remove soap.

- d. Spray with a cresylic disinfectant, such as liquor cresolis saponatus, 4 ounces to a gallon of water, or sodium orthophenylphenate, 1 1/3 ounces (1 heaping tablespoonful) to a gallon of hot water.

2. In the hatcher, cleaning and disinfection include the following procedures:

- a. Remove trays and all controls and fans for separate cleaning. Thoroughly wet the ceiling, walls and floors with a stream of water; then scrub with a hard bristle brush. Rinse until there are no deposits on the walls, particularly near the fan opening.

- b. Replace cleaned fans and controls. Replace trays, preferably still wet from cleaning, and bring the incubator up to the normal operating temperature.

- c. Before placing eggs in the hatcher, it should be fumigated.

- d. If eggs are hatched in the same machine as they are incubated, clean the entire machine after each hatch. Use a vacuum cleaner to remove chick down from the egg trays.

*Fumigation.* Fumigation of eggs and incubators is an essential part of a hatchery sanitation program.

1. Preincubation fumigation of eggs should be done as follows:

- a. Provide a room or cabinet proportionate to the number of eggs to be handled.



The room should be relatively tight and equipped with a fan to circulate the gas during fumigation and to expel it after fumigation.

- b. Place the eggs in the room on wire racks which will not prohibit air circulation, and expose to circulating formaldehyde gas.
  - c. Formaldehyde gas is provided by mixing 0.6 grams of potassium permanganate with 1.2 cc of formalin (37.5 percent) for each cubic foot of space in the room. Mix ingredients in an earthenware or enamelware container having a capacity of at least ten times the volume of the total ingredients.
  - d. Circulate the gas within the room for 20 minutes; then expel.
  - e. Humidity for this type of fumigation is not critical but the temperature should be around 70 degrees F. Extra humidity may be provided in dry weather.
2. Eggs which have not been fumigated prior to setting should be fumigated as soon as possible and no later than 12 hours after setting using the following procedure:
- a. Determine the size of the incubator by multiplying the length times the width times the height.
  - b. After setting the eggs and allowing temperature and humidity to regain normal operating levels, release formaldehyde gas into the incubator.
  - c. For each cubic foot of space in the incubator, use 0.4 grams of potassium permanganate and 0.8 cc of formalin (37.5 percent). Use a container having a capacity of at least ten times the volume of the total ingredients.
  - d. Close vents and doors but keep circulating fan operating and continue fumigation for 20 minutes with normal operating temperature and humidity.
  - e. After 20 minutes of fumigation, open vents to the normal operating positions to release the gas.
3. Eggs not fumigated as described in paragraph 1 or in paragraph 2 of this section should be fumigated after the ninety-sixth hour of incubation. Follow the procedure described in paragraph 2 of this section. Single or repeated fumigation of eggs in the setter may be practiced, but the fumigation schedule should be such that no eggs

are fumigated during the period from the twenty-fourth to the ninety-sixth hour of incubation.

4. Refumigate all eggs after transfer to the hatcher, preferably as soon as the temperature and humidity regain normal operating levels. Follow the procedure described in paragraph 2 of this section.

5. Fumigate empty hatchers between each hatch. After the interior of the hatcher has been cleaned thoroughly and the cleaned trays returned, follow the procedure below:

- a. After temperature and humidity are brought to normal operating levels, use 0.6 grams of potassium permanganate and 1.2 cc of formalin (37.5 percent) per cubic foot of space in the hatcher.
- b. Close doors and vents and leave closed overnight.

#### PRINCIPLES OF REASONABLE DRUG ADMINISTRATION

Drugs and chemicals are used widely in poultry production. Arsenicals and antibiotics are added to the ration as growth stimulants. Various compounds known as coccidiostats are added to the ration to prevent coccidiosis. Often drugs are included in rations "just in case they might help prevent some potential disease." Antibiotics sometimes are recommended at times of "stress" such as moving, vaccination or debeaking.

While the use of any drug on a nonspecified basis may be of value, any real benefit is difficult to determine. A tremendous amount of money is spent by the poultry industry yearly for drugs that are of little or no value in preventing or reducing disease.

The most valid use of drugs is in the application of known effective treatments for specific diseases. Such treatments must be based on a reliable diagnosis. Recommendations for the treating of poultry diseases are changing constantly as new, more effective drugs are developed, or as once effective compounds become ineffective because organisms have developed resistance or for other reasons.

In using any drug, follow the recommendations of persons qualified to give such directions or follow the manufacturer's recommendations.

#### Drug Administration

Drugs may be administered to poultry in several ways. The choice of method depends upon a number of factors including: (1) the disease in question; (2) the drug to be used; (3) available labor and administration equipment; (4) the condition of birds; and (5) the length of the medication period.

The following outline shows commonly employed methods of drug administration:

1. Mass methods
  - a. Incorporation into feed
  - b. Incorporation into drinking water
  - c. Aerosol, or dusting the drug into the air over the birds
2. Individual bird treatment
  - a. Parenteral (subcutaneous, intramuscular or intravenous injection)
  - b. Drenching

Mass methods of drug administration are popular because they save labor; eliminate the necessity of handling each bird, a "stress" which may aggravate the disease state; and allow for continuous medication over a prolonged period. Disadvantages of mass methods include the inability to control individual bird doses. Conversely, individual bird treatment requires more labor, "stresses" the birds and does not lend itself to continuous medication. However, it does allow for accurate controlled dosage to each bird and offers a means of treating birds when an existing disease is not subject to mass methods of treatment.

Drugs should always be used in accordance with the manufacturer's recommendations which take into consideration: (1) safe levels, (2) approved combinations and (3) adequate withdrawal time to avoid residues, etc.

### Preventive Medication

It is doubtful that there is a justification for the continuous use of drugs in the average poultry operation other than for growth stimulation and the prevention of coccidiosis and, on occasion, blackhead.

There are several reasons why indiscriminate preventive medication should be discouraged. Among these are:

1. Expense
2. Indiscriminate use of drugs frequently allows drug-resistant strains of bacteria to develop. When this happens, previously effective drugs lose their value for the treatment of actual disease outbreaks.
3. Preventive medication often allows the grower to develop a false sense of security about disease control. As a result, management and sanitation practices are neglected.
4. Preventive medication often masks the true nature of a particular disease and may make diagnosis extremely difficult. Frequently, it is impossible to isolate causative organisms by laboratory

techniques when the birds have been on continuous medication.

### Treatment of Disease Outbreaks

Initiate drug treatment of disease only after a reliable diagnosis has been established. To do otherwise is costly and often produces serious bird losses. For example, an outbreak of erysipelas in turkeys does not respond to the usual treatments employed for fowl typhoid or fowl cholera. Misdiagnosis results in drug expense and a continued loss due to mortality. Hemorrhagic anemia syndrome of chickens is confused easily with coccidiosis. If a flock affected with the former condition is treated for coccidiosis, the existing problem is aggravated and severe losses may occur.

Once an accurate diagnosis has been established, follow the prescribed recommendations for treatment very closely. Many drugs produce toxic effects if used improperly.

## VACCINATION TO PREVENT POULTRY DISEASES

### What Vaccines Are

Vaccines are suspensions of large amounts of the disease-causing organism or virus in a diluent. Most virus vaccines contain living organisms (except for the killed type of Newcastle disease vaccine). Virus vaccines are produced by growing laboratory strains of virus in embryonated chicken eggs or in cell culture systems. Strains of virus differ just as do strains of chickens within a particular breed. Strains selected for making vaccines usually are mild so they will not cause a serious infection but still stimulate immune body production.

Bacterial vaccines (*bacterins*) are produced by growing selected strains of bacterial organisms in artificial media. The organisms are killed after they are harvested for bacterin production. These products are incapable of producing infection but will stimulate antibody production.

In general, living vaccines produce better immunity than dead ones; however, the dangers associated with vaccination are greater.

### Dangers of Vaccination

Most vaccines contain living viruses intended to produce a mild infection. In other words, vaccines make the birds sick. The sickness will be mild if:

1. Birds are healthy at the time of vaccination.
2. Chicken house or brooder house is clean and dry.
3. There are no sudden climate changes.
4. Birds are at the proper age for vaccination.



5. There is ample heat available to the birds. (Raise the temperature about 5 degrees F. for a few days after vaccination.)
6. Instructions in the package of vaccine are followed.

Since most vaccines contain living agents, vaccine virus may spread to unprotected birds on the same premise. This may cause adverse effects, particularly in unprotected laying flocks. Seek expert assistance when it appears desirable to initiate a vaccination program on property where unprotected layers are present. The wing-web type of Newcastle vaccine, infectious bronchitis vaccine, fowl pox vaccine, infectious laryngotracheitis vaccine, infectious bursal agent (IBA) vaccine and epidemic tremor vaccine may spread from flock to flock on the same farm.

#### Vaccination — No Substitute for Sanitation

A sound vaccination program is part of a good management and sanitation program and not a substitute for it.

It is unnecessary to vaccinate against certain diseases in some parts of the country and in some areas within a particular state. Vaccination should be tailored to meet the needs of a particular operation in a particular area. For example, Texas poultrymen should not use laryngotracheitis vaccine or attempt to move laryngotracheitis vaccinated birds into the state. This disease is not a problem in Texas and the Animal Health Commission has issued a restraining order against the introduction of this vaccine into the state.

#### Diseases for Which Vaccines Are Available

Of the products on the market for the control of virus infections, respiratory virus infections lead the list but others are becoming increasingly important.

1. Newcastle disease
2. Infectious bronchitis
3. Infectious laryngotracheitis (to be used only with the permission of the Animal Health Commission)
4. Fowl pox (may cause symptoms and lesions of the respiratory system, but not primarily a respiratory disease)
5. Avian encephalomyelitis (*epidemic tremors*)
6. Gumboro (*infectious bursal disease*)—to be used only under the direction of the state regulatory people or poultry pathologists
7. Marek's disease vaccine (Turkey Herpes virus)—newest of vaccines and should be used under strict compliance with manufacturer's directions

Bacterins are available commercially for the control of:

1. Erysipelas
2. Fowl cholera
3. Mixed (not in wide acceptance)

Coccidiosis control with the use of live oocysts administered early in the baby chicks' life (Coc-civac\*) has gained acceptance with poultrymen as one approach toward controlling coccidiosis outbreaks in both light and heavy breed layers.

#### Administration of Vaccines

Vaccines must be used properly if they are to be effective. For best results:

1. Store vaccines in the refrigerator according to manufacturer's recommendations.
2. Do not use outdated vaccines.
3. After a vial of vaccine has been opened, destroy by burning any remaining contents after use. Do not set back for use at a later date.
4. Administer in accordance with manufacturer's instructions.
5. Record serial numbers of all vaccines used.

Vaccines, as drugs, may be applied in different ways, depending on the product used, age of birds and other factors. In general, Newcastle disease, infectious bronchitis, epidemic tremor, gumboro and coccidiosis vaccines are adapted to mass methods of administration, whereas fowl pox, infectious laryngotracheitis, erysipelas, fowl cholera and Marek's disease vaccines are adapted for individual bird administration.

1. Mass methods:
  - a. Drinking water — Newcastle, infectious bronchitis, epidemic tremors, gumboro, cocci-vac
2. Individual methods:
  - a. Intranasal; intraocular — Newcastle infectious bronchitis
  - b. Wing-web stab — Newcastle, fowl pox
  - c. Feather follicle — fowl pox (pigeon pox vaccine)
  - d. Subcutaneous; intramuscular injection — Newcastle (killed), erysipelas, fowl pox, Marek's and fowl cholera

Vaccines frequently are combined to reduce labor and the number of immunization procedures required. Newcastle disease vaccine often is combined with that of infectious bronchitis. While the use of such combination products may have

\*Trade name of a product containing sporulated oocysts to be used in the immunization of chickens against coccidiosis.

merit in some cases, their routine use is not recommended. There are two good reasons why:

1. Evidence exists that the host response to one virus fraction may interfere with the development of adequate immunity to the other.
2. It is impossible to evaluate the vaccine reaction. (Is the reaction due to one or both fractions in the vaccine?)

After live virus vaccination, birds must be observed for "takes" to the vaccination. A discussion of this is included in the individual disease sections.

### **A Suggested Vaccination Program for Chickens**

Because of the fast changing size of poultry operations within the state, it must be pointed out that each operation should have its own "tailor-made" vaccination program. Seek advice from a poultry disease specialist and/or vaccine manufacturer in developing a program.

For those with smaller, isolated flocks the programs as outlined should work.

1. *Replacement Chicks (Commercial egg or breeders)*
  - a. Vaccinate for Newcastle at 7 days and 4 weeks of age with intranasal Newcastle vaccine using the drinking water method.
  - b. At 6 weeks of age, vaccinate for infectious bronchitis using the drinking water method.
  - c. When birds are 8 weeks old, vaccinate for fowl pox by the wing-web method.
  - d. Revaccinate pullets against Newcastle disease when they are moved into the laying house if intranasal type of vaccine is used. If wing-web method is to be used, vaccinate 4 weeks before pullets start production.
2. *Chicks for Broiler Production*
  - a. Vaccinate against Newcastle at 7 days of age using intranasal vaccine by the drinking water method. A second dose may be administered at 4 weeks.
  - b. At 2 weeks of age, vaccinate against infectious bronchitis using the drinking water method.
  - c. Vaccination of broilers against fowl pox is not recommended in areas except where experience shows it needs to be done, and then at a time experience indicates it will give the most satisfactory results.

### **Use of Other Vaccines in Chickens and Turkeys**

Undertake vaccination of chickens for disease other than specifically outlined above only after getting expert advice.

Vaccination of turkey flocks for Newcastle disease, fowl pox, erysipelas and fowl cholera is discussed under individual disease headings. Usually the decision to vaccinate, except for fowl pox, should be made on the basis of area experience and expert advice.

### **USING THE DIAGNOSTIC LABORATORY**

The Texas Agricultural Experiment Station, under the direction of the College of Veterinary Medicine at Texas A&M University, operates Poultry Disease Investigation Laboratories at College Station, Center and Gonzales. These laboratories serve several functions:

1. To serve as diagnostic and information centers where Texas poultrymen can obtain assistance with their poultry disease problems.
2. To accumulate information relative to the incidence and importance of the various poultry diseases in the state.
3. To help formulate and carry out poultry disease research projects which reflect the needs of the Texas poultry industry.

### **Laboratory Locations**

Locations of the Poultry Disease Investigation Laboratories are:

1. College Station. Room 101, Veterinary Medical Sciences Building, Texas A&M University, Telephone 713/845-5941.
2. Center. Corner of Childs Street and Malone Drive near Center High School. Telephone 713/598-4451.
3. Gonzales. One block South of U. S. Highway 90A on Water Street (1972). Telephone 512/437-2212.

Laboratories are open from 8 a.m. to noon and 1 p.m. to 5 p.m. Monday through Friday, University holidays excluded. Make prior arrangements to assure service at other times.

In addition to the laboratories listed, there are some practicing veterinarians who are well qualified by training and interest to assist with poultry disease problems.

### **Using the Laboratories**

Disease prevention is the best approach to disease control. Utilize laboratory personnel to assist in developing disease prevention programs and not solely as "firemen" to help when disease appears.

When a disease problem develops, however, get help immediately rather than calling the laboratories as a last resort. If emergency treatment is



necessary, remove a sample of birds for diagnostic purposes before treatment is started.

The selection of a sample of birds for the laboratory should not be a culling operation. Birds submitted should be representative of the condition thought to be the flock problem, and should be in various stages of the disease. Submit three or four birds, or more if young chicks or poults. When

birds are dying rapidly with few preliminary symptoms, bring in several dead birds with the sample. Dead birds should be placed in plastic bags and refrigerated.

Diagnosis often is difficult if birds are submitted without adequate information. The following outline includes information which should be routinely available.

Owner \_\_\_\_\_

Address \_\_\_\_\_ Phone No. \_\_\_\_\_

Number in flock \_\_\_\_\_ Breed \_\_\_\_\_ Age \_\_\_\_\_

Hatchery source \_\_\_\_\_

Type of operation (Floor, cage, range, etc.) \_\_\_\_\_

Feeding program \_\_\_\_\_

Vaccination history \_\_\_\_\_

Illness first seen \_\_\_\_\_

Morbidity (No. affected) \_\_\_\_\_ Mortality \_\_\_\_\_

Evidence of illness \_\_\_\_\_

Medication \_\_\_\_\_

Remarks (other flocks on farm, previous problems, etc.) \_\_\_\_\_

#### What to Expect

When the pathologist is able to diagnose the cause of trouble without the necessity of time-consuming tests, recommendations are made directly to the individual submitting the birds.

In other instances, only a tentative diagnosis can be established when birds are submitted. In this case, preliminary recommendations pending final diagnosis may be made depending upon the particular situation.

Laboratory tests (culture, virus isolation, tissue pathology) are time consuming. When these tests are necessary for final diagnosis, a few days to a few weeks may be required to complete them. Then final diagnosis and recommendations are made to the owner by letter or phone.

For the protection of the industry, it is the policy of the laboratories to dispose of all poultry submitted.

## Section 2.

### BACTERIAL DISEASES

Bacteria are microscopic living organisms. They are grouped into spherical forms, straight rods, curved or spiral rods and filamentous forms. Growth requirements of different species vary considerably, but most can be grown on artificial media. Bacteria, like other living organisms, have certain requirements as to environmental temperature, moisture and nutrition for propagation.

Not all bacteria are detrimental to animal health. In fact, most bacteria are necessary for such processes as food digestion. Classification of bacteria into species so that disease-producing organisms may be separated from those that are harmless or beneficial is based on such laboratory techniques as staining of organisms for microscopic examination, determination of fermentation reactions in various liquid media, serologic reactions and others.

Successful control of bacterial diseases is based upon isolation and identification of the disease-producing species, if present, and prevention of multiplication or spread of the organism within the animal body or to other animals. This should be done with a minimum detrimental effect upon the beneficial organisms.

#### SALMONELLA AND PARACOLON INFECTIONS

There are more than 2,000 different species or serotypes of organisms belonging to the genus *Salmonella*, all of which are potential pathogens of poultry. Systemic effects usually are observed when infection occurs, but since the digestive system is affected primarily, they often are referred to as enteric organisms. The same is true of the group of organisms referred to as paracolons. Because of similarities produced by infections by these groups of organisms, they are grouped under one heading. Both groups are worldwide in distribution.

#### Pullorum Disease

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

*Etiology:* *Salmonella pullorum* — first isolated by Rettger in 1900

*Transmission:* primarily egg transmitted but transmission may occur by other means

1. Infected hen=egg=infected chick=spread in incubator=in chicken boxes=in brooder house

and on range = survivors become infected breeder birds

2. Mechanical transmission — carried about on shoes, equipment
3. Carrier birds — apparently healthy birds which shed organisms
4. Contaminated premises — from previous outbreaks

Portal of entry may be the respiratory (as in incubator) or digestive system.

Most outbreaks of acute pullorum disease in chicks or poults result from infection while in the hatchery.

*Incubation period:* 5 to 7 days

*Symptoms:* Pullorum disease is highly fatal to young chicks or poults but mature birds are more resistant. Young birds may die so soon after hatching that no symptoms are observed. Most acute outbreaks occur in birds under 3 weeks of age. Mortality in such outbreaks may approach 90 percent if untreated. Survivors usually are stunted or unthrifty.

#### TYPICAL SYMPTOMS IN CHICKS OR POULTS: (not constant)

- Droopiness
- Ruffled feathers
- Chilled appearance, huddled around source of heat
- White diarrhea and pasted down around vent
- Labored breathing

#### SYMPTOMS IN ADULTS: (Usually no recognizable symptoms)

*Lesions:* Young birds dying during an acute outbreak may present no recognizable gross lesions. Representative lesions in young birds include:

- Necrotic foci in liver (varying from pinpoint to pea size)
- White nodular areas in muscle of heart and gizzard; occasionally in wall of intestine
- Multiple small, firm, nodular areas in lungs
- Yellow or cream-colored cecal cores or plugs

#### *Lesions in adults:*

- Gross lesions may be lacking.



- "Blighted" ova may be present.
- Livers may contain areas of necrosis.
- Oviducts may contain cheesy deposits.

*Diagnosis:*

- Blood testing may indicate presence of the disease.
- May be suspected from history, symptoms and lesions.
- Positive diagnosis depends upon isolation and identification of the organism by laboratory methods.

*Prevention:* Complete eradication is the only sound way to prevent pullorum disease. Test all hatchery supply flocks and accept only pullorum-free flocks for the production of hatching eggs.

The National Poultry Improvement Plan (NPIP) and the National Turkey Improvement Plan (NTIP) are national organizations formed primarily for eradicating pullorum disease. These organizations began in 1935 and since then the disease virtually has been eliminated from areas where most hatcheries participate in the plan. These organizations are helped and coordinated by the Agricultural Research Service (ARS) and each state has a local organization with state supervisors to administer the plan.

*Serologic tests:* All tests for pullorum disease are agglutination tests; however, the three procedures accepted as official testing methods are:

1. Whole blood rapid plate test. This test employs stained antigen which is mixed with a drop of blood on a glass plate. The test ordinarily is conducted and read in the field.

2. Rapid serum plate test. This test is identical to the whole blood test except that serum is used instead of whole blood.

3. Tube agglutination test. The test is conducted with an unstained antigen and serum, incubated at 37.5 degrees C. for 24 hours prior to reading and is performed in NPIP approved laboratories. The tube test is considered more reliable than other tests and is the only official test for turkeys in Texas.

Advantages of the tube test:

1. Conducted under controlled laboratory conditions.
2. A small number of designated well-trained individuals read and interpret the test.
3. The ratio of antigen to serum can be varied to help in interpreting the reaction.

Advantages of whole blood rapid plate test:

1. Faster
2. Less expensive
3. Reactors can be removed from flock at the time of testing so that birds do not have to be handled a second time.

Other procedures which must be followed in maintaining pullorum-free breeder flocks:

1. If a flock is infected it is advisable to dispose of the flock rather than try to dispose of reactors and establish a negative flock.

2. After disposing of infected birds, thoroughly clean and disinfect the house and equipment. A 3 percent water solution of cresol (liquor cresolis saponatus) is excellent for such purposes.

For incubator fumigation procedures follow procedures outlined in Section 1.

Why "breaks" occur in a previously "clean" flock:

1. Introduction of the infection since last test
2. Infection with a "variant" strain of the organism not detected by the standard test

*Treatment:* It is primarily a salvage operation and does not prevent birds from becoming carriers. Consequently, do not keep recovered flocks for egg production.

The preferred drug for treating infected flocks is furazolidone, marketed under the trade names of NF-180 by Hess and Clark or Furox by Norwich Agriculture Products.

The recommended level is 2 pounds NF-180 concentrate or Furox 50 (100 grams furazolidone) per ton of feed for 10 days to 2 weeks or until mortality stops. In severe cases the drug level may be increased to 4 pounds (200 grams furazolidone) per ton of feed.

**Fowl Typhoid**

Fowl typhoid is an infectious, contagious bacterial disease that is usually acute, but may be chronic. It affects most domestic and wild fowl including chickens, turkeys, ducks, pigeons and pheasants although in Texas, outbreaks most often occur in turkeys. Do not confuse it with typhoid fever of humans which is caused by a separate and distinct organism.

*Etiology:* *Salmonella gallinarum*, formerly known as *Shigella gallinarum*, discovered by Klein in England in 1889.

*Incubation period:* Variable but usually 4 to 5 days.

*Duration:* Varies from 5 to 6 days in the acute form to weeks or months in the chronic form in which the daily mortality rate is often low.

*Transmission:* Methods of transmission are the same as for pullorum disease, including egg transmission, but mechanical transmission is more important than it is in pullorum disease.

*Age susceptibility:* Any age bird may be affected, but the disease occurs primarily in young adults (usually those past 12 weeks of age).

*Mortality rates:* Variable, ranging from less than 1 to 40 percent or higher, especially if treatment is not initiated promptly.

*Symptoms:* Symptoms may be suggestive of fowl typhoid but they are not specific. Some typical symptoms include:

- Sudden or sporadic mortality
- Listlessness
- Green or yellow diarrhea with pasting of the vent feathers
- Loss of appetite
- Increased thirst
- Pale, anemic appearance of comb and wattles

*Lesions:* In addition to the symptoms mentioned, certain lesions observed at necropsy will help substantiate a diagnosis of fowl typhoid. These include:

- Enlargement of the spleen which may be mottled
- Liver usually considerably enlarged and varying in color from yellow to greenish brown, often with visible necrotic foci
- Small pinpoint hemorrhages in the muscles and fat, particularly that surrounding the organs
- A slimy type inflammation of the anterior third of the small intestine

In turkeys, the presence of small, white plaque-like areas visible through the walls of the intestine is very suggestive of fowl typhoid.

*Diagnosis:* A tentative diagnosis usually may be made from consideration of the history, symptoms and lesions. Final diagnosis must be based on isolation and identification of the causative organism since other diseases often will closely resemble fowl typhoid.

*Serology:* Blood tests used for detection of pullorum reactors also are used in control of fowl typhoid. Both organisms bear such close antigenic relationship that one test will suffice for both. But the test is suggestive rather than conclusive.

#### *Prevention and control:*

Vaccination — bacterins presently available are of little or no value and may cause the flock to react to the pullorum test for approximately 60 days after vaccination.

Prevention and control depends upon:

- Hatching from disease-free flocks as established by the pullorum test
- Strict sanitation on the farm
- A clean, safe water supply (Avoid watering from ponds or surface tanks)
- Use of an incinerator or disposal pit for disposal of all dead birds

(The causative organism may live for at least 6 months under certain conditions. Following an outbreak, thoroughly clean houses and equipment. When feasible, practice range rotation and other special precautions to prevent a carryover of infection to the next flock.)

Drugs (e.g. furazolidone at the rate of 50 grams per ton of feed or even higher levels) cannot be depended upon as a means of prevention and are not recommended.

*Treatment:* Furazolidone is the choice drug and levels of 100 grams per ton of feed for 10 to 14 days usually will stop mortality. However, since some strains of the fowl typhoid organisms are resistant to this drug, it may be necessary to use higher dosage levels (200 grams per ton of feed) for longer periods of time. After mortality is controlled the furazolidone level may be decreased to 50 grams per ton of feed. Give the lower level continuously until the flock can be marketed.

In rare instances it may be possible to stop treatment after 30 days, but if mortality should recur, use the treatment level again for a few days.

Sulfaquinolaxine is effective as a treatment and may be used for medication, except in laying chickens, through water while waiting for feed containing the furazolidone. It also may be used for treating flocks which do not respond favorably to furazolidone.

Commercial liquid preparations vary in concentration so the manufacturer's recommendations should be followed to achieve the desired water concentration. For the first 2 days, the drug is mixed in the drinking water at a concentration of 1:2500. Then reduce to a concentration of 1:4000. Give the lower level until mortality is controlled.

#### **Paratyphoid Infections**

The term "paratyphoid" was used first to designate a group of human, feverish conditions resembling typhoid fever. Related to poultry, para-

typhoid is a term denoting the disease produced by any of the many *Salmonella* species other than *S. pullorum* and *S. gallinarum*. Infection may result in acute or chronic disease. Acute clinical disease is common in young birds and rare in adult birds. Over 2000 species or serotypes of *Salmonella* organisms are recognized, and most birds, reptiles and mammals can serve as host to one or more species. Economically, the disease is of greatest concern to the turkey industry.

*History:* In 1895, Moore recorded the first authentic case of paratyphoid when he isolated and identified a *Salmonella* from pigeons with an enteritis. The first report of paratyphoid infection occurring in turkey poults in the United States was that of Rettger *et al.* in 1933, although Pomeroy and Fenstermacher observed the infection in turkeys in Minnesota in 1932, according to a report published in 1939.

*Etiology:* The organisms of this group are serologically related and are Gram negative, nonspore forming, flagellated, motile rods. They can be separated from *S. pullorum* and *S. gallinarum* by morphology and biochemical characteristics, but one paratyphoid organism cannot be differentiated from another member of the group except by serologic methods.

*Pathogenicity:* Most acute paratyphoid infections occur in birds less than 4 weeks old, except in pigeons and canaries in which acute disease and high mortality may occur in any age group.

Mortality in young turkey poults usually varies from less than 1 percent to 10 or 20 percent, although rarely it may exceed 80 percent. Most death losses in young birds occur during the first 2 weeks after hatching, the mortality curve closely resembling that of pullorum disease. Outbreaks in ducks ("keel disease") often result in severe losses.

Severity of infection with each of the various serotypes has been determined. There is a tendency to consider *S. typhimurium* as a type species representative of the whole group and as the most important cause of paratyphoid in poultry. This species commonly is isolated from birds involved in paratyphoid outbreaks throughout the country.

Results of research to date indicate that all *Salmonella* species must be considered as potential pathogens in poultry. Outbreak severity depends upon age and species of host, serotype or species of *Salmonella* involved and certain environmental or management factors.

*Host distribution:* The host range of the *Salmonella* species probably is as great as that of any pathogenic organism. It includes most domestic

animals, birds, rodents, snakes, lizards and man. The greatest reservoir of *Salmonella* is probably poultry, and the organisms are encountered most frequently in turkeys, chickens and ducks.

*Transmission:* The transmission of paratyphoid includes all factors involved in the transmission of pullorum and typhoid plus several other. Because of the multiplicity of hosts and wide distribution in nature, chances of clean flocks becoming infected are greater than for either of the other two diseases. Direct ovarian transmission may occur in both chickens and turkeys but is more common in ducks. Of greater importance in chickens and turkeys is contamination of the egg shell by fecal material during or after passage through the cloaca. The organism, being motile, rapidly penetrates the shell and shell membrane (rate of penetration depends upon such factors as temperature and humidity) and gains access to the interior where it may survive until hatching. Most instances of shell penetration occur during the first week of incubation.

During incubation, "blow-up" of infected eggs aids in spreading the organism. Many infected chicks or poults hatch which increases contamination of the incubator. The incubator environment favors survival of the organisms to infect subsequent hatches unless fumigation procedures are practiced routinely.

An additional source of infection may be indicated by reports of recovery of many serotypes from poultry feeds. Investigators in a number of states including Texas, have reported recovering one or more species from 20 to 30 percent of the samples taken from certain materials such as animal by-products used as protein sources. The incidence is much lower in vegetable protein sources and there appears to be no incidence in pelletized or crumbled feeds.

*Symptoms and lesions:* Symptoms are variable, depending mainly upon the species and age of the bird, pathogenicity of species of *Salmonella* involved and method of transmission, but in general, the symptoms resemble those associated with pullorum disease. Some characteristic symptoms include:

- Huddling around the source of heat with lowered head, eyes closed and wings drooping
- Increased thirst with decreased food consumption
- Watery diarrhea and pasting of feathers around the vent
- Increased "peeping" or "chirping" sounds

Arthritis and swollen joints are observed commonly in paratyphoid outbreaks in pigeons and sometimes occur in outbreaks in turkeys or chickens.



In acute outbreaks gross lesions may be absent in young birds. Characteristic lesions in young birds may include:

- Emaciation and dehydration
- Unabsorbed or coagulated yolks
- Congested livers, sometimes with hemorrhagic streaks and/or pinpoint white foci of necrotic tissue
- Inflammation of the intestine, especially the upper portion
- Cores in the ceca having creamy or yellow color

Acute infections in adult birds usually produce few if any lesions other than enteritis. Carriers and chronically infected adults usually have no specific lesions.

*Diagnosis:* The disease may be suspected from flock history, symptoms and necropsy lesions, but a definite diagnosis depends upon isolation and identification of the organisms by qualified laboratory personnel.

*Prevention and control:* This is difficult because of the wide range of hosts harboring organisms and because no single species or serotype of the organism is suitable as an antigen for detecting all other species in the testing program. *S. typhimurium* is used commonly as a representative for the group in attempting to standardize testing procedures. The pullorum-typhoid test often will detect infected flocks even when the typhimurium antigen fails. Because of the many sources of infections, known clean flocks may become infected at any time subsequent to the last test.

An organized effort to control and eradicate paratyphoid exists in many states. Some hatcheries in other states reject eggs from flocks which have not been tested with the *S. typhimurium* antigen. Such control efforts are not the final answer to this problem, but they should be encouraged and additional information utilized as it becomes available. The possibility of contamination by organisms in feeds is now an important area in research and control programs.

Pending additional information, hatchery and flock sanitation management practices are the most important factors in paratyphoid prevention and control. Some practices known to aid in paratyphoid control include the following:

- Do not use flocks known to be infected as a source of hatching eggs.
- Follow hatchery and egg sanitation practices that reduce chances of introducing infection

into the incubator through fecal contamination.

- Early fumigation of eggs with formaldehyde gas (before incubation or within 24 hours after the eggs are placed in the incubator).
- Rodent control
- Isolation from other sources of infection such as pigeons and ducks.
- Serological testing of hatchery supply flocks and following outlined requirements of voluntary programs.

*Treatment:* Proper use of drugs may reduce mortality in acute outbreaks of paratyphoid. The drug of choice is furazolidone but some species or serotypes are more resistant to treatment than the pullorum or fowl typhoid organisms. Some paratyphoids have or will develop resistance to the effects of furazolidone in time.

The recommended level of furazolidone for treatment is 100 grams per ton of a complete ration as the only source of feed. Continue treatment for 2 weeks or until mortality is controlled.

Continuous use of furazolidone at a rate of 50 grams per ton of feed included frequently as a prophylactic measure. There is no assurance that such a practice will be effective in all cases. Field information indicates that in some instances such practices actually prevent the flock owner from discovering a paratyphoid infection in the flock.

Sulfonamides, such as sulfaquinoxaline and sulfamethazine, have some value in treating paratyphoid outbreaks, but they are much less effective than furazolidone.

No treatment is known that will eliminate infection from the flock following an outbreak, and efforts to test and eliminate individuals harboring the organisms have been unsuccessful. Prevention is of primary importance. Regardless of treatment, never use infected birds to supply hatching eggs.

### Paracolon Infections

The paracolon bacteria comprise a large group of related organisms that have certain characteristics in common with the paratyphoids and also with the common coliforms. Most pathogenic paracolon organisms are placed in the group known as Arizona paracolons. They can be differentiated from the paratyphoids by their biochemical reactions, but the similarity between groups causes some delay and confusion in correct identification. These organisms are distributed widely in nature and have a host range which coincides with the *Salmonella*.

The role of the paracolons in causing poultry disease is poorly established. Under certain con-

ditions, these organisms may cause disease in young turkey poults, and thus be of economic importance.

Consider the disease produced, symptoms, lesions, transmission, prevention and treatment as identical to the paratyphoid infections until research further clarifies the situation. Differentiation of paracolon from paratyphoid infections now depends on careful laboratory examination with isolation and identification of the causative organism.

The importance of Arizona paracolon infection in chickens is not well defined. However, the Arizona organisms may produce severe losses in young turkey poults. The nature of the disease, including transmission, is similar to paratyphoid from which it cannot be differentiated except by laboratory means.

Procedures employed to control Arizona infection are similar to those in use for paratyphoid infections. However, since infection is so widespread in many turkey breeder flocks, the disease in young poults is controlled largely by day old poult injection programs.

#### **COLIFORM INFECTIONS, COLIBACILLOSIS**

Coliform infections refer to the many and various diseases resulting from infection with bacteria referred to as *Escherichia coli*. In recent years these infections have become recognized as an important cause of morbidity, mortality and condemnations in both chickens and turkeys. The incidence and severity of coliform infections have increased rapidly, and current trends indicate they are likely to become an even bigger problem in the future.

The problems attributed to coliform infections are often complex. There is a marked variation in severity. They 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 disease from generalized infections, an enteritis from intestinal infection or a combination of any or all of these. Disease may result from 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 classical air sac disease syndrome as a complication of *Mycoplasma gallisepticum* infections.

All ages may be affected; however, it is more common in young<sup>2</sup> growing birds, especially the acute septicemia in young turkeys and airsacculitis in young chickens. High early mortality may occur as the result of omphalitis or navel infections.

*Cause:* The disease is caused by bacteria known

as *E. coli* and from toxins it produces as it grows and multiplies. There are many different strains or serological types within the group of *E. coli* bacteria. Many of these types are considered to be normal inhabitants of the intestinal tract 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. On one extreme are the severely pathogenic strains which may alone cause disease. On the other extreme are the supposedly nonpathogenic strains which are considered to be innocuous. All degrees of pathogenicity exist between these two extremes. Certain types are recognized which normally live in harmony with the bird although they are capable of causing disease under certain conditions.

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

*Symptoms:* The symptoms vary with the different types of infections. In the acute septicemic form, mortality may begin suddenly and progress rapidly. Birds may die in good condition and morbidity may not be apparent. However, in most cases, morbid birds are evident as listless birds with ruffled feathers and indications of fever. In the chronic infection, debilitation and growth retardation are obvious. In the event of respiratory infection, additional symptoms of labored breathing, occasional coughing and rales may be apparent. In the case of enteritis, diarrhea may be evident. Mortality may be high in recently hatched chicks or poults as a result of omphalitis due to coliform infections.

*Lesions:* Extremely acute septicemic infection may result in sudden death with very few, if any, lesions apparent. The death resulted from the effects of the organisms' toxins on the functions of the vital organs. However, lesions are usually present in various tissues depending on the location of the infection. In acute infection, common lesions include dehydration, swelling and congestion of the liver, spleen and kidneys and pinpoint hemorrhages in the viscera. The presence of caseous or fibrinous 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 areas of hemorrhage and excess amounts of mucous. In addition to these lesions, navel infections may be seen in young birds similar to those described for omphalitis.

*Diagnosis:* Differential 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 readily accomplished; however, mere isolation is not sufficient to make a diagnosis. One must take into consideration the organ from which the organism was isolated, the pathogenicity of the particular isolate and the presence of other disease agents.

*Prevention:* Management and sanitation practices designed to minimize the exposure level of these types of organisms in the birds' environment are necessary in any preventive program. In addition, these programs should include avoiding stress factors and other disease agents which may lower the resistance and predispose the birds to infection.

Important points in these management and sanitation practices include providing adequate ventilation, good litter and range conditions, properly cleaned and disinfected equipment and facilities and feed and water supply free of contamination. In addition, these programs should include avoiding overcrowding and environmental stresses such as chilling and overheating and avoiding vaccination and handling at critical times. Proper egg handling as well as a good hatchery management and sanitation program is necessary to prevent early exposure.

It should be emphasized that problems due to one of the more pathogenic strains may occur even under the most ideal conditions.

*Treatment:* The response of coliform infections to various medications is erratic and often difficult to evaluate. Under practical conditions, treatment is disappointing and the results so variable that no one treatment can be recommended. Drug sensitivity varies with the strain, some of which may be partially or completely resistant to many if not all of the commonly used antibiotics. Tests performed in the laboratory to determine the sensitivity of the organism to the various drugs may prove useful in selecting the drugs which may be most beneficial. When practical, moving the birds to 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. The management and sanitation practices listed under the heading "Preventive Medication," Section 1, also should be considered as an integral part of any treatment.

### 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).

*Cause:* Considerable research as to the cause or causes of omphalitis has been undertaken during recent years. 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 usually can be traced to faulty incubation, poor hatchery sanitation or chilling or 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.

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

*Symptoms and lesions:* 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 is observed. Mortality usually begins within 24 hours and peaks by 5 to 7 days.

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

*Diagnosis:* 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 aids in confirming the diagnosis.

*Treatment and prevention:* 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.

### FOWL CHOLERA

Fowl cholera was recognized as a separate, distinct disease by Pasteur in 1880. Salmon first reported its presence in the United States in 1880-83. The 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, water fowl, sparrows and other free flying birds.



*Cause:* The causative organism of fowl cholera is *Pasteurella multocida* (also called *P. avicida*), a bacterial organism in the form of a small oval rod, distinctly bipolar when stains are made from blood or tissues. It is grown easily on artificial media provided its demanding nutritive requirements are met. The organism is identified by staining techniques which permit observation of the typical morphology and by determination of biochemical reactions in artificial media.

*Transmission:* *Pasteurella multocida* will survive for (1) at least 1 month in droppings, (2) 3 months in decaying carcasses or (3) 2 to 3 months in soil. The organism may enter the body through the digestive tract or the respiratory system. The disease is not transmitted through the egg.

Major sources of infection are:

- Body excreta of diseased birds which contaminates soil, water, feed, etc.—this may be from visibly sick birds or apparently healthy carriers
- Carcasses of birds which have died of the disease
- Contaminated water supply such as surface tanks, ponds, lakes or streams
- Mechanical transmission by contaminated shoes or equipment

Recent studies indicate that animals other than birds, such as racoon, opossum, dog and pig may serve as reservoirs of infection and actively spread the disease.

*Symptoms and lesions:* The disease seldom is seen in chickens under 4 months of age, but is commonly seen in turkeys under this age. The usual incubation period is from 4 to 9 days and outbreaks may vary from peracute to chronic in nature. In the peracute form, symptoms may be entirely absent; in the acute form some birds may die without showing symptoms, but many others are visibly ill before death. Characteristic symptoms include:

- Stupor
- Complete loss of appetite
- Rapid weight loss
- Lameness resulting from joint infection
- Swollen wattles
- Difficult breathing
- Watery yellowish or green diarrhea
- Dull blue or purple color of head and wattles due to cyanosis

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 include any or all of the following:

- 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
- Numerous small white necrotic foci throughout the liver
- Enlarged and congested spleen
- Creamy or solid collection of material in joints
- 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

*Diagnosis:* A tentative diagnosis may be made on flock history, symptoms and postmortem lesions. Demonstration of bipolar staining rods in blood smears or impression smears of the lungs, liver or spleen helps to substantiate the diagnosis. A definite diagnosis depends upon isolation and identification of the organism.

*Prevention and treatment:* Bacterins properly applied 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, to expect 100 percent protection is unrealistic.

- Use commercial bacterin first and try auto-genous bacterin only in situations where commercial bacterins have failed.
- Follow manufacturer's recommendations as to age, time to vaccinate and dosage (this is usually two times, at 8 and 12 weeks of age).
- If breaks occur after 2 vaccinations, do not revaccinate; try to salvage on the market.
- Vaccination in conjunction with treatment is not recommended.

Sanitation practices which aid in preventing the disease are:

- Complete depopulation each year between older birds and replacements
- A good rodent control program
- Proper disposal of dead birds
- A safe, sanitary water supply

- Adequate cleaning and disinfection of all houses and equipment on premises where outbreaks have occurred after disposal of affected flocks
- Keeping birds of susceptible age confined to the house
- Allowing contaminated ranges or yards to remain vacant for at least 3 months

Sulfaquinoxaline is the preferred drug for the treatment of fowl cholera, except in laying hens, using the same level as recommended for fowl typhoid. It may be administered in feed or water, but treatment may be necessary for 3 to 4 weeks or longer. In outbreaks requiring prolonged treatment, give the medication in the ration to guard against danger of water starvation. Sulfaquinoxaline at the rate of 0.033 percent (1 pound of pure drug per 3,000 pounds of complete ration) can be used continuously without toxic effects.

Since the sulfonamides are not cleared for use in laying chickens, the tetracyclines are commonly used for the treatment of fowl cholera in layers.

### ERYSIPELAS

Erysipelas is a bacterial disease caused by *Erysipelas insidiosa* (formerly *Erysipelas rhusiopathiae*) and was once considered to be a serious disease only in swine and sheep. The disease in swine frequently is referred to as "diamondskin disease." It affects several species of birds including chickens, ducks and geese, but the only fowl in which it has been of importance is the turkey. Man is susceptible to infection and may contract the disease from turkeys. However, in man the disease known as erysipelas is caused by a bacteria of the genus *Streptococcus*, whereas infection of man with *Erysipelas insidiosa* is called erysipeloid. Since this organism is pathogenic for man, care should be taken when handling infected birds or tissues.

*Cause:* Erysipelas of fowl is caused by the bacterium, *Erysipelas insidiosa*. In turkeys it occurs most often in the fall and winter months and usually affects birds which are 4 to 7 months old, although any age bird is susceptible. Incidence has often been reported to be higher in males than in females, possibly because in fighting, males receive numerous skin abrasions which may serve as portals of entry. However, in some instances the incidence may be higher in hens than toms and frequently follows artificial insemination which may be 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. Recurrence of the disease on a premise is common.

Predisposing or aggravating factors include overcrowding, damp or inclement weather and poor sanitation and range management.

*Symptoms:* 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, inappetence and sometimes a yellowish or greenish diarrhea. Occasionally, the snood of toms may be turgid, swollen and purple in appearance. Some birds may be found lame with swollen leg joints due to localization of the infection. In breeding flocks, it has occasionally been associated with decreased fertility and hatchability. Daily morbidity and mortality are usually low; however, in untreated cases mortality may persist for some time and become excessive.

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

*Diagnosis:* 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.

*Prevention:* Good management practices which aid in preventing erysipelas include avoiding the use of ranges previously occupied by swine, sheep or turkeys in areas where erysipelas is known to exist, debeaking, removal of the snoods of toms and other measures which prevent injury from fighting, avoiding overcrowding and providing well-drained ranges.

Bacterins are available and are useful on premises where history indicates outbreaks may be expected. Three weeks are required to produce the maximum protection following an injection. If birds are vaccinated at 10 to 12 weeks of age, the procedure should be repeated for birds held over as breeders. The amount and duration of protection is relative to the amount of exposure and may not be sufficient for the laying period. Bacterins should be administered in accordance with the manufacturer's directions.

Antiserum prepared in horses is available and effective under certain conditions. This product is expensive and its use is very limited.

*Treatment:* Sick birds should be removed from the flock 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 will also contaminate another range. In addition, bacterin may be used on unaffected birds.

Various antibiotics have shown efficacy in the treatment of erysipelas; however, penicillin is the drug of choice. Injections of 150,000 to 200,000 units of penicillin into the leg or breast musculature of visibly sick birds is very effective in decreasing mortality. One injection is usually sufficient, but may be repeated if necessary. Water and feed medication may be of value under certain conditions.

### AVIAN VIBRIONIC HEPATITIS

Avian vibrionic hepatitis is a widespread transmissible disease of chickens characterized primarily by swelling and necrosis of the liver. It may appear in an acute form resulting in death of affected birds, or it may occur in a chronic form and produce economic loss by increasing flock cull rates. Birds of all ages may be affected, but the disease commonly occurs in semi-mature and mature birds.

*Cause and transmission:* The causative agent of vibrionic hepatitis is a bacterial organism belonging to the vibrio group.

The disease apparently spreads by contact, direct or indirect, between infected and susceptible birds. Ingestion of infectious material is the most likely method of transmission. Some outbreaks present an appearance that suggests possible egg transmission.

*Symptoms and lesions:* Usual disease signs are listlessness, shrunken comb, loss of body weight and diarrhea. Acutely affected birds, however, may die while still in good flesh. Egg production may drop as much as 35 percent in severely affected flocks. Mortality usually is low but may be as high as 10 to 15 percent.

The liver is the primary site of infection. Livers of affected birds usually are swollen and have necrotic and hemorrhagic foci. The heart and kidneys may be swollen, and there may be excess fluids in the abdominal cavity and the heart sac.

*Diagnosis:* Liver lesions are found in birds affected with many diseases. Because of this, vibrionic hepatitis may be confused with diseases such as pullorum, typhoid, bluecomb, hemorrhagic disease, blackhead and leukosis. Positive diagnosis is established by laboratory means.

*Treatment and prevention:* Outbreaks are treated best by adding furazolidone to feed at a level

of 200 grams per ton. Supply medicated feed for approximately 10 days.

Furazolidone usually prevents outbreaks, but the use of the drug for preventive purposes is not recommended. Routine management and sanitation practices for disease prevention offer the most economical and reliable method of prevention.

### BOTULISM

#### (Limberneck; Food Poisoning)

Botulism is a disease caused by the ingestion of a toxin produced by the anaerobic bacterium *Clostridium botulinum*. All domestic fowl and most wild birds are susceptible. An interesting exception is the vulture which is apparently immune and feeds on decaying carcasses. Many human deaths have been attributed to eating food or drinking water containing the toxin.

*Cause:* Botulism is not a bacterial infection but rather a condition produced by ingestion of a toxin produced by the bacterium *Cl. botulinum*. 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 the growth and multiplication of the bacteria and its subsequent toxin production. The organism grows best under conditions of high humidity and relatively high temperature and in an environment containing decaying organic material. Acid conditions are detrimental. Stagnant pools or damp areas containing decaying matter with an alkaline reaction are a danger area. Botulism may result from consumption of any decaying animal or vegetable material. Decaying carcasses are a frequent source of toxin and fly maggots feeding on such tissue may contain enough toxin to cause the disease when ingested. The toxin is water soluble; consequently, water sources may become contaminated.

The toxin is one of the most potent known, being about 17 times as deadly for the guinea pig as is cobra venom. The toxin is relatively heat stable but may be destroyed by boiling. There are different types of the toxin. Types A and C usually are responsible for the disease in birds while type B most frequently affects man.

*Symptoms:* First signs of illness generally are weakness, followed by progressive flaccid paralysis of the legs, wings and neck. When neck muscles are affected, the head hangs limp and this is often referred to as "limberneck." Affected birds may have a peculiar trembling, loose feathers in the follicle and dull and partly closed eyes. 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.



*Lesions:* Usually there are no significant lesions evident in affected birds with the possible exception of loose feathers and an excess amount of mucous and dirt in the mouth. Examining the contents of the crop and intestines may reveal maggots or other material to indicate that the birds may have consumed the toxin.

*Diagnosis:* A tentative diagnosis may be made from the history and such factors as loose feathers, mucoid accumulations in the mouth and the absence of other postmortem lesions. As an aid to diagnosis, sick birds may be given water into the crop, kept in a cool environment and treated with antitoxin intravenously. Recovery of a large percentage of birds thus treated would substantiate the diagnosis. Additional supportive evidence for diagnosis may be obtained by production of the identical signs in other birds or laboratory animals by inoculating them with material from the digestive tract of affected birds or material from the suspected source of toxin.

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

*Treatment:* Remove all visibly sick birds from the flock. Place in a cool shaded area and give water into the crop, twice daily. Mild laxatives may be used for birds which have been exposed but do not yet show the symptoms. Antitoxin may be useful in treating affected birds, but it is difficult to obtain. In turkeys administer 2 to 4 cc of polyvalent antitoxin intravenously, interperitoneally or intramuscularly.

#### ARTHRITIS/SYNOVITIS

Inflammations of the joints and synovial membranes are a major cause of economic loss to the poultry industry. Inflammatory processes of this nature cause losses due to mortality, retarded growth, poor feed conversion, condemnation and downgrading.

There are many causes of arthritis/synovitis. Among them are injury, nutritional disturbances and infectious agents. Two of the most common infectious conditions are staphylococcal arthritis and infectious synovitis.

#### Staphylococcal Arthritis

Staphylococcal (staph) arthritis is an acute to chronic disease affecting chickens and turkeys, but

is of greater incidence among turkeys. The cause of this condition is *Staphylococcus aureus*.

*Transmission:* The organism of staph arthritis is universally present wherever poults are raised. The manner by which the disease is established is not well understood, but injury may predispose the condition in some instances.

*Symptoms and lesions:* Morbidity is usually low. Acute cases of staph arthritis are commonly accompanied by a septicemia which may lead to death in several days. Diarrhea, depression and swollen joints are common signs. More chronic cases are characterized by lameness and loss of condition in addition to the above signs. Death due to cannibalism occurs commonly in affected turkeys.

Lesions include synovitis and arthritis, particularly of the hock joint and the surrounding region. Affected joints and membranes contain large amounts of serous and caseous exudates. The livers and spleens of affected birds are commonly congested and swollen.

*Diagnosis:* A diagnosis of staph arthritis is based upon the presence of characteristic lesions and the recovery of *Staphylococcus aureus* from tissues or exudates of affected birds.

*Treatment and prevention:* Affected birds should be segregated from the flock. In general, they will respond poorly to treatment. Of the many drugs and antibiotics which have been used in attempts to treat the disease, novobiocin at levels of 200 to 350 grams per ton of feed has produced the best results.

Management practices which reduce the chances of injury are important in the prevention of the disease.

#### Infectious Synovitis

Infectious synovitis is a chronic disease of chickens and turkeys and is characterized by inflammation of joints and synovial membranes. It is found in all major poultry-producing areas of the country and may cause severe losses due to mortality, retarded growth, poor feed conversion and downgrading at processing plants. Although the disease may appear in adults, it is primarily a disease of growing birds, particularly in the 4-to-12-week age groups.

*Cause:* The cause of infectious synovitis is *Mycoplasma synoviae*.

*Transmission:* As with other pathogenic avian mycoplasma, *M. synoviae* is egg transmitted, and the infected breeder must be considered the principal reservoir of infection within an infected flock. Contact transmission readily occurs between infected and susceptible pen mates.

*Symptoms and lesions:* Morbidity of infectious synovitis may reach 20 percent or more before an outbreak has run its course. Mortality is low. Lameness is the first symptom observed. Swelling associated with the hocks, foot pads and shanks usually is seen in chickens; however, shank swelling is seen infrequently in turkeys. Rapid loss of condition, dehydration and diarrhea usually are seen. Respiratory signs may be present.

Autopsies of affected birds often reveal the following lesions. Joints and synovial membranes of legs and wings usually are inflamed and contain tenacious mucoid exudates. In turkeys, such lesions are rare except as associated with the hocks and foot pads. Inflammation of the sternal bursa (breast blister) is also a common finding. Visceral lesions may include swollen livers with greenish discoloration, enlarged spleens and airsacculitis.

*Diagnosis:* A presumptive diagnosis may be based on flock history, symptoms and lesions. Laboratory tests may be necessary to differentiate the disease from staphylococcal arthritis and other conditions producing leg weakness and sternal bursitis.

*Treatment and prevention:* Birds affected with infectious synovitis respond poorly to treatment. Treatment, however, alters the course of an outbreak, primarily by reducing disease spread. The antibiotics of choice are chlortetracycline and oxytetracycline. The drug is incorporated into the feed at a level of 200 grams per ton and fed for 7 to 14 days, depending upon flock response. If practical, remove crippled birds from the flock since they respond poorly to treatment and could serve as a continued infection source.

If relapses occur, it may be necessary to feed lower levels of antibiotics continuously until time of market.

At present, the adoption of a sound sanitation program offers the best hope to prevent introduction of infectious synovitis. Without question, an eradication program, comparable to that for *M. gallisepticum*, will be developed in the foreseeable future.

### HEMORRHAGIC ENTERITIS

Hemorrhagic enteritis is an acute and fatal intestinal disorder of turkeys.

*Cause:* Unknown. Numerous organisms have been isolated in laboratories from infected birds. However, experimental studies indicate that the disease is infectious and that the agent responsible for the disease is a filterable agent which can be serially passed with streptococci, suggesting that the cause of the disease may be a toxin elaborated by streptococci.

*Symptoms and lesions:* Hemorrhagic enteritis has been observed in many strains of turkeys and on various feed programs. Greatest incidence appears to be during hot, dry weather. It is seen most often in range birds 9 to 13 weeks of age. Fortunately, total mortality seldom exceeds 10 percent.

Usually, the only sign is one or more dead birds on range. Mortality may continue for a few days and then stop. In some cases, daily loss of a few birds may last several weeks.

Occasionally, a few birds may appear sick before they die. Symptoms are not characteristic. Affected birds may appear drowsy and pale. Although there may be some bloody droppings, they usually are not observed in an infected flock.

Lesions are confined primarily to the intestinal tract. The most characteristic finding is a severe hemorrhagic inflammation of the intestinal lining from the gizzard to the ceca. The intestines are filled with blood and debris having a jam-like consistency. Free dark blood may extend into the ceca and gizzard.

Occasionally, small hemorrhages may occur in the muscles of the breast and legs and on the heart, liver, kidneys and other internal organs.

*Diagnosis:* Gross lesions are sufficiently characteristic to allow a diagnosis in most cases.

*Treatment and prevention:* There has been no specific treatment. Changing the ration has appeared effective in some cases; however, spontaneous recovery may have taken place irrespective of the change. Moving birds to new range may be beneficial. Provide affected flocks with an abundance of fresh, pure water; give them adequate shade from the sun if possible. Remove dead birds from the range promptly. Recent reports, however, indicate that the injection of serum collected from recovered birds effects a favorable response on a flock basis.

## Section 3.

### RESPIRATORY DISEASES

Diseases are often grouped according to the body system they affect. Those affecting mainly the air passages, windpipe, lungs and air sacs are classified as "respiratory diseases" and are among the major threats to poultry health.

At first, all diseases of the respiratory system were known as "colds" and often were considered to be caused by environmental factors such as drafts or chilling. As more information accumulated, it became apparent that "colds" were actually a group of separate infectious diseases having many common characteristics.

Possibly all infectious agents causing respiratory symptoms have not been recognized, but many have been isolated and the diseases they produce well defined. Because the nature of the causative organism tells much about a disease, the following outline according to cause is useful in understanding these diseases.

#### *Caused by viruses:*

- Newcastle disease
- Infectious bronchitis
- Laryngotracheitis
- Quail bronchitis
- Influenza/parainfluenza

#### *Caused by bacteria:*

- Mycoplasmosis
- Infectious coryza
- Endemic fowl cholera (roup)
- Psitticosis/ornithosis

#### *Caused by molds:*

- Aspergillosis (Brooder pneumonia)

#### NEWCASTLE DISEASE

Newcastle disease is a contagious viral infection, causing a respiratory nervous disorder in several species of fowl including chickens and turkeys. It was first recognized in England in 1926 and was named after the town of Newcastle. It first appeared in the United States in 1944. Within the next few years, Newcastle disease was present throughout the country.

*Cause:* Newcastle disease is caused by a virus. Different types or strains, varying in their ability to cause death and nervous disorders, have been

recognized. Some are highly fatal, but most American strains are more fatal in young birds than in adult birds.

*Transmission:* Newcastle disease is highly contagious. All birds in a flock usually become infected within 3 to 4 days. The virus can be transmitted through contaminated equipment, shoes, clothing and possibly free-flying birds. During the active respiratory stage, it can be transmitted through the air. Probably the virus does not travel any great distance by this method. Recovered birds are not considered carriers, and the virus usually does not live longer than 30 days on the premises.

*Symptoms and lesions:* Signs of Newcastle disease are not greatly different from those of other respiratory diseases. The ones most frequently observed are: (1) nasal discharge, (2) excessive mucous in the trachea, (3) cloudy air sacs, (4) casts or plugs in the air passages of the lungs and (5) cloudiness in the cornea of the eye.

The disease in young chickens begins with difficult breathing, gasping and sneezing. This phase continues for 10 to 14 days and may be followed by nervous symptoms. If nervous disorders develop, they may consist of paralysis of one or both wings and legs or a twisting of the head and neck. The head is often drawn over the back or down between the legs. Mortality may vary from 0 to near 100 percent.

In adult chickens, respiratory symptoms predominate. Only rarely do nervous disorders develop. If the flock is laying, egg production usually drops rapidly. When this occurs, it takes from 4 to 6 weeks or longer for the flock to return to the former production rate. During the outbreak, small, soft-shelled, off-colored and irregular-shaped eggs are produced. Mortality in adult birds usually is low but may be fairly high from some virus strains.

In turkeys, the symptoms usually are mild and may be unnoticed unless nervous disorders develop. During an outbreak, turkeys will produce eggs with a chalky white shell. Reduced production in breeder flocks is the main economic loss from this disease in turkeys.

*Diagnosis:* The flock history, signs of a respiratory nervous disorder and other typical lesions often may be sufficient to allow a tentative diagnosis. Usually, however, the disease cannot be differentiated from infectious bronchitis and some of the other respiratory infections except by lab-



oratory methods. Laboratory procedures necessary to establish a definite diagnosis sometimes are complex and time consuming. They can be conducted only in a well-equipped laboratory.

*Prevention and treatment:* There is no treatment for Newcastle disease. The disease does not always respect even the best management program, but practices outlined in other sections of this bulletin will help reduce the possibility of exposure to Newcastle disease virus.

Vaccination is practiced widely and is the recommended method for prevention. Several types of vaccine are available but the most successful and widely used is the mild live virus vaccine known as the B<sub>1</sub> type. This vaccine was used originally by dropping it into the nostril or eye. Now the vaccine is usually added to the drinking water.

Broilers usually are vaccinated when 7 to 10 days old with this B<sub>1</sub> type of vaccine. In some operations, two doses of vaccine are used. In such cases, the first dose is administered when birds are approximately 7 days of age and a second dose is applied at about 4 weeks.

Chickens to be kept for egg production should be given three applications of the vaccine. The vaccine usually is given when the birds are approximately 7 days, 4 weeks or 4 months of age.

Vaccination is not widely practiced in turkeys. It sometimes is used to protect egg production in breeder flocks. Give one dose of the B<sub>1</sub> type vaccine after selecting the breeder birds.

### INFECTIOUS BRONCHITIS

Infectious bronchitis is an extremely contagious respiratory disease of chickens characterized by coughing, sneezing and rales (rattling).

*Cause:* Infectious bronchitis is caused by a virus which affects chickens only. Other fowl or laboratory animals cannot be infected with this virus.

*Transmission:* Infectious bronchitis is considered the most contagious disease known. When it occurs, all susceptible birds on the premises become infected regardless of sanitary or quarantine precautions. The disease can spread through the air and can "jump" unknown distances during an active outbreak. It also can be spread by mechanical means such as clothing, poultry crates and equipment. The disease is not egg transmitted and the virus will survive only for a short time, probably not more than 1 week in a poultry house. It is destroyed easily by heat or the ordinary disinfectants.

*Symptoms and lesions:* The infection is confined to the respiratory system. Symptoms are

difficult breathing, gasping, sneezing and rales. Some birds may have a slight watery nasal discharge. The disease never causes nervous symptoms. It prevails 10 to 14 days in a flock. Symptoms lasting longer than this usually are from some other cause.

In chickens under 3 weeks of age, mortality may be as high as 30 to 40 percent. The disease does not cause a significant mortality in birds over 5 weeks old. Feed consumption decreases sharply and growth is retarded.

When infectious bronchitis occurs in a laying flock, production usually drops to near zero in a few days. Four to 6 weeks or longer may be required before the flock returns to production. Some flocks never regain an economical rate of lay. During an outbreak, small, soft-shelled, irregular-shaped eggs are produced.

*Diagnosis:* Infectious bronchitis is difficult to differentiate from several of the other respiratory diseases. For this reason, a definite diagnosis usually requires laboratory procedures.

*Treatment and prevention:* Infectious bronchitis is highly contagious and does not always respect sanitary barriers. Vaccinate chickens to be retained for egg production. Whether broilers should be vaccinated depends upon many factors and is an individual decision. Numerous vaccines are available commercially. Most of them represent a modified or selected strain of infectious bronchitis virus. All vaccines contain live virus, and those that give good protection also are capable of producing symptoms and reducing egg production. The vaccine virus will spread to other susceptible birds. Vaccine is usually added to the drinking water, but may be administered by dropping it into the eye or nostril.

There is no treatment for this disease. In young chickens it is helpful to increase the brooder temperature and to provide as nearly ideal environmental conditions as possible.

### LARYNGOTRACHEITIS

Infectious laryngotracheitis is an acute, highly contagious disease of chickens and pheasants. It is characterized by respiratory distress, rapid spread and high mortality.

*Cause:* This disease also is caused by a virus.

*Transmission:* Recovered birds remain carriers for as long as 2 years. Carriers also develop following vaccination if the virus becomes established in the respiratory system. During an active outbreak, the disease can spread by mechanical methods such as clothing and equipment. The most important factor in spreading the disease is the carrier bird.

*Symptoms and lesions:* The disease usually occurs in semi-mature or adult birds. It is acute and affected birds usually die or recover in 5 to 6 days. Some virus strains are more mild and the course of the disease may be as long as 15 days or more. Coughing, sneezing and vigorous shaking of the head with a gurgling or rattling sound is characteristic. The sound sometimes resembles a whistle and such birds have been known as "callers." A blood-tinged exudate may be coughed up or shaken from the mouth. Mortality often is high. Effect on egg production is variable.

The main signs usually are confined to the respiratory tract and vary from free blood in the windpipe to a cheesy or blood-tinged membrane formation.

*Diagnosis:* This condition must be differentiated from Newcastle disease, infectious bronchitis and fowl pox. A tentative diagnosis sometimes can be made from the history and typical post-mortem lesions. Definite diagnosis can be made only by isolating the virus in chicken embryos or by inoculating susceptible and immunized birds with materials from suspected cases.

*Treatment and prevention:* Occurrence of this disease varies in different geographic locations in the United States. While the disease is endemic and frequently occurs in some areas, it is rare in Texas and is not a pressing problem for poultrymen of this state. In areas where the disease prevails, vaccination is necessary for prevention. In other areas where the disease does not occur often, vaccination is strongly discouraged. The standard vaccine should be used with care because it is a virulent virus. It is applied to the mucous membrane of the cloaca. Newer vaccines are milder and may be applied by the intraocular route. Vaccinated birds can become carriers of the infection. One vaccination gives good protection.

There is no treatment for this disease. When an outbreak occurs, vaccinate the flock immediately. This usually will stop the spread of infection among the group. Never use the vaccine for this purpose unless the diagnosis is definitely confirmed.

Reference must be called to the list of vaccines available. The restriction for the use of laryngotracheitis vaccine is outlined and should be strictly complied with for the good of Texas poultrymen.

### QUAIL BRONCHITIS

Quail bronchitis is a contagious, highly fatal disease in young quail. The virus causing this disease also infects chickens and turkeys. This agent also is known as CELO virus. It has been isolated from chicken eggs but does not produce a recognizable disease in chickens or turkeys. This

agent may play a part in respiratory diseases and in infertility problems, but its importance must be established by additional research. It is important because it is one of the agents that may be isolated from birds with respiratory symptoms and may be difficult to separate from other agents such as infectious bronchitis virus.

### INFLUENZA/PARAINFLUENZA

This virus disease or group of virus diseases affecting turkeys, ducks and quail must have more research and study to fully determine their economic importance to Texas poultrymen. To date, on the basis of limited investigation, influenza is not a problem in commercial turkey flocks in Texas. It must be pointed out, however, that this group of viruses could become a problem in the future.

### MYCOPLASMOSIS

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

### Chronic Respiratory Disease — Air Sac Syndrome and Infectious Sinusitis

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

The third condition, infectious sinusitis of turkeys, was recognized as early as 1905. It causes a sinus swelling under the eye as well as an inflammation of other respiratory organs. It is a chronic disease adversely affecting growth and feed conversion. In young poults it may cause significant mortality.

*Cause:* A peculiar bacterial organism known as *Mycoplasma gallisepticum* (Mg) is common to all three conditions. CRD, in a strict interpretation, is caused by a pure Mg infection. On the

other hand, the air sac syndrome has a complicated cause and is a result of infection with several organisms. This condition is caused by Mg in combination with another common bacterial organism, *E. coli*, and is triggered by an acute respiratory virus infection such as Newcastle disease or infectious bronchitis.

The cause of infectious sinusitis of turkeys is an uncomplicated Mg infection.

*Mycoplasma gallisepticum* is widespread and affects many species of birds. Until recently most, if not all, chicken flocks and about 40 percent of turkey flocks were infected. Eradication programs have reduced the incidence in recent years.

**Transmission:** The primary method by which Mg is spread is through the egg. Infected hens transmit organisms and the chick or poult is infected when it hatches. Organisms also may be transmitted by direct contact with infected or carrier birds and possibly by other unknown methods.

**Symptoms and lesions:** 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 often are cloudy and contain large amounts of exudate. There is often a film of exudate covering the liver as well as the heart muscle and heart sac. Affected birds become droopy, feed consumption decreases and there is a rapid loss of body weight.

Infectious sinusitis of 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 usually are present in the flock and frequently are present in the same bird.

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

**Treatment and prevention:** The treatment of CRD, air sac syndrome and the lower form of infectious sinusitis usually is not 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 either in the feed, drinking water or by injection. The "upper" form of infectious sinusitis can be treated with success by injecting antibiotics into the swollen sinus.

The answer to the Mg problem in both chickens and turkeys is eradication of the diseases. This goal has been achieved essentially in turkeys through voluntary programs conducted under The National and State Turkey Improvement Plans. Because all commercial chickens were infected until recently, obtaining Mg-free chicken flocks was more difficult. However, most foundation broiler breeder stock is now free of Mg, and many foundation breeders of commercial egg stock have Mg-free birds available.

### **Mycoplasma Meleagridis Infection**

*Mycoplasma meleagridis* (Mm) infection is a cause of airsacculitis in young turkey poults. As in the case of other avian *Mycoplasma* infections, the agent is egg transmitted, and the infected breeder must be considered the prime perpetuator of infection.

The true significance of Mm infection is not known. The air sac lesions associated with the egg transmitted disease are usually transitory in nature and disappear early in life, usually by the sixth or eighth week; and the performance of most infected birds does not appear to be impaired significantly. In recent years, however, Mm has been incriminated in an assortment of conditions including osteodystrophy and other skeletal deformities. There is no solid evidence to substantiate these claims.

The dipping of turkey hatching eggs in antibiotic solutions is being widely practiced. The practice is said to reduce the incidence of air sac lesions and to increase the livability and performance of poults produced by Mm-infected breeders. Breeders and hatcheries contemplating such procedures should seek expert advice before proceeding.

### **INFECTIOUS CORYZA**

Infectious coryza is a specific respiratory disease of chickens which occurs more often in semi-mature or adult birds. It is often confused with endemic fowl cholera. Infection may result in a slow spreading, chronic disease which affects only a small number of birds at one time or in a rapid spreading disease with a higher percentage of the birds affected. This variation in the spread of the infection apparently depends upon the virulence of the organism, certain management practices and other factors. The occurrence of infectious coryza is not widespread and the incidence is relatively low in Texas. However, the incidence appears to have increased in recent years in certain areas, where the infection has been introduced and has become endemic.

**Cause:** 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 or onto a farm. Transmission of the infection within the flock occurs by direct contact, airborne infective dust or droplets or drinking water contaminated with infective nasal exudate. Incubation period following experimental inoculation may be as short as 18 to 36 hours; however, susceptible birds exposed by contact to infected birds usually develop symptoms in 1 to 3 days. Individuals which have recovered from the disease may appear normal, yet remain carriers of the organism for long periods. Once a flock has been infected, each individual bird must be considered a carrier.

*Symptoms and lesions:* 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 around the eyes. 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:* 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. Birds should be submitted to a diagnostic laboratory early in the course of the infection, before complications of secondary bacterial infections occur and make recovery of the organism even more difficult.

*Prevention:* Prevention is the only sound approach in controlling infectious coryza. It usually can be prevented by management programs that prevent contact between susceptible and infected birds. It requires only separating affected or carrier birds from the susceptible population. In order to prevent the introduction of the infection, introduce started or adult birds only from sources known to be free of the infection. If infection does occur, complete depopulation is necessary for elimination of the disease and should be followed by thorough cleaning and disinfection of facilities and equipment.

Bacterins do not provide complete protection but may be useful in certain instances on infected farms where depopulation is not feasible.

*Treatment:* There are a number of drugs which have efficacy in treating this infection. Various antibiotics and sulfonamides are used. In the event sulfonamides are used, caution should be exercised in the administration since lengthy treatments or high drug levels can result in toxicity, a drop in egg production and mortality. One should consult

with diagnostic personnel concerning a recommended treatment regimen. Treatment does not eliminate the infection in carrier birds; therefore, dispose of affected flocks as soon as practical to eliminate them as a source of infection.

#### ENDEMIC (LOCALIZED) FOWL CHOLERA

This disease is very similar to infectious coryza. The two diseases are often confused even in textbooks on poultry disease and pictures of this disease are often mislabeled as infectious coryza. The disease is a specific respiratory infection caused by an organism similar to the one causing acute fowl cholera. This condition, however, is not associated with acute fowl cholera. It never reverts to the acute form of the disease but chronic cases following an outbreak of acute fowl cholera may show symptoms and lesions closely resembling this condition. The disease affects chickens primarily, although turkeys can be infected by inoculation.

Twenty years ago, it was estimated that 80 percent of the chicken flocks in Texas were infected. Today the disease occurs only rarely in commercial operations, having been largely eliminated by management programs.

*Cause:* The disease is caused by a bacterium belonging to the Pasteurella genus, and the organism cannot be differentiated by ordinary methods from *Pasteurella multocida* which causes acute fowl cholera.

The disease is transmitted only by direct contact between susceptible and infected birds. Recovered birds may remain carriers indefinitely. Separation between susceptible and carrier birds to prevent such contact will prevent the spread of the disease.

*Symptoms and lesions:* This is a chronic disease which affects only a small percent of the birds at one time. It causes nasal discharge, inflammation of the eye and swelling of the sinus under the eye. The sinus becomes filled with hard caseous-type exudate that has a characteristic odor. Sometimes the lungs and air sacs are affected. The disease causes little direct mortality but does result in poor performance, reduced egg production and an increase in cull birds.

*Diagnosis:* A trained diagnostician can recognize this infection by the history, symptoms and lesions. A confirmed diagnosis can be made only by the isolation and identification of the causative organism. This is sometimes difficult because of the chronic nature of the infection and the presence of secondary and complicating bacterial organisms.

*Prevention:* As with infectious coryza, this disease can be readily prevented by certain manage-

ment practices. It requires only separating affected or carrier birds from the susceptible population. In order to prevent the introduction of the infection, introduce started or adult birds only from sources known to be free of the infection. In the event of infection, complete depopulation is a necessity and should be followed by thorough cleaning and disinfection of facilities and equipment.

*Treatment:* In general, treatment is unsatisfactory. Various sulfonamides and antibiotics are used and lengthy treatments are usually necessary. Treatment only serves to prevent other birds from developing the disease and is not a cure for those already affected.

If sulfonamides are used, caution should be exercised in its administration since lengthy treatments on high drug levels can result in toxicity, a drop in egg production and mortality.

### PSITTACOSIS AND ORNITHOSIS

Psittacosis is an acute or chronic bacterial infection of psittacine birds (parrots, parakeets, lovebirds, etc.). The term *psitticosis* also is used in reference to the generalized infection of man when such infection is contracted from birds. The term ornithosis is reserved for infections of similar etiology in nonpsittacine birds (ducks, pigeons, turkeys, chickens, etc.). In domestic poultry production, the disease has been a problem only in turkeys. Its importance is magnified because it is transmissible to man. Workers in poultry processing plants have become infected as a consequence of handling the infected turkeys.

*Cause:* The disease is caused by agents now considered to be bacteria with the scientific name being *Chlamydia psittaci*. They are very specialized bacteria in that they need living cells in which to multiply. The pathogenicity of different isolates may vary with some producing a mild disease in turkeys and others causing significant mortality.

*Transmission:* It is not known how turkey flocks become infected. The disease is not egg transmitted and recovered birds do not appear to remain carriers. It is suspected, but not proved, that migratory shore and wading birds may introduce the infection. Many turkey flocks having the disease have been in contact with surface water frequented by such birds.

*Symptoms and lesions:* Infected turkeys become droopy, go off feed and usually have a greenish-yellow diarrhea. Symptoms can be confused with many other diseases. When a turkey that died from this infection is examined, the main findings are an inflammation of the heart sac resulting in an accumulation of exudate in this organ, cloudy

air sacs which may contain exudate and a film of clear exudate over the liver.

*Diagnosis:* Ornithosis in turkeys must be differentiated from infectious sinusitis, fowl cholera and some other diseases. Postmortem lesions are suggestive but a definite diagnosis can be made only by isolating the agent in chicken embryos or mice. Blood tests also may be useful in establishing a diagnosis.

*Prevention and treatment:* No specific methods of prevention can be given until more is known about transmission of the disease. Do not let turkeys have access to ponds, lakes or other bodies of surface water.

When ornithosis is suspected, obtain a definite laboratory diagnosis because of the public health aspect of the disease. Once the disease is diagnosed, quarantine the flock and give a 3-week supervised treatment with aureomycin at a rate of 200 gm/ton of ration. This treatment usually will stop flock losses and allow birds to be processed without danger of human infection.

### ASPERGILLOSIS (Brooder pneumonia)

Aspergillosis has been observed in almost all birds and animals including man. The disease is encountered in poultry in two main forms: (1) acute outbreaks with high morbidity and high mortality in young birds and (2) in adults as a chronic condition affecting individual birds. It is more of a problem in turkeys but also may affect chickens.

*Cause:* This condition is caused by *Aspergillus fumigatus*, a mold or fungus-type organism. Occasionally, other types of molds are involved. These organisms are present in the environment of all poultry. They grow readily on many substances such as litter, feed, rotted wood and other similar materials.

*Transmission:* The bird comes in contact with the organisms through contaminated feed, litter or premises. The disease is not contagious and does not spread from one bird to another. Host healthy birds can withstand repeated exposure to these organisms. Inhalation of large numbers of the infectious stage of the mold or reduced resistance apparently results in infection. In adult turkeys, the disease more often affects the male.

*Symptoms and lesions:* In the acute form in young birds, main symptoms are gasping, sleepiness, loss of appetite and sometimes convulsions and death. Occasionally the organism invades the brain, causing paralysis or other forms of nervous symptoms. The more chronic form in older birds usually

results in the loss of appetite, gasping or coughing and a rapid loss of body weight. Mortality is usually low and only a few individual birds are affected at one time.

The disease produces hard nodular areas in the lungs and an infection of the air sacs. Sometimes the air sac lesions are similar to those produced by infectious sinusitis or CRD. In some birds, colonies of mold growth can be seen on the air sac membranes.

*Diagnosis:* Diagnosis usually can be made from history, symptoms and lesions. Sometimes it is necessary to base diagnosis on microscopic lesions.

*Treatment and prevention:* There is no treatment for the flock or the affected bird. The disease usually can be prevented by avoiding moldy litter, feed or premises. A careful examination of the environment usually reveals the trouble source which should be eliminated. Often this means replacing the litter.



## Section 4.

### VIRAL DISEASES (EXCLUSIVE OF RESPIRATORY DISEASES)

A number of viral diseases of poultry produce symptoms and lesions primarily exclusive of the respiratory system. Among them are some of the most devastating diseases of chickens and turkeys. Considered in this group are avian pox, leukosis, avian encephalomyelitis (epidemic tremor) and bluecomb disease.

#### AVIAN POX (Fowl pox, Canker, Avian diphtheria)

Avian pox is a relatively slow spreading viral infection of birds, characterized by wart-like nodules on the skin and diphtheritic necrotic membranes lining the oral cavity and upper respiratory system. It has been present in birds since the earliest available history, is universal in distribution and may cause severe economic loss in chickens and turkeys due to poor growth, feed efficiency, reduced production, increased cull rates and downgrading. Mortality usually is not significant unless the respiratory involvement is marked. The disease may occur in any age bird at any time during the warm months, particularly when mosquito populations are high.

*Cause:* Avian pox is caused by a viral agent. There are at least three different strains or types of avian pox virus: fowl pox virus, pigeon pox virus and canary pox virus. Although some workers include turkey pox virus as another distinct strain, many feel that it is identical to fowl pox virus.

Each virus strain is infective for a number of species of birds in addition to its primary host. For example, among others, fowl pox virus may infect chickens, turkeys, pheasants, quail and ducks; pigeon pox virus may infect pigeons, chickens and turkeys; and canary pox virus may infect canaries, chickens, pigeons and sparrows.

Natural occurring pox in chickens, turkeys and other domestic fowl is considered to be caused by fowl pox virus.

*Transmission:* Fowl pox can be transmitted by direct or indirect contact. The virus is highly resistant in dried scabs, and under certain conditions may survive for months on contaminated premises. The disease may be transmitted by a number of species of mosquitoes, this being the usual manner by which the infection is introduced to a premise. Mosquitoes may harbor infective virus for a month or more after feeding on affected birds. After the infection is introduced, it spreads within the flock

by mosquitoes as well as by direct and indirect contact. Recovered birds do not remain carriers.

*Symptoms and lesions:* Since fowl pox usually spreads slowly, a flock may be affected for several months. The course of the disease in the individual bird is 3 to 5 weeks. Affected young birds are retarded in growth. Adult birds drop in production. Birds of all ages which have oral or respiratory system involvement have difficulty in eating and breathing.

The disease manifests itself in one or two ways.

*Cutaneous or dry pox:* Lesions start as small whitish foci which develop into wart-like nodules. The nodules eventually are sloughed and scab formation precedes final healing. Lesions are seen most commonly around the featherless facial parts (comb, wattles, ear lobes and eyes) but may be found on the body.

*Diphtheritic or wet pox:* Lesions are associated with the oral cavity and the upper respiratory tract, particularly the larynx and trachea. The lesions are diphtheritic in character and involve the mucous membranes to such a degree that when removed, an ulcerated or eroded area is left.

*Diagnosis:* Fowl pox is readily diagnosed on the basis of flock history and presence of typical lesions. In some instances, laboratory diagnosis by tissue or transmission studies is necessary.

*Treatment and prevention:* There is no treatment for fowl pox. Disease control is accomplished best by preventive vaccination since ordinary management or sanitation practices will not prevent it. Several kinds of vaccines are available: pigeon pox, pigeon pox-like and fowl pox vaccine. The pigeon pox vaccine is of questionable value and its use is not usually recommended. The pigeon pox-like vaccines are newer and have wider acceptance. Fowl pox vaccine is an efficient product and is the vaccine in common use in Texas. Its use varies according to the type of operation, but generally the following recommendations apply:

*Broilers:* Vaccination usually is not required; but in some areas where the mosquito population is high, as in parts of Texas, it may be necessary to prevent the disease. In such instances, the vaccine is applied to chicks (as young as 1 day) using the wing-web method but using only one applicator needle.

*Replacement birds:* Vaccinate all replacement chickens against fowl pox. One application of fowl

pox vaccine results in permanent immunity. Birds can be vaccinated at any convenient time during the growing period, usually between 6 and 10 weeks of age.

*Turkeys:* Fowl pox vaccine does not produce lasting immunity in turkeys. Vaccinate turkeys when they are between 4 and 10 weeks of age. Turkeys to be retained as breeders should be re-vaccinated as adults. This usually is done as the breeding flock is selected. Birds not selected and vaccinated should be marketed within a day or two.

Examine vaccinated birds for "takes" about 7 to 10 days following vaccination. A high percentage showing a reaction indicates a satisfactory vaccination.

### AVIAN LEUKOSIS

The diseases which make up the avian leukosis complex are transmissible virus diseases of birds characterized by tumor formations. The diseases are widespread and have been the most devastating of those affecting mature laying chickens, and in recent years have become increasingly significant as a cause of losses in broilers and growing birds.

Of the separate and distinct diseases which form the complex, *lymphoid leukosis* and *Marek's disease* produce the most losses.

#### Lymphoid Leukosis (L.L.)

Characteristically, lymphoid leukosis is a disease of adult chickens; however, the disease appears to be of increasing importance in turkeys and other species, e.g. the pheasant. Although the virus of lymphoid leukosis may produce various responses (e.g. blood forms—erythroblastosis and myeloblastosis; bone forms—osteopetrosis), the lymphoid tumor response is the most common.

*Cause and transmission:* Lymphoid leukosis is caused by a group of enveloped RNA viruses which closely resemble those of the myxovirus group. The disease is transmitted in a number of ways. The agent is eliminated naturally from the body of the infected bird via eggs and feces. The virus may be transmitted mechanically from infected birds to susceptibles by blood-sucking parasites or by man in such procedures as fowl pox vaccination.

Most infections are acquired during the first few weeks of life. This suggests that most flocks acquire the disease by egg transmission or by direct or indirect contact with older infected birds during the early brooding period.

*Manifestations of disease:* Lymphoid leukosis is characterized by the formation of lymphoid tumors, particularly in the liver and spleen. Affected

birds may die without preliminary symptoms, but the disease is usually chronic in nature with affected birds showing loss of appetite, progressive emaciation and diarrhea. Clinically affected birds invariably die. Although losses due to the disease may be most severe shortly after the onset of production, losses in the affected flocks will continue as long as it is retained and may total 20 percent or more during the productive life of the flock.

Autopsies of affected birds reveal tumors. Although the liver and the spleen are commonly involved, other visceral organs may be affected. The neoplastic process may be diffused involving 100 percent of the affected organ, or it may be a nodular type. Affected structures, the liver in particular, may be greatly enlarged.

Osteopetrosis is the bone form of the disease. Until recently it was thought to be a disease primarily of older birds, particularly males; however, it is now known to be quite common in young chickens and is one of the more serious causes of broiler condemnations. The disease is characterized by a thickening and deformation of bone, the long bones in particular. This frequently results in lameness and faulty body conformation.

Blood forms of L.L. are diagnosed infrequently.

*Diagnosis:* The clinical diagnosis of L.L. is based upon flock history and disease manifestations. The lymphoid disease cannot be readily distinguished from the visceral response to Marek's disease (M.D.); however, there are some features which aid in differential diagnosis. Some of these features are outlined in the following table.

#### DIFFERENTIAL DIAGNOSIS OF L.L. AND M.D.

Feature	Lymphoid Leukosis	Marek's Disease
Incubation period	Prolonged	Short
Usual age incidence	Over 6 months	Under 6 months
Visceral lesions	Most commonly liver and spleen	Generalized
Skin lesions	No	Yes
Ocular lesions	No	Yes
Neural lesions	No	Yes

It should be kept in mind that the clinical differentiation of L.L. and M.D. is often presumptive at best. Histopathological differentiation is more accurate, but even it is not completely so.

*Treatment and prevention:* There is no treatment for lymphoid leukosis. Although the disease cannot be prevented completely, there are certain steps which can be taken to help control the level of infection in a flock. Some of these are listed below.

Buy resistant strains of birds. Most reputable breeders have invested a great deal of time and money breeding for L.L. resistance.

Brood in isolation. Most L.L. is acquired early (under 6 weeks of age). If replacement birds are brooded in strict isolation, contact transmission (direct and indirect) from adult carriers will be minimal.

Keep incubator sanitary.

Control blood-sucking parasites.

Do not use gimmicks in disease control (e.g. explosive outbreaks of L.L. have occurred following the indiscriminate use of turkey blood in M.D. "control" programs).

#### **Marek's Disease (M.D.)**

Marek's disease is characteristically a disease of young chickens; however, the disease is commonly seen in adult birds. In contrast to L.L., the tumor response of M.D. is limited to that of the lymphoid type. However, the response may be much more diverse in location than that usually seen in lymphoid leukosis.

*Cause and transmission:* Marek's disease is caused by a virus belonging to the Herpesvirus group. Much is unknown about the transmission of the virus; however, it appears that virus is concentrated in the feather follicles of affected birds and is shed in dander. The virus apparently has a long survival time in dander and viable virus may be demonstrated in depopulated houses months after infected birds have been removed. The usual mode of transmission is by aerosols containing infected dander and dust. As is the case with L.L., young birds are most susceptible to infection with M.D.; however, since the incubation period of M.D. is short, clinical disease can appear much earlier than is the case with lymphoid leukosis.

*Manifestations of disease:* Marek's disease may produce a variety of clinical responses, all lymphoid in character. These are acute visceral, neural, ocular and skin; or, as is commonly the case, a combination of the responses may be seen.

Marek's disease of the *visceral type* may be characterized by widespread involvement. The lesions are most commonly associated with the gonads (testes or ovaries), liver, spleen and kidney; however, other organs such as the lungs, heart and musculature are commonly involved. The disease is often acute in nature, with apparently healthy birds dying very rapidly and having massive internal tumors. The disease may appear in broiler-age birds and be a significant cause of death loss and condemnations. More commonly the disease produces the most severe losses in replacement pullets near or at the onset of production. At this time, the disorder is seen frequently in birds with acute coccidiosis leading some to suspect that there is a relationship between the two diseases. Presently,

the only relationship is considered to be that of a bird with coccidiosis or M.D., whichever the case, being more susceptible to the other disease.

Marek's disease of the neural type is the classical type of the disease. *Neural leukosis* was the first disease known as M.D. before the etiologic relationships of the various diseases in the leukosis complex were established. Neural leukosis is characterized by a progressive paralysis of the wings, legs and neck. Loss of body weight, anemia, labored respiration and diarrhea are common symptoms. When affected birds are autopsied, lesions, if observed in uncomplicated cases, are confined to the nerve trunks and plexuses innervating the paralyzed extremities. Affected nerve tissue is swollen as a result of an accumulation of lymphocytes and tissue fluids. Frequently no gross lesions are observed.

*Ocular leukosis* (gray eye) is responsible for much of the blindness in chickens. This type of M.D. usually is seen in early maturity. Morbidity and subsequent mortality are usually low but in some instances approach 15 to 25 percent. Ocular leukosis is characterized by spotty depigmentation or diffuse graying of the iris of the eye caused from lymphocytic infiltrations. The pupil develops an irregular shape and fails to accommodate light. Emaciation, diarrhea and death usually follow because of partial to complete blindness.

*Skin leukosis* is the form of M.D. that produces the most severe losses in broilers. Losses are more commonly due to condemnation at processing time. The disorder is characterized by enlargement of the feather follicles due to accumulations of lymphocytes. As has been stated, most infective virus is produced in the regions of the feather follicle and is shed with skin dander.

*Course of disease:* Acute M.D. can be extremely rapid in its course producing mortality in apparently healthy birds. However, it has been demonstrated that the lesions of M.D., particularly of skin leukosis, may regress and clinically affected birds may make complete recoveries.

*Diagnosis:* The clinical diagnosis of M.D. is based upon flock history and disease manifestations (refer to table under the diagnosis of lymphoid leukosis). Accurate diagnosis may depend on the employment of sophisticated laboratory procedures.

*Treatment and prevention:* As is the case with L.L., there is no treatment for Marek's disease and until recently, there have been no effective preventive measures.

A vaccine is now available which appears to be extremely effective (90 percent) in the prevention of M.D. The vaccine is made with a Herpes-



virus of turkeys (H.V.T.) that prevents the virus of M.D. from transforming cells to produce tumors. The vaccine is quite expensive and therefore is used principally in replacement layers and breeders. The vaccine should be applied in strict accordance with the manufacturer's recommendations.

Prevention of the disease in market broilers by vaccination is not economically feasible at this time. Different empiric approaches to control have been attempted, but results have been erratic. A program, still in experimental stages, that may have merit in the future is the use of filtered air positive pressure (F.A.P.P.) housing to start broiler chicks. Chicks started in such a controlled environment can be maintained free of exposure to M.D. virus during the first few weeks of life. When transferred to conventional housing at an age of several weeks, the chickens may be exposed to M.D. virus; but disease does not have sufficient time to develop before marketing.

Other approaches to control include the genetic and management/sanitation.

#### **EPIDEMIC TREMOR (Avian Encephalomyelitis)**

Avian encephalomyelitis is a viral infection which affects chickens of all ages, but usually produces clinical manifestations only in young birds. In recent years, the disease also has been observed in turkeys. Signs of infection include incoordination, nervousness, a jerky or irregular gait, falling over on the side with outstretched wing and muscular tremors that are especially noticeable in the head and neck. The commonly used term *epidemic tremor* is misleading because muscular tremors are not evident in many otherwise typical outbreaks.

This disease, reported first in New England in 1932, now exists in all poultry-producing areas of the United States and has been reported in several other countries.

The custom of hatcheries to adjust for losses due to epidemic tremor has led other segments of the poultry industry to regard the disease as of minor importance and significant primarily to hatcherymen. Such losses are costly, troublesome and reduce efficiency in all operations, especially the breeder and broiler flocks. Thus, epidemic tremor poses a major problem for the entire poultry industry.

*Cause:* Epidemic tremor is caused by a relatively small virus which produces microscopic lesions in the bird's nervous system.

*Transmission:* The virus is transmitted through eggs of infected parent flocks. Such outbreaks in parent flocks often are unnoticed and usually last

21 to 30 days. It appears that affected flocks do not remain carriers and are not susceptible to the disease again for a reasonable time; consequently, such flocks are desirable as hatchery supply flocks.

Apparently, other modes of transmission are responsible for outbreaks in production flocks. The disease can be transmitted by direct or indirect contact, but this is not of major significance.

*Symptoms and lesions:* In a small percentage of outbreaks, the disease may be suspected because of poor hatchability or morbidity in birds at hatching time. The incubation period varies from 5 to 40 days with an average of 9 to 21 days. The typical outbreak becomes noticeable when birds are 17 to 21 days old. Some individuals in flocks exposed during hatching may develop clinical evidence of infection up to 7 weeks later. Morbidity rates vary from only a few individuals to 30 percent but average 5 to 10 percent.

Outbreaks in young chicks are characterized by an inability to walk normally or they become paralyzed and lie propped on one wing. Visible trembling of the head and neck may be present, but is not apparent in many outbreaks. Affected birds usually do not recover, but they will survive for long periods if food and water are provided. New cases developing after the fifth or sixth week are rare. Mortality usually is negligible but visibly affected individuals should be removed and destroyed.

No lesion is visible with the naked eye. Microscopic lesions are widespread and are a diagnostic aid.

In adult flocks, there may be no evidence of infection other than a 5 to 10 percent drop in egg production, with a decrease in hatchability. Most outbreaks in adult flocks are not suspected unless the caretaker is a keen observer and keeps good records.

*Diagnosis:* The disease usually is diagnosed on the basis of case history and typical signs. Atypical cases present diagnostic problems and every reliable aid must be used to make an accurate diagnosis.

*Prevention and treatment:* There is no treatment. Remove and kill all birds showing clinical evidence of the disease, since they do not develop into profitable birds.

The disease is readily prevented by vaccinating breeder replacements prior to the onset of production. Vaccine is applied in the drinking water when the birds are approximately 10 to 14 weeks old. The vaccination of breeder stock in this way prevents subsequent infection and egg transmission of the virus.

## INFECTIOUS BURSAL DISEASE (Gumboro)

Infectious bursal disease (I.B.D.), commonly referred to as Gumboro disease, is an acute, highly contagious viral disease of young chickens. It is found most often in the highly concentrated poultry-producing areas of the state. It causes marked morbidity and mortality in affected flocks but usually disappears after 1 to 2 weeks.

*Cause:* A virus or virus-like agent referred to as the infectious bursal agent causes this disease.

*Transmission:* The transmission or spread of the disease can occur by direct contact (bird to bird), contaminated litter and feces, caretaker, contaminated air, equipment, feed, servicemen and possibly insects and wild birds. It is extremely contagious.

*Symptoms and lesions:* Birds have ruffled feathers, a slight tremor at onset of the disease, strained defecation, loss of appetite and are dehydrated. Affected birds have a tendency to sit, and when made to move have an unsteady gait. Vent picking is common. Early in the disease, there is a rise in body temperature but it soon becomes subnormal. This is often followed by prostration and death. The litter of contaminated houses becomes sticky during the course of the disease.

Postmortem lesions include dehydration and changes in bursa, skeletal muscle, liver and kidney. All affected birds have some bursal changes commonly characterized by swelling, change in shape (oblong), color (pink, yellow, red, black) and the formation of a gelatinous film around the bursa.

*Diagnosis:* The diagnosis of I.B.D. is usually based on flock history and postmortem lesions. Laboratory procedures may be used to substantiate the diagnosis.

*Treatment and prevention:* There is no specific treatment for I.B.D. In fact, indiscriminate medication with certain drugs (e.g. the sulfonamides) may severely aggravate mortality. Supportive measures, such as increasing heat and ventilation and increasing water consumption, are beneficial.

Management and sanitation practices alone cannot be relied upon to prevent the occurrence of the disease.

Early exposure (before 14 days of age) to the agent of I.B.D. apparently acts as an immunization procedure, and many flocks that are exposed and infected do not develop noticeable disease symptoms.

Vaccines are available but must be given under the supervision of a poultry pathologist or regulatory official. If given correctly, good control can be obtained.

## Section 5.

### DISEASES OF UNDETERMINED CAUSE

The causes of some disease conditions have not been established. Some of the diseases in this group have the characteristics of infectious diseases, but many of them appear to be associated with disturbances in nutrition or metabolism.

#### TRANSMISSIBLE ENTERITIS OF TURKEYS (Bluecomb)

Transmissible enteritis is an acute to chronic disease of turkeys characterized by sudden onsets, marked depression and severe diarrhea. Death losses may be high, particularly in young poults; but heaviest losses in adults are due to loss of condition. The disease at one time was considered to be the same as so-called "bluecomb" of chickens. Now it is recognized as a distinct entity.

*Cause:* The etiologic agent of turkey bluecomb is considered to be a virus. Substantial evidence exists; however, the disease as seen in the field is the result of an interaction of several agents, among which the virus is considered to be primary.

*Transmission:* The disease spreads by contact with infected birds or premises or droppings of infected birds being especially rich in virus. No specific environmental factors appear to influence the occurrence.

*Symptoms and lesions:* When the disease strikes young poults under 3 or 4 weeks of age, onset is sudden. Affected poults appear cold and seek heat. Feed and water consumption drops markedly and poults lose weight rapidly. Morbidity and mortality may approach 100 percent in uncontrolled outbreaks.

Young poults show few lesions other than those associated with the intestinal tract. Intestines usually are distended and lack muscle tone. Intestinal contents are fluid and gaseous (foamy).

Morbidity is variable in older flocks of turkeys. It may be extremely low in some flocks but extremely high in others. Feed intake drops markedly and birds may lose up to 4 to 5 pounds of body weight in just a few days. Birds usually have profuse diarrhea. Cyanosis of the head parts is common.

When older birds are autopsied, the following lesions may be seen. The body musculature is dehydrated. Minute hemorrhages may be seen on the viscera and necrotic foci on the liver. Kidneys are commonly swollen and contain an excess of

urates. Severe catarrhal enteritis is seen often and mucous casts may be present. The pancreas usually presents multiple chalky white areas. The crop frequently is distended and contains sour smelling contents.

*Diagnosis:* Transmissible enteritis must be differentiated from common bacterial infections such as paratyphoid, fowl cholera, fowl typhoid and erysipelas. Diagnosis usually is based on history, symptoms, lesions and negative bacteriological findings for the common bacterial infections.

*Treatment and prevention:* Older turkeys can be flushed with molasses at the rate of 1 pint molasses to 5 gallons of drinking water for 1 day. Then give antibiotics in feed or drinking water at the rate of at least 200 grams/ton of feed or 200 to 400 milligrams/gallon of water. Continue treatment for at least 5 to 7 days.

Do not flush young turkey poults. Give antibiotics at the rate of up to 400 grams/ton of feed or 1 gram/gallon of drinking water. Give this high level for 2 to 3 days, after which time antibiotics may be reduced, depending on flock response. Total treatment period should be at least 5 to 7 days.

Until more is known about the spread of bluecomb, no specific recommendations for prevention can be made. However, consider recovered birds as potential carriers. Clean and disinfect houses in which outbreaks have occurred. Leave vacant for at least 30 days. Apply routine management and sanitation practices for disease prevention.

#### HEMORRHAGIC ANEMIA SYNDROME (Hemorrhagic Disease: Aplastic Anemia)

Hemorrhagic anemia syndrome is a disease characterized by hemorrhage and anemia. It is considered to be a disease of chickens only, although poorly substantiated reports indicate the occurrence of a similar disease of turkeys. The condition may affect birds of all ages, but usually affects those between the ages of 4 and 12 weeks. Economic loss results from mortality and retarded growth.

*Cause:* The cause of hemorrhagic anemia syndrome has not been determined, but it is not considered infectious.

*Symptoms and lesions:* Usual signs are ruffled feathers, weakness, loss of body weight, diarrhea and anemia. Morbidity usually is high, but mortality is extremely variable, depending upon



whether the disease is acute or chronic. Acutely affected birds may die with few preliminary symptoms. Flock mortality may approach 20 to 30 percent although it usually is lower.

Lesions vary from anemia to frank hemorrhages. Hemorrhages may be found anywhere on or in the body. Usually they are found in the musculature, particularly of the thighs and breast. Hemorrhages are commonly petechia (pinpoint in size), but they may be diffuse involving large areas. Hemorrhages often are seen in the wall of the intestines, the proventriculus, the musculature of the gizzard and the heart musculature. Less often there may be hemorrhage into the anterior chamber of the eye and into the wattles. Anemia is characterized by paleness of the comb, mucous membranes and other tissues. Commonly, the bone marrow appears pale yellow and fatty (aplastic anemia).

*Diagnosis:* Diagnosis is based on history, symptoms and lesions. When intestinal lesions are present, care must be taken to differentiate from coccidiosis.

*Treatment:* There is no specific treatment for hemorrhagic anemia syndrome, but good response frequently is obtained by adding liver solubles to feed at a level of 8 gallons per ton of feed for 5 days, followed by a level of 5 gallons per ton for an additional 5 to 7 days.

Avoid sulfa drugs and high levels of antibiotics since they may aggravate the condition.

### AORTIC RUPTURE

Aortic rupture is a disease of turkeys characterized by rapid onset and immediate death due to internal hemorrhage. It seldom is observed in other birds, but may occasionally affect chickens. Male turkeys, usually the most rapidly growing birds in the flock, are affected most frequently. The disease usually appears in growing birds between the ages of 8 and 20 weeks, although older birds may be affected.

*Cause:* The cause has not been determined. High energy intake during rapid growth appears to be related to occurrence. Deposition of fatty substances in the blood vessel walls weaken the vessels, making them more subject to rupture. Subsequent increases in blood pressure, common in adolescent male turkeys, produce the actual rupture.

*Transmission:* Aortic rupture is not infectious and is not transmitted from one bird to another.

*Symptoms and lesions:* Seldom are preliminary symptoms observed. Affected birds usually are found dead. Occasionally, an apparently healthy

bird drops to the ground in terminal convulsions and dies within minutes. Daily losses are low, but total losses may approach 10 percent or more in serious outbreaks.

Autopsies on affected birds reveal massive amounts of free blood in the body cavities. The site of aorta rupture usually is in the kidney region but may be anywhere posterior to the aorta origin at the heart.

*Diagnosis:* Diagnosis is based on lesions.

*Treatment and prevention:* Losses can be reduced by limiting energy intake. Tranquilizers, such as reserpine, are also of value.

Prevention is accomplished best by limiting energy intake or by continuous low level feeding of tranquilizers during the critical 12- to 20-week-old period.

### CAGE FATIGUE

#### (Cage Layer Fatigue, Cage Layer Paralysis)

Cage fatigue is a paralytic condition observed in birds held in cages. The disease is most common among high-producing young pullets during summer. It was prevalent during the late 1950's, but it is now seen infrequently. The decrease probably is due to dietary changes in the last few years.

*Cause:* The exact cause is not understood; however, the disorder is considered to be a disturbance in mineral metabolism.

*Symptoms and lesions:* Affected birds are paralyzed but they will continue to eat and drink if feed and water are within reach. Many birds lay on the day paralysis develops and some may continue to lay for a day or two after becoming paralyzed. Shell quality remains good. Morbidity usually is low but may approach 20 percent. Bones of affected birds are extremely fragile and are broken easily when the birds are handled routinely. The walls of the long bone are thin due to erosion of bone from the interior.

*Diagnosis:* Diagnosis is based on history, symptoms and lesions. Other causes of paralysis such as neural leukosis should be ruled out.

*Treatment:* Although impractical in most operations, affected birds usually make a spontaneous recovery if placed on the floor or on a cage bottom covered with newspaper or other material. Proper ration fortification usually prevents the disorder.

### FATTY LIVER SYNDROME

Fatty liver syndrome is characterized by deranged fat metabolism resulting in the deposition of excess fat in the liver and body cavities. It is seen most

commonly in caged birds, but on occasion may strike floor birds, particularly in heavy breeds.

*Cause:* The cause is unknown. Factors which predispose the condition, however, include reduced activity as in cage operations and use of high-energy feeds.

*Symptoms and lesions:* Affected birds usually experience a drop in egg production. They may be anemic and occasionally may be found dead without preliminary symptoms. Diarrhea, though common in caged birds, especially is pronounced in birds with fatty liver syndrome.

Postmortem findings are characteristic, but vary with severity. Livers of affected birds are pale, yellow and extremely friable. The livers may have subcapsular hemorrhages varying in size from pinpoint to massive. Deaths associated with fatty liver syndrome usually are due to hemorrhage from spontaneously ruptured livers. Fat content of the liver may be 55 to 70 percent on a dry-weight basis, as compared to 5 to 10 percent for normal birds. Deposition of excess abdominal fat is also a common finding; such fat usually is extremely liquid in nature.

*Diagnosis:* Diagnosis is based on history, symptoms and lesions.

*Treatment:* The treatment most likely to effect a favorable response is to add 500 grams of choline, 12 milligrams of vitamin B<sub>12</sub>, 5,000 to 10,000 units of vitamin E and 500 mg of Inositol per ton of feed. The reduction of energy by 50 calories per pound of feed and adjustment of protein level to at least 19 percent is effective in treatment as well as prevention. These modified rations must be fed for an indefinite period.

#### **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 in young quail or in pullets being raised for egg production.

*Cause:* The cause of the disease is not definitely known, but it is probably caused by a bacterial organism.

*Transmission:* Infection spreads by the droppings of sick or carrier birds. The disease organism is very resistant to disinfectants and will persist under varying environmental conditions (e.g. hot, cold, moist or dry).

*Symptoms and lesions:* Birds with the acute form may die suddenly while in good flesh, whereas the most chronically affected birds will become list-

less, have ruffled feathers and develop a humped-up attitude. Such birds will die in an extremely emaciated state.

The droppings may be confused with those of birds with coccidiosis. These two diseases often are seen in the same bird. The droppings of birds with only ulcerative enteritis will 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.

*Diagnosis:* Although the disease is characteristic in nature, anyone suspecting the infection should seek professional confirmation before treatment is started.

*Treatment and prevention:* Streptomycin and bacitracin are effective drugs in the treatment and prevention of this disease. If streptomycin is used, it should be incorporated into the drinking water at a level of 5 grams per gallon for 5 to 10 days, followed by 1 gram per gallon for an additional 5 days. If bacitracin is used, it should be incorporated into the feed at a level of 200 grams per ton and fed over a period of 2 weeks.

Raising birds on wire is an effective preventive measure. Bacitracin, fed at a rate of 20 grams per ton of feed, is an effective prevention in problem operations where the use of wire is impractical.

#### **NECROTIC ENTERITIS**

Necrotic enteritis is an acute disease, particularly of broiler type chickens, that is characterized by marked destruction of the intestinal lining. Common field names applied to the condition are quite descriptive, (e.g. rot gut, crud and cauliflower gut).

*Cause:* The cause of the disease is not well defined. Evidence indicates that bacterial organisms and their toxins are primary but that coccidiosis is a contributing factor. Most of the damage to the intestinal lining is apparently due to toxins produced by the bacterial organisms.

*Transmission:* Little is known about the spread of necrotic enteritis, but in all likelihood, transmission takes place by oral contact with the droppings of infected birds.

*Nature of the disease:* Necrotic enteritis appears very suddenly in the affected flock. Birds which are apparently healthy may become acutely depressed and die within hours. Mortality in affected flocks is usually between 2 and 10 percent, but it

may be as high as 30 percent in severe outbreaks. Losses due to retarded growth and poor feed conversion may be more costly than mortality in many outbreaks.

The 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 presence of a diphtheritic cauliflower-like membrane is characteristic.

*Diagnosis:* Diagnosis is based upon history, symptoms and the findings of characteristic lesions.

*Treatment and prevention:* Bacitracin is an effective treatment and is given for 72 to 96 hours at a level of 100 grams per ton of feed or at a

level of 10 to 25 grams per 50 gallons of drinking water. Streptomycin is also effective if applied for 72 hours at a level of 5 to 15 grams per 50 gallons of drinking water.

Supportive vitamin treatment may increase response.

Preventive medication may be of value on some premises where prior experience with the disease dictates the need. Bacitracin (25 grams/ton of feed) may be used during the first 6 weeks for this purpose.

Since coccidiosis may be a contributing factor, attention should be given to the coccidiosis control program.



## Section 6.

### PROTOZOAN DISEASES

Protozoa are the smallest members of the animal kingdom. Although many microscopic protozoan organisms are harmless, others can produce severe disease. This section includes five of the more common and serious poultry diseases caused by these organisms.

#### COCCIDIOSIS

Coccidiosis is a protozoan disease of fowl characterized by diarrhea, unthriftiness and variable mortality. It is a problem in all poultry-producing areas. Despite recent advances in control and treatment, the disease remains one of the principal causes of economic loss to the poultry industry.

*Cause:* Coccidiosis is caused by minute, microscopic animal forms called coccidia. There are a number of species of coccidia, each of which produces a distinct disease process. Following an outbreak of coccidiosis, a flock will be protected against subsequent exposure to the species which produced the outbreak, but it will remain susceptible to other species. This means a given flock may have several outbreaks of coccidiosis, depending on the number of coccidia species in an area and exposure to them.

The species of coccidia affecting chickens and turkeys are:

Chickens	Turkeys
<i>Eimeria tenella</i> *	<i>Eimeria adenoides</i> *
<i>Eimeria necatrix</i> *	<i>Eimeria meleagridis</i> *
<i>Eimeria acervulina</i> *	<i>Eimeria gallopavonis</i> *
<i>Eimeria brunetti</i> *	<i>Eimeria meleagridis</i>
<i>Eimeria maxima</i> *	<i>Eimeria dispersa</i>
<i>Eimeria mivati</i> *	<i>Eimeria innocua</i>
<i>Eimeria mitis</i>	<i>Eimeria subrotunda</i>
<i>Eimeria hagani</i>	
<i>Eimeria praecox</i>	

\*Considered the major causes of clinical outbreaks.

*Transmission:* Coccidiosis is transmitted by direct or indirect contact with droppings of infected birds. When a bird ingests coccidia oocysts, the organisms invade the intestinal tract lining where they produce tissue damage while undergoing reproduction. Within a week after ingesting coccidia oocysts, an infected bird will shed descendant coccidia in its droppings. So great is the reproductive potential that a single organism may produce about a million descendants. This means that contamination can build up rapidly, even in a new house.

Coccidia shed in droppings are incapable of infecting other birds until certain maturation changes (sporulation) take place. These changes occur in 24 to 72 hours if the litter is warm and damp.

The number of infective coccidia that a bird eats determines whether an infection will be mild enough to go unnoticed or severe enough to cause visible illness.

Coccidia are extremely hardy and may survive for long periods outside of the bird's body. They are transmitted easily from one house or premise to another by such things as dirty boots, free-flying birds, feed sacks and equipment.

*Symptoms and lesions:* Coccidiosis usually occurs in growing birds and young adults. It seldom is seen in birds under 3 weeks of age unless they are brooded on contaminated litter. Old birds usually are immune because exposure during early life is difficult to avoid.

Signs of a coccidiosis outbreak are usually general. Affected birds become pale and droopy, tend to huddle, consume less feed and water, have diarrhea and may become emaciated and dehydrated. Laying birds will experience a drop in production.

Cecal coccidiosis of chickens, caused by *E. tenella*, is often acute and characterized by bloody droppings, severe anemia and high mortality. In turkeys, cecal coccidiosis, caused by *E. adenoides*, also is often acute, producing high mortality but seldom bloody droppings.

Intestinal coccidiosis may be acute, but more frequently is chronic in nature. Droppings of affected birds are usually tan and watery, although acute *E. necatrix* infection may produce considerable hemorrhage. Since there is a slower buildup of infection of intestinal coccidia, intestinal coccidiosis usually occurs in birds in the latter part of the growing period or in early production. Mortality usually is not significant unless acute *E. necatrix*, *E. brunetti* or *E. maxima* infection is present.

Autopsies of birds with coccidiosis reveal lesions that vary, depending upon type of coccidiosis present and severity and stage of the disease.

Chickens in the acute phase of cecal coccidiosis will have ballooned cecal pouches full of free blood. In the recovery stage, cheesy cores tinged with variable amounts of blood will be present in the cecal pouches. Free blood usually is not found

in the ceca of turkeys with cecal coccidiosis, but the ceca contains a white-to-gray, semi-gelatinous material resembling cottage cheese in consistency.

Lesions of intestinal coccidiosis vary from a rather mild enteritis to a severe necrotic/hemorrhagic type of enteritis.

**Diagnosis:** Cecal coccidiosis may be confused with blackhead and salmonellosis, both of which may produce similar cecal lesions. Intestinal coccidiosis may be confused with hemorrhagic anemia syndrome and other diseases characterized by enteritis. Establish definite diagnosis by laboratory means so that medication can be specific. Do this by microscopic examination of intestinal or cecal scrapings to demonstrate the presence or absence of coccidial organisms. Since most healthy birds possess a few organisms, it is necessary to correlate microscopic findings with flock history and autopsy lesions before making diagnosis and recommendations.

**Treatment and prevention:** There are a number of drugs which are quite effective in the treatment of coccidiosis. Drug selection is dependent upon many factors including withdrawal regulations (if approved for layers), toxicity problems, species involved and other disease conditions. Obtain expert advice in setting up a treatment program.

It is difficult, if not impossible, to prevent coccidiosis by sanitation practices alone. Coccidiosis is prevented best by feeding a coccidiostat. A coccidiostat is a drug added to feed at low levels and fed continuously to prevent coccidiosis. A good coccidiostat should:

- Prevent clinical outbreaks of coccidiosis
- Have no undesirable side effects (e.g. depressed weight gain)
- Allow a natural immunity to coccidiosis to develop in the flock if exposure is present
- Be inexpensive

Feed broilers a ration containing a coccidiostat continuously until the last week before marketing. Feed replacement birds a ration containing a coccidiostat continuously until they are about 16 weeks old. Give turkeys a coccidiostat during the growing period while confined and for an added week or 10 days after moving them to range.

Many coccidiostats are available on the market. Selection should be based on several factors including cost, type of operation and past experience.

A coccidiosis vaccine is available commercially. The product is useful in certain types of operations, but it should not be used indiscriminately. Seek expert advice before using the product.

## BLACKHEAD

### (Histomoniasis, Infectious Enterohepatitis)

Blackhead is an acute or chronic, infectious protozoan disease of fowl, primarily affecting the ceca and liver. The disease is present wherever poultry is raised. Blackhead is one of the critical diseases of growing turkeys. It may cause stunted growth, poor feed utilization and death loss. It is of lesser economic importance in chickens since chickens are more resistant.

**Cause:** Blackhead is caused by a protozoan parasite called *Histomonas meleagridis*.

**Transmission:** The blackhead organism, *Histomonas meleagridis*, is passed in the fecal material of infected birds. In many instances, the organism is shed within the eggs of *Heterakis gallinae*, the cecal worm of chickens and turkeys. Free-living forms do not survive long in nature, but organisms contained within cecal worm eggs may survive for months or years. Because of this, most blackhead transmission is considered due to ingestion (eating) of cecal worm eggs infected with the blackhead organisms.

Chickens frequently are infected with blackhead without showing signs of the disease. These chickens may shed enormous numbers of blackhead organisms, many of which are protected by cecal worm eggs since cecal worms are so common in chickens. Because of this, outbreaks in turkeys often can be traced to direct or indirect contact with ranges, houses or equipment previously used by chickens.

Free-flying birds also may introduce an infection.

**Symptoms and lesions:** Most blackhead losses occur in birds 6 to 16 weeks old. Among the symptoms are: loss of appetite, increased thirst, droopiness and drowsiness, darkening of the facial regions ("blackhead") and diarrhea (sulfur-colored droppings). Morbidity and mortality are variable, but mortality is seldom above 10 to 15 percent; however, it may approach 80 to 90 percent in uncontrolled turkey outbreaks. In chickens, losses usually are low.

Lesions of uncomplicated blackhead are confined to the ceca and liver, thus the reason for the synonymous term, enterohepatitis. The ceca are ballooned and walls may be thickened, necrotic and ulcerated. Caseous (cheesy) cores which may be blood tinged usually are present. Peritonitis may be present if ulcers have perforated the ceca walls. Livers are swollen and display circular depressed areas of necrosis about 1/2 inch in diameter. Lesions are yellowish to yellow green and extend deeply into the underlying liver tissue. Healing lesions may resemble those seen in visceral leukosis.

Lesions observed in chickens frequently are atypical as the liver lesions may be absent or less pronounced.

*Diagnosis:* Blackhead presenting typical lesions is diagnosed readily on the basis of the lesions. Atypical forms, particularly in chickens, must be differentiated from cecal coccidiosis and Salmonella infection in particular. Laboratory tests may be required for positive diagnosis in such cases.

*Treatment and prevention:* A number of drugs on the market can be added to drinking water to bring blackhead outbreaks under control. Use these compounds in accordance with the manufacturer's recommendations. Palatability may be a problem with certain compounds, thus making it difficult to get proper drug intake.

Good management practices can do much to control the blackhead problem. Do not maintain turkeys and chickens on the same premises. Do not range turkeys on ground previously used by chickens unless several years have elapsed. Rotate ranges at periodic intervals if possible. Cecal worm control may help reduce blackhead incidence. Wire or slatted floors around feeders and waterers will reduce exposure.

Despite good management practice, there are certain premises which are so contaminated that it is necessary to feed drugs continuously at a low level to prevent blackhead. Such drugs are used in the same manner as coccidiostats are used to prevent coccidiosis.

### TRICHOMONIASIS

Trichomoniasis is an infectious protozoan disease of fowl, primarily affecting the upper digestive tract. It is universal in distribution. It is a particularly hazardous disease of turkeys, pigeons and quail, and is of lesser importance in chickens.

*Cause:* A protozoan parasite called *Trichomonas gallinae* is the cause in turkeys and chickens. Other species affect pigeons and quail.

*Transmission:* Birds most frequently acquire trichomoniasis by ingestion of contaminated feed and water. Stagnant water ponds and ditches frequently are contaminated. Free-flying birds may introduce an infection to a premise. Once introduced, the disease spreads as birds eat or drink materials contaminated by droppings or oral discharges of infected birds. Recovered birds may become carriers indefinitely.

*Symptoms and lesions:* Most turkey losses due to trichomoniasis occur in young and growing birds. Among the symptoms are loss of appetite, droopiness, loss of weight and darkened heads. The chest may be depressed as the crop usually is

empty, although occasionally the crop is distended and filled with foul smelling fluid contents. Morbidity in a flock may be high, but mortality usually is low except in severe outbreaks. The course of the disease is prolonged in turkeys unless affected birds are cannibalized.

Lesions of uncomplicated trichomoniasis usually are confined to the upper digestive tract, affecting the crop in particular. Occasionally oral lesions are observed. Lesions consist of necrotic ulcerations with accumulations of caseous material which build up over the affected areas.

Early reports described lesions of trichomoniasis involving the ceca and liver which resembled those of blackhead. Although trichomonads frequently are found in the lower intestinal tract, it is now recognized that blackhead-type lesions are not associated with the organisms.

*Diagnosis:* Trichomoniasis is diagnosed on the basis of lesions and demonstration of the causative organism viewed on slides through the microscope. Since other diseases, such as crop capillaria infection and fungus infections of the crop, may produce similar lesions, do not make a diagnosis without a microscopic examination.

*Treatment and prevention:* Move birds to sanitary surroundings, if possible. Birds may be treated with copper sulfate ("bluestone") in the drinking water at a 1:2000 dilution for 4 to 7 days. Make the copper sulfate solution as follows:

*Stock solution:* Add 1 pound of copper sulfate to a gallon of water containing 1 cup of vinegar. Mix thoroughly to get into solution. (Never give undiluted stock solutions to birds.)

*Drinking water solution:* Add 1 tablespoon (½ ounce) of stock solution to each gallon of drinking water.

Trichomoniasis is not a problem if birds are supplied with sanitary surroundings, including well-drained ranges and clean drinking water and feed.

### HEXAMITIASIS (Infectious Catarrhal Enteritis)

Hexamitiasis is an acute infectious disease of turkeys, quail, ducks, chukar partridges and pigeons. Heavy losses have been reported in one outbreak among ringnecked pheasants. Chickens apparently are not affected. It was reported first in ducks and pigeons in 1923 and in turkeys in 1938. Before establishing the true nature of the disease in turkeys, the condition was thought to be trichomoniasis.

Hexamitiasis is recognized as a disease problem in every commercial turkey-producing area. It may



be a major problem in localized areas during a particular year followed by one or more years in which incidence is very low.

**Cause:** Hexamitiasis is caused by a bilaterally symmetrical, flagellated, one-celled parasite of the genus *Hexamita*. *Hexamita meleagridis* is the cause in turkeys; in pigeons it is *Hexamita columbae*. Experimentally, the *Hexamita* of turkeys can be transmitted to young quail, chicks and ducklings, and that of quail and partridges can be transmitted to poults. However, poults cannot be infected with the organism isolated from pigeons.

**Transmission:** Hexamitiasis is primarily a disease of young birds and outbreaks seldom occur in poults past 10 or 11 weeks of age. Losses are most severe in birds 3 to 5 weeks old. Apparently, resistance develops rapidly with increasing age, regardless of previous exposure.

The primary infection source is droppings from carrier birds. About a third of recovered birds become carriers. Most outbreaks result from a build-up of organisms through several broods of poults in such manner that exposure of the following brood is overwhelming. Indirect transmission may result from fecal material carried from one location to another on shoes or equipment. Free-flying birds such as quail also may be carriers.

**Symptoms and lesions:** Symptoms primarily are listlessness and foamy or watery diarrhea with rapid weight loss due to the dehydrating effect. Birds often will huddle together near the heat source and cry or "chirp" constantly as though in pain. Convulsions due to lowered blood sugar levels shortly precede death. Affected birds suffer losses in weight and survivors remain stunted for long periods.

Dehydration and emaciation are the principle gross lesions. The intestine usually appears to have lost tone with local bulbous areas of congestions. Intestinal contents usually are thin and watery.

**Diagnosis:** Diagnosis depends upon history, symptoms and microscopic examination of intestinal contents. A definite diagnosis cannot be made unless typical flagellates can be demonstrated in intestinal contents taken from the duodenum area. Most flagellates observed in the ceca are nonpathogens.

**Treatment and prevention:** Prevention depends upon sanitation with particular emphasis upon separating age groups. If an individual must care for several age groups, the younger group should be cared for first.

Hepzide, fed continuously at levels of 0.025 and 0.035 percent, will aid in preventing losses.

Hepzide at a level of 0.02 percent in water now appears to offer the most promise as a therapeutic measure.

The disease does not respond well to treatment but 1:2000 solutions of copper sulfate with dried whey (3 to 4 ounces per gallon of the dilute solution) is an old stock remedy. This solution should serve as the only source of drinking water for 5 to 7 days, repeating after a 3-day rest if necessary.

Aureomycin at a level of 200 grams per ton of ration is of some benefit.

## LEUCOCYTOZOONOSIS

Leucocytozoonosis is an acute, sometimes highly fatal disease of young turkeys and ducklings. The causative protozoan parasites invade the circulatory system where they destroy great numbers of leucocytes (monocytes and macrophages). In certain respects, the disease resembles true malaria, but birds are the sole hosts of the genus *Leucocytozoon*.

The disease occurs in many areas of the country, but is found more frequently in the South and Southeast. Mortality may reach 100 percent in ducklings up to 8 weeks of age, and losses may be severe in turkeys up to 12 weeks of age. Clinical symptoms usually are not apparent in older birds, but they may remain carriers for months.

**Cause:** A protozoan parasite similar to the true malaria parasite is the cause. The organism responsible for the disease in turkeys is designated as *Leucocytozoon smithi*; in ducks it is called *Leucocytozoon simondi*.

**Transmission:** The disease is transmitted by several species of the black fly, *Simulium sp.*, which breeds in running streams. After feeding on infected birds, the flies can transmit the disease at the end of 4 days and remain infective for about 18 days. Direct transmission from bird to bird does not occur. Recovered birds remain carriers and serve as reservoirs of infection in subsequent years.

**Symptoms and lesions:** Younger affected birds may lack appetite and exhibit droopiness, weakness, increased thirst and rapid labored breathing. If drowsy birds are made to move they may become greatly excited. The course of infection usually is rapid with visible symptoms seldom lasting more than 2 or 3 days, terminating in death or beginning recovery. Recovered birds may appear stunted with the flock as a whole appearing to lack uniformity. In adult birds, clinical symptoms are seldom detectable.

The most consistent pronounced gross lesion is spleen enlargement and congestion. Anemia

and emaciation usually are evident in clinically ill birds. The flesh of affected birds often is flabby and yellowish. Mild congestion of the upper intestinal tract is common.

*Diagnosis:* A positive diagnosis may be rendered only after demonstration of the causative organism in stained blood smears (Giemsa or Wright's stain may be used).

*Treatment and prevention:* Prevention depends upon control of black fly populations and turkeys

being reared away from running streams. Segregate breeding and brooding operations since adults may be carriers. Brooding in screened houses will prevent infection in young birds.

Drugs effective against malaria appear to have little if any value in the treatment of leucocytozoonosis. Sulfaquinoxaline is considered valuable in reducing losses. Administer it in the drinking water at a level of 0.025 percent for 5 to 7 days, and following by adding it in feed at a level of 0.0175 to 0.025 percent until losses are controlled.

## Section 7.

### PARASITIC DISEASES

#### POULTRY LICE

The chief effects of lice on their host are due to the irritation they cause. The birds become restless and do not feed or sleep well and may injure themselves or damage their feathers by pecking or scratching the parts irritated by lice. Weight gains and egg production may drop.

All lice infecting poultry and birds are of the sucking and chewing type. Mites may be confused with lice. The mites suck blood.

In general, each species of lice is confined to a particular kind of poultry, although some may pass from one kind of poultry to another when birds are closely associated. Chickens usually are infested with one or more of seven different species; turkeys have three common species.

All species of poultry lice have certain common habits. All live continuously on feathered hosts and soon die if removed from them. The eggs are attached to the feathers. Young lice resemble adults except in color and size. They differ in preferred locations on the host, and these preferences have given rise to the common names applied to various species.

In general, the incubation period of lice eggs is 4 to 7 days, and development of the lice from hatching to the adult stage requires 17 to 21 days. Mating takes place on the fowl, and egg laying begins 2 or 3 days after lice mature. The number of eggs probably ranges from 50 to 300 per female louse.

#### The Head Louse

As the name suggests, this species (*Cuclotogaster heterographus*) is found mainly on the head, although it occurs occasionally on the neck and elsewhere. It usually is located near the skin in the down or at the base of the feathers on the top and back of the head and beneath the bill. In fact, the head of the louse often is found so close to the skin that poultrymen may think it is attached to the skin or is sucking blood. Although it does not suck blood, the louse is very irritating and ranks first among lice as a pest to young chickens and turkeys. Heavily infested chicks soon become droopy and weak and may die before they are a month old. When the chickens become fairly well feathered, head lice decrease but they may increase again when the fowls reach maturity.

This louse is oblong, grayish and about 1/10 inch long. The pearly-white eggs are attached

singly to the down or at the base of the small feathers on the head. They hatch in 4 or 5 days into minute, pale, translucent lice resembling adults in shape.

#### The Body Louse

The body louse (*Menacanthus stramineus*) of chickens prefers to stay on the skin rather than on the feathers, and it chooses parts of the body that are not densely feathered, such as the area below the vent. In heavy infestations, it may be found on the breast, under the wings and on other parts of the body, including the head.

When the feathers are parted, straw-colored body lice may be seen running rapidly on the skin in search of cover. Eggs are deposited in clusters near the base of small feathers, particularly below the vent, or in young fowls, frequently on the head or along the throat. Eggs hatch in about a week and lice reach maturity in 17 to 20 days.

This is the most common louse infesting grown chickens. When present in large numbers, the skin is irritated greatly and scabs may result, especially below the vent.

#### The Shaft Louse

The shaft louse, or small body louse (*Menopon gallinae*) is similar in appearance to the body louse, but smaller. It has a habit of resting on the body feather shafts of chickens where it may be seen running rapidly toward the body when feathers are parted suddenly. Sometimes as many as a dozen lice may be seen scurrying downward along a feather shaft.

Since the shaft louse apparently feeds on parts of the feathers, it is found in limited numbers on turkeys, guinea fowl and ducks kept in close association with chickens. It does not infest young birds until they become well feathered.

#### Other Kinds of Chicken Lice

Four other kinds of lice usually are found on chickens, but they are less abundant and more important than the ones previously discussed. The wing louse (*Lipeurus caponis*), a slender gray species resembling the head louse, is the most widely distributed and is found in the greatest numbers. It is sluggish and usually is seen resting between the barbules of the wing and tail feathers or occasionally on the neck hackles and back feathers.

The fluff louse (*Goniocotes gallinae*), which is found (as the common name implies) on the fluff



of the body feathers, is small, rather broad, yellow and inactive. As it stays mostly in the fluff, it causes little irritation or other injury.

The large chicken louse (*Goniodes gigas*) is a robust, dark, smoky-gray species of striking appearance. It is seldom found in poultry flocks.

The brown chicken louse (*Goniodes dissimilis*), occurring mainly in the southern states, is large and reddish brown. It seldom occurs in large enough numbers to cause serious damage.

### Poultry Mites

All classes of poultry are susceptible to mites, some of which are blood-suckers, while others burrow in the skin or live on or in the feathers. Still others occur in the air passages and in the lungs, liver and other internal organs.

Poultry mites cause retarded growth, reduced egg production, lowered vitality, damaged plumage and even death. Much of the injury, consisting of constant irritation and loss of blood, is not apparent without careful examination.

### Common Chicken Mite

This mite (*Dermanyssus gallinae*) is probably the most common mite found in all types of poultry. It is a blood-sucker, and when present in large numbers, loss of blood and irritation is sufficient to cause anemia. Egg production is reduced seriously.

This mite feeds at night, and usually remains hidden in cracks and crevices during the day and attacks birds at night while on the roosts. In very heavy infestations, some mites may remain on the birds during the day. About a day after feeding, the female lays eggs in cracks and crevices. The eggs hatch and the mites develop into adults in about 1 week. During cold weather, the cycle is slower. A poultry house remains infested 4 to 5 months after it is vacated.

Since the mite will feed on wild birds, they may be responsible for some infestations, but spread more likely is caused by using contaminated coops. Human carriers are important. Since these mites do not stay on the birds during the day, apply treatment only to the houses and equipment and not on the birds.

### Feather Mite

This mite (*Ornithonyssus sylviarum*) is an occasional but serious pest of chickens. Heavy infestations result in lowered condition of the birds and egg production, as well as a scabby skin condition. This mite remains on the bird and does more damage than the common chicken mite. It

resembles the common chicken mite, but can be differentiated in that it is present on birds in large numbers during the day. It prefers the feathers below the vent and around the tail, but can be found on all parts of the body.

Females lay eggs on feathers where the young mites complete their development without leaving the host. Since they remain on the fowl most of the time, treatment of birds is necessary to destroy the mites.

### Scaly-Leg Mite

This mite (*Cnemidocoptes mutans*) is one of the itch mites and lives under the scales on feet and legs. It also may attach to the comb and wattles.

This mite causes a thickening of scales on the feet and legs. It spends its entire life cycle on the birds and spreads from bird to bird, mainly by direct contact.

### Depluming Mite

This mite (*Cnemidocoptes gallinae*) causes severe irritation by burrowing into the skin near the base of feathers, and frequently causes feathers to be pulled out or broken. The mite is barely visible to the naked eye and can be found in follicles at the base of the feathers. The mites crawl around the birds at times, thus enabling them to spread from bird to bird.

### Fowl Tick or Blue Bug

This pest (*Argas persicus*) is one of the most serious parasites of poultry when it becomes numerous in poultry houses or on a poultry range. The tick is a blood-sucker, and when present in large numbers it results in weakened birds, reduced egg production, emaciation and even death.

The fowl tick is found throughout most of the South and is extremely hardy. Ticks have been kept alive without food for more than 3 years. Ticks will feed on all fowl types.

The ticks spend most of their life in cracks and hiding places, emerging at night to take a blood meal. Mating takes place in the hiding areas. A few days after feeding, the female lays a batch of eggs. She may lay several batches with a blood meal between each. In warm weather, the eggs hatch in 10 to 14 days. In cold weather, they may take up to 3 months. Larvae that hatch from the eggs crawl around until they find a host fowl. They remain attached to the birds for 3 to 10 days. Then they leave the birds and find a hiding place. After a few days, they molt and then seek another blood meal. This is followed by another molt and blood meal.

Ticks are difficult to eradicate, and methods employed must be performed very carefully. It is not necessary to treat the birds, but houses and surrounding areas must be treated thoroughly.

#### Chiggers, Red Bugs or Harvest Mites

These pests (*Eutrombicula alfreddugesi*) attack chickens and turkeys, as well as human beings. Normally these small mites feed on wild animals, birds, snakes and lizards. Only the larvae of chiggers attack poultry or animals; adult mites feed on plants.

Larvae usually attach to the wings, breasts and necks of poultry. They inject a poisonous substance that sets up local irritation and itching. After a few days, it becomes engorged and drops off. Injury to grown fowl may not be apparent or noticed until the bird is dressed; then the lesion shows up and greatly reduces carcass value. Young chickens or turkeys may become droopy, refuse to eat and die. Due to methods of raising poultry, turkeys are affected more frequently than chickens.

#### Large Roundworms

One of the most common parasitic roundworms of poultry (*Ascaridia galli*) occurs in chickens and turkeys. Adult worms are about 1½ to 3 inches long and approximately the size of an ordinary pencil lead. Thus, they can be seen easily with the naked eye.

Birds heavily infected may show droopiness, emaciation and diarrhea. Death may occur in very heavy infections, but the primary damage is reduced efficiency.

Chickens 3 to 4 months old show resistance to infection.

Specimens of this parasite are found occasionally in eggs. The worm apparently wanders from the intestine up the cloaca and is incorporated in the egg as it is formed.

The life history of this parasite is simple and direct. Females lay thick heavy-shelled eggs in the intestine and these pass in the feces. A small embryo develops in the egg. They do not hatch. The larvae in the egg reaches infective stage in 2 to 3 weeks. These embryonated eggs are very hardy and under laboratory conditions may remain alive for 2 years. Under ordinary conditions, probably not many live more than 1 year. Disinfectants and other cleaning agents do not kill eggs under farm conditions. Birds become infected by eating eggs after they have reached the infective stage.

Available drugs will remove only the adult parasite. The immature form probably produces

the most severe damage. The treatment of choice is piperazine. Many forms of piperazine are produced and all are effective if administered properly. Follow the manufacturer's instructions carefully.

The parasite can be controlled by strict sanitation. If the birds are confined, clean the house thoroughly and completely before a new group is brought in. Segregate birds by age groups, with particular care applied to sanitation of young birds. If birds are on range, use a clean range for each group of birds.

#### Capillaria

There are several species of *Capillaria* that occur in poultry. *Capillaria annulata* and *C. contorta* occur in the crop and esophagus. These may cause thickening and inflammation of the mucosa, and occasionally severe losses are sustained in turkeys and game birds. However, the parasites are not usually present in large enough numbers to produce economic loss.

In the lower intestinal tract there may be several different species but usually *C. obsignata* is the most prevalent. The life cycle of this parasite is direct. The adult worms may be embedded in the lining of the intestine. The eggs are laid and passed in the droppings. Following embryonation which takes 6 to 8 days, the eggs are infective to other poultry which may eat them. The most severe damage occurs within 2 weeks of infection. The parasites produce a catarrhal inflammation and sometimes cause hemorrhage. Erosion of the intestinal lining may be extensive and result in death. These parasites may become a severe problem in deep litter houses. Reduced growth, egg production and fertility may result from heavy infections.

If present in large numbers, these parasites are usually easy to find at necropsy. Eggs may be difficult to find in droppings, due to the small size and time of infection.

Since there is no good treatment for capillaria infection, control is best achieved by preventive measures (refer to the paragraphs on flock health management in Section 1). Some drugs, fed at low levels, may be of value in reducing the level of infection on problem farms. Game birds should be raised on wire to remove the threat of infection. As some species of capillaria have an indirect life cycle, control measures may have to be directed toward the intermediate host.

#### Cecal Worms

This parasite (*Heterakis gallinae*) is found in the ceca of chickens, turkeys and other birds. The worms are small, white and measure ¾ to ½ inches in length.

This parasite, probably the most common worm parasite of poultry in the United States, apparently does not seriously affect the health of the bird. At least no marked symptoms or pathology may be blamed on its presence. The main importance is that it has been incriminated as a vector of *Histomonas meleagridis*, the agent that causes black-head or infectious enterohepatitis. This protozoan parasite is apparently carried in the cecal worm egg and is transmitted from bird to bird through the egg.

The life history of this parasite is similar to that of the common roundworm. The eggs are produced in the ceca and pass in the feces. They reach the infective form in about 2 weeks. In cooler weather, this may take longer. The eggs are very resistant to environmental conditions and will remain for long periods.

The cecal worm can be removed by treatment with piperazine compounds. Since the worm itself produces no observable damage and the eggs live for long periods, it is advisable and necessary to keep chickens and turkeys separated to prevent spread of infectious enterohepatitis.

### Tapeworms

Tapeworms or cestodes are flattened, ribbon-shaped worms composed of numerous segments or

divisions. Tapeworms vary in size from very small to several inches in length. The head or anterior end is much smaller than the rest of the body. Since the tapeworm may be very small, careful examination often is necessary to find them. A portion of the intestine may be opened and placed in water to assist in finding the tapeworms.

The pathology or damage tapeworms produce in poultry is controversial. In young birds, heavy infections result in reduced efficiency and slower growth. Young birds are more severely affected than older birds.

All poultry tapeworms apparently spend part of their life in an intermediate host, and birds become infected by eating the intermediate hosts. These hosts include snails, slugs, beetles, ants, grasshoppers, earthworms, houseflies and others. The intermediate host becomes infected by eating the eggs of tapeworms that are passed in the feces.

Although several drugs are in use to remove tapeworms from poultry, most are of doubtful efficacy. In general, tapeworms are most readily controlled by preventing the birds from eating the infected intermediate hosts.

Trade names are used occasionally for better understanding of information presented. No endorsement of name products is intended nor is criticism implied of similar products not mentioned.



## Section 8.

### APPENDIX

#### GLOSSARY OF TERMS

*Active immunity* — immunity or resistance to disease that has been acquired by host response to a disease agent. It can be acquired by having a disease and recovering or by vaccination.

*Acute* — as applied to disease, one which has a short and relatively severe course.

*Anemia* — a condition in which the blood is deficient in quantity or quality. If deficient in quality there is a reduction in the hemoglobin content of the blood or in the number of circulating red blood cells, or both. Anemia is characterized by paleness of skin and mucous membranes and loss of energy.

*Antibody* — an immune substance found in the blood produced in response to stimulation by an antigen.

*Antigen* — a suspension of microorganisms. A substance which, when taken or injected into the body, will stimulate antibody production.

*Diagnostic Antigen* — used to detect the presence of specific antibodies in the blood of an animal; used in serological tests.

*Antiserum* — serum containing specific antibody used to treat a specific disease.

*Antitoxin* — a specific kind of antibody that will neutralize toxin.

*Bacteria* — microscopic, single-celled plant forms widely distributed in nature. Those capable of producing disease are referred to as pathogenic bacteria.

*Bacterin* — killed suspension of bacterial organisms used as an immunizing agent.

*Bipolar* — as applied to a bacterial cell, one which will stain deeply at the cell ends and takes little stain centrally.

*Bivalent* — as applied to antigens or bacterins, one which is made up of two strains of organisms.

*Carrier* — an apparently healthy animal that harbors disease organisms and is capable of transmitting them to other susceptible animals.

*Catarrhal* — describes an inflammatory process involving the mucous membranes characterized by an increased flow of mucous.

*Chronic* — as applied to disease, one of long duration.

*Cocci* — bacterial forms which, when fully developed and free, are spherical.

*Coccidiostat* — drug incorporated into the feed

at low levels and fed continuously to prevent coccidiosis.

*Congestion* — excessive accumulation of blood in a part.

*Contagious* — as “contagious” disease, refers to an infectious disease that may be transmitted readily from one individual to another.

*Culture* — used as a verb, to attempt to isolate a causative organism from a diseased bird. Used as a noun, a population of microorganisms propagated in artificial media.

*Cyanosis* — bluish discoloration of the skin, particularly the comb and wattles in birds.

*Diffuse* — as applied to hemorrhage, one which is spread over considerable area.

*Disease* — any departure from a normal state of health.

*Ecchymotic* — as applied to hemorrhage, a rather large hemorrhagic spot.

*Edema* — presence of abnormal amounts of fluid in tissues.

*Etiology* — study of the causes of disease.

*Exudate* — fluid associated with an inflammatory reaction.

*Flagellated* — an organism, bacterial or protozoan, possessing slender whip-like processes.

*Fomite* — inanimate object that may harbor disease organisms.

*Friable* — easily pulverized or crumbled.

*Fungi* — low order of vegetable organisms; some are capable of producing disease.

*Gross* — as applied to tissue changes which can be seen with the naked eye.

*Hemorrhage* — escape of blood from the vessels, bleeding.

*Immune* — resistant to a particular disease.

*Immunity* — condition of being immune.

*Infection* — invasion of the tissues by pathogenic organisms resulting in a disease state.

*Infectious* — as applied to disease, one produced by living organisms. As applied to living organisms, those which are capable of producing disease.

*Inflammation* — response of tissues to an injury or other irritant.

“*Itis*” — suffix denoting an inflammatory state, such as enteritis — inflammation of the intestines, airsacculitis — inflammation of the air sacs.

*Lesion* — visible change in size, shape, color or structure of an organ.

*Listless* — indifferent to surroundings.

*Microscopic* — invisible to the naked eye, visible only by the aid of a microscope.

*Morbidity* — incidence of disease in a flock, the percentage of diseased individuals in a population; percentage affected.

*Mortality* — death rate.

*Necrosis* — death of a circumscribed portion of tissue.

*Neoplasm* — abnormal growth such as a tumor.

*Parasite* — as used in this publication, an animal form that lives on or within a bird to the detriment of the bird.

*Parboiled* — having a boiled or cooked appearance.

*Parenteral* — as applied to drug or vaccine administration, to inject as subcutaneously, intramuscularly.

*Pathogen* — as applied to organisms, one capable of producing disease.

*Pathogenicity* — disease-producing capability of a disease organism.

*Pathognomonic* — as applied to symptoms and lesions, one which is specific or characteristic for a particular disease.

*Peracute* — excessively acute, as applied to disease that has extremely sudden onset and a short, severe course.

*Petechial* — as applied to hemorrhage, characterized by small hemorrhagic spots.

*Polyvalent* — as applied to antigens and bacterins; one made up of several strains of organisms.

*Predispose* — to confer a tendency toward disease.

*Protozoa* — unicellular animal forms, some of which are parasitic.

*Rales* — abnormal respiratory sound; rattling, wheezing.

*Rickettsial* — a group of microorganisms intermediate between the bacteria and the viruses, some of which are pathogenic to man and animals.

*Rod* — as applied to bacteria, a cylindrical shaped organism.

*Serological test* — test performed on the serum of an animal to determine if specific disease antibodies are present.

*Serotype* — as applied to microbial organisms, a strain of microorganisms as determined by serological methods.

*Sign* — any objective or discernible evidence of disease; symptoms and lesions.

*Sporadic* — as applied to disease outbreak, those occurring here and there; not widely diffused.

*Spore* — as applied to bacteria and fungi, a re-

productive element capable of resisting unfavorable environmental conditions.

*Stress* — factor tending to lower resistance of an animal to disease, such as chilling, moving, etc.

*Symptom* — detectable signs of disease.

*Toxin* — poison produced by microorganism.

*Tumor* — neoplasm; a mass of new tissue which persists and grows independently of its surrounding structures, and which has no physiologic use.

*Vaccine* — suspension of large amounts of disease organisms used to produce immunity in the animals to which it is administered.

*Virulence* — as applied to a pathogenic microscopic organism, its ability to overcome the body defenses of the host.

*Virus* — ultramicroscopic microorganisms; some capable of producing disease.

## TABLES OF WEIGHTS AND MEASURES

### Weights

1 gram (g) = 1000 milligrams (mg)  
1 kilogram (kg) = 1000 grams  
1 ounce (oz) = 28.35 grams  
1 pound (lb) = 16 ounces = 0.4536 kilograms  
= 453.6 grams

### Liquid Measure

1 Liter (L) = 1000 milliliters (ml)  
= 33.81 ounces  
1 gallon = 4 quarts = 8 pints = 231 cubic inches = 3.785 liters  
1 gallon of water = 8.34 pounds  
1 teaspoon = 4.93 milliliters  
1 tablespoon = 3 teaspoons = 14.79 milliliters  
1 fluid ounce = 2 tablespoons  
1 cup = 8 fluid ounces = 16 tablespoons  
= 236.6 milliliters  
1 pint = 2 cups = 16 fluid ounces  
= 473.2 milliliters

### Working Tables of Weights and Measures (Approximate Values)

*Parts per million (p.p.m.)*  
1 gram in 1 ton (2000 lb.) = 1 p.p.m.  
1 pound in 500 tons = 1 p.p.m.  
4 milligrams in 1 gallon of water = 1 p.p.m.

If feed is to be medicated at a level of 200 p.p.m., add the medicant at the rate of 200 grams per ton.

If water is to be medicated at a level equivalent to a feed level of 200 p.p.m., add the medicant at the rate of 400 milligrams per gallon. (Based on water intake being 2 times that of feed.)

### Dilutions

To make a 1-2000 dilution of a drug in drinking water, add 1 ounce of drug to 16 gallons of water.

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