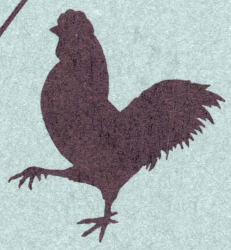
B-1031

A Manual of POULTRY DISEASES



TEXAS A&M UNIVERSITY
TEXAS AGRICULTURAL EXTENSION SERVICE · · · TEXAS AGRICULTURAL EXPERIMENT STATION
College Station, Texas

[Blank Page in Original Bulletin]

A MANUAL OF POULTRY DISEASES
(Errata Sheet)

1. Page 6 -- Paragraph 2, 2.b. (1.). Change "infection" to "injection".

2. Page 11 -- Footnote. Insert "not" in the last phrase so as to read ".....", but it is not primarily a respiratory disease".

3. Page 35 -- Paragraph 2. Insert "not" in sentence 3 so as to read ".....", and not primary causative agents".

4. Page 39 -- Paragraph 4, Sentence 3. Change "flood tinged" to "blood tinged".

5. Page 41 -- Paragraph 2, Sentence 2. Change "dimondi" to "simondi".

6. Page 45 -- Under Antigen, sentence 2. Change "antigen" to "antibody" so as to read ".....", will stimulate antibody production".

7. Page 46 -- Under Rickettsial. Change "bacterin" to "bacteria" so as to read "..... between bacteria and the viruses, ..."

Written and Edited
by a
Poultry Disease Bulletin Committee
Comprised of

R. R. BELL, Associate Professor of
Veterinary Parasitology

A. I. FLOWERS, Professor of
Veterinary Microbiology

L. C. GRUMBLES, Professor and Head,
Department of Microbiology

C. F. MEINECKE, Assistant Professor
of Veterinary Microbiology

C. M. PATTERSON, Extension Veterinarian

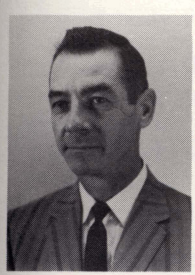
B. C. WORMELI, Extension Poultry Husbandman

C. F. HALL, Associate Professor of
Veterinary Microbiology, Chairman

Texas A&M University

A MANUAL OF POULTRY DISEASES

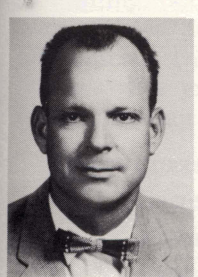
R. R. BELL. D.V.M., Univ. of Georgia, 1952; M.S., Texas A&M Univ., 1955. Research and teaching in veterinary parasitology.



A. I. FLOWERS. D.V.M., Texas A&M Univ., 1950; B.S., 1942; M.S., 1959. Research and teaching in poultry diseases; special interest, Salmonellosis, respiratory diseases and avian encephalamyelitis.

L. C. GRUMBLES. D.V.M., Texas A&M Univ., 1945; M.S., 1957. Eighteen years of experience in poultry disease research and teaching.

C. F. MEINECKE. D.V.M., Kansas State Univ., 1952; M.S., Oklahoma State Univ., 1960. In charge of diagnostic and pullorum testing laboratories.



C. M. PATTERSON. D.V.M., Texas A&M Univ., 1947. Special field of interest, preventative veterinary medicine.

B. C. WORMELI. M.S., Univ. of Wisconsin, 1933; B.S., 1929. Twenty-three years of experience in commercial poultry industry. Current major emphasis, poultry production.

C. F. HALL. D.V.M., Kansas State Univ., 1951; M.S., Michigan State Univ., 1959; B.S., Kansas State Univ., 1949. Eleven years of experience in poultry disease research and teaching.

Contents

SECTION 1. GENERAL INFORMATION

THE POULTRY INDUSTRY is the third largest farm enterprise in the United States. In Texas poultry and eggs rank fourth as a source of farm income. The annual Texas gross value to the farm is about 160 million dollars. The production of poultry and eggs is highly commercialized, 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 requisite 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 down grading.

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 feeder and waterer space, over medication and others. 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 most important poultry diseases. They are caused by bacteria, viruses, rickettsia and fungi. Some protozoan diseases, such as coccidiosis and blackhead, behave much as infectious diseases and they are considered here with infectious disease. More correctly, they should be classed with the other parasitic illnesses.

When living agents, such as bacteria, enter the body, multiply and cause a disturbance of normal function, infection has occurred. 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, but not turkeys. Other organisms affect a large number of species. For example, some of the Salmonella organisms affect a large variety of species ranging from and 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 frequent ways infectious diseases are introduced into poultry flocks are:

1. Contact with a diseased individual.
2. Contact with a healthy bird that has recovered, but still is a carrier.
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 man, who moves from flock to flock.
9. Feed or contaminated feed bags.
10. Contaminated premises through soil, old litter.
11. Airborne — organisms do not spread far through the air. This source of infection is probably not as important as was once believed.
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 wave-like action, move foreign material out of such structures as the trachea (wind pipe).

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.) Infection of antiserum
      (2.) Transferred from dam to offspring

Inherited resistance may be complete or partial; for example, turkeys are not susceptible to laryngotracheitis. And while chickens are more resistant than turkeys to blackhead, they may become infected under certain conditions. Inherited resistance or susceptibility to the fowl leucosis complex 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 it is 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 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

The 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 "grey eye" of ocular leucosis is specific.

Hatchery and Breeder 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 developing 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 a term "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. Purchase only day-old chicks or hatching eggs. Some require frequent access to replacement birds, thus making it difficult for each individual to grow all of his replacements. 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. 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.

5. Keep chickens and turkeys separate. Preferably, only chickens or only turkeys should be kept on the same premises.

6. Maintain hatchery supply flocks on separate premises from other birds.

7. Select a reliable commercial feed, or, if farm mixing is done, mix carefully according to a dependable formula.

8. Provide an adequate supply of safe water. Avoid watering from surface tanks, streams or ponds.

9. 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. For example, in some areas it is necessary to vaccinate against laryngotracheitis, while in other areas such vaccination actually leads to a disease problem.

10. Discourage persons other than the caretaker or essential personnel from visiting the poultry house or yard.

11. If a disease problem develops, obtain an early, reliable diagnosis and apply the best treatment, control and eradication measures for that specific disease.

12. Dispose of all dead birds by burning, deep burying or preferable by a disposal pit. This phase of management often is overlooked.

13. 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 in collecting the eggs and take precautions to prevent contamination from organisms that may be on hands or clothing of the person making the collections.

2. 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.

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

4. 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.

5. 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 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. Clean and disinfect all crates and vehicles used for transporting started or adult birds after each use.

Cleaning and disinfecting.

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

   a. Settle dust by spraying lightly with the disinfectant to be used.

   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, 1⅓ ounces (1 heaping tablespoonful) to a gallon of hot water.

2. In the hatchers, cleaning and disinfecting includes 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 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%) 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%). 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 96th 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 24th to the 96th 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 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. Other drugs, such as the antibiotics and NF 180* are included in many 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 such a nonspecific 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.

*Trade name of a product which contains 50 grams of furazolidone per pound.

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 medication period.

The following table 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.

Preventive Medication

It is doubtful that there is 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 strictly the prescribed recommendations for treatment. 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 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 the virus in embryonated chicken eggs. Fluids and tissues from the infected embryos contain large amounts of virus and are collected to make the commercially available vaccines. 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 virus 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 will 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 and infectious laryngotracheitis vaccines 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. Tailor vaccination programs to meet the needs of a particular operation in a particular area. For example, it is *not* recommended that Texas poultrymen use laryngotracheitis vaccine since the disease is not a current problem. Individual premise or area experience also governs the efficient use of such products as erysipelas or fowl cholera bacterin.

Diseases for Which Vaccines Are Available

Most products now on the market are for the control of virus infections, particularly the virus respiratory infection. These diseases are:

1. Newcastle disease
2. Infectious bronchitis
3. Infectious laryngotracheitis
4. Fowl pox*

In addition, experimental vaccines are undergoing extensive testing for the control of avian encephalomyelitis (epidemic tremors).

Bacterins are available commercially for the control of:

1. Erysipelas
2. Fowl cholera

A "so-called" vaccine to control coccidiosis has received much publicity in recent years. The use of the product presently is not encouraged in Texas.

*Fowl pox may cause symptoms and lesions of the respiratory system, but it is primarily a respiratory disease.

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 out-dated 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.

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

1. Mass methods:
   a. Drinking water – Newcastle, infectious bronchitis.
   b. Dust – Newcastle, infectious bronchitis.
   c. Spray – Newcastle, infectious bronchitis.
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 and fowl pox.

Vaccines frequently are combined to reduce labor and 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 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 discus-

sion of this is included in the individual disease sections.

A Suggested Vaccination Program for Chickens

Following is an outline of one vaccination program that will produce good results in Texas. As previously suggested, individual farm situations will call for varied procedures to meet individual needs.

1. *Chicks for Market Eggs or Hatching Egg Flock Replacement.*
   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 begin production.
2. *Chicks for Broiler Production*
   a. Vaccinate against Newcastle at 4 days and 4 weeks of age using intranasal vaccine by the drinking water method.
   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 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 School of Veterinary Medicine of Texas A&M University, operates Poultry Disease Investigation Laboratories at College Station, Center, Gonzales and Stephenville. 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 Medicine Building, Texas A&M University, Telephone 846-5781.

2. Center. Adjacent to the Shelby County Fairgrounds. Telephone LY 8-4451.

3. Gonzales. Texas Agricultural Experiment Station, Substation 21, 9 miles southwest of Gonzales, off State Highway 97. Telephone 437-3621.

4. Stephenville. On the corner of the College Poultry Farm across the street from the Tarleton State Campus. Telephone WO 5-5749.

Laboratories are open from 8 a.m. to noon and 1 p.m. to 5 p.m. Monday through Friday, college 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 begins.

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. Submit

three or four birds, or more if young chicks or poults. When birds are dying rapidly with a few preliminary symptoms, bring in several dead birds with the sample.

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 will be 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 generally considered as belonging to the plant kingdom, although they may possess certain characteristics such as motility, common to members of the animal kingdom. Usually they are grouped according to 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. Nutritive requirements are satisfied by absorption through a thin permeable cell membrane, but moisture is necessary before such processes can occur. The growth requirements of the species dictate the environment in which it may be encountered; soil, water or animal tissues.

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, serological 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.

Antibiotics in disease control create an environment unfavorable to multiplication of the species being attacked. Most antibiotics are selective in action, effective only against certain species or possibly types of organisms, and then only while present in the blood and tissues at sufficient levels. In the latter respect, antibiotics are very different from vaccines. Control of disease by vaccination is completely different than control through antibiotics and consequently, using antibiotics for prevention of disease is unsound basically.

SALMONELLA AND PARACOLON INFECTIONS

More than 800 different species or serotypes of organisms belong 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 world-wide 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 chick 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 where mortality may approach 90 percent if untreated. Survivors usually are stunted or unthrifty.

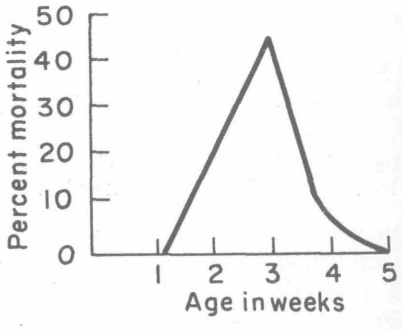


Figure 1. Typical mortality curve in young birds.

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 pin point 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 hatching purposes.

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 wherein most hatcheries participate in the plan. In other areas, the disease appears to be gaining ground. 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 rapid plate whole blood 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 there is a high percentage (over 10 percent) of reactors, it generally is advisable to dispose of the flock rather than try to dispose of reactors and establish a negative test.

2. If an isolation and identification of the organism has been made from a flock, consider the whole flock infected and do not keep it as a breeder flock. It may be held as a commercial egg flock if circumstances warrant and no breeder flocks are maintained on the premises, but it will serve as a potential source of infection to the other breeder flocks in the area.

3. After disposing of infected birds, thoroughly clean and disinfect the house and equipment. A 3 percent water solution of cresol (Liquor Cresolis Compositus) is excellent for such purposes.

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 organisms. Antigenic structure is the only difference between variant and standard strains of the organism. Differences are shown by the following illustration of antigenic composition:

Standard	1X,	X11	(1)	2	(3)
Variant	1X,	X11	1	(2)	3

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 best drug in treating infected flocks is furazolidone, marketed under the trade name of NF-180 by Hess and Clark (Furoxone by Eaton Laboratories).

The recommended level is 2 pounds NF-180 (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 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, but 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 instituted 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 pin-point 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, practice range rotation and other special precautions to prevent a carry over of infection to the next flock.)

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 is not recommended.

Treatment: Furazolidone (NF-180, Hess and Clark) is the choice drug and levels of 100 grams per ton of feed for 10 to 14 days usually will stop mortality. In view of recent experimental results indicating that some strains of 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.

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

The commercial liquid preparation contains 3.44 percent sulfaquinoxaline. For the first 2 days, mix the drug in the drinking water at the rate of 3 tablespoonsful (or 1½ ounces) per gallon, giving a concentration of 1:2500. Then reduce to 2 tablespoonsful (1 ounce) per gallon, making 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. Literally, the term means resembling typhoid fever of the typhoid bacterium. Related to poultry, paratyphoid is a term denoting the disease produced by any of the many Salmonella species except *S. pullorum* and *S. gallinarum*. Infection may result in acute or chronic disease. Acute clinical disease occurs more often in young birds and rarely in adults. Over 600 species or serotypes of Salmonella organisms are recognized with most birds, reptiles and mammals serving as host to one or more species. Economically the disease is of greatest concern to the turkey industry.

History: Moore, in 1895, recorded the first authentic case of paratyphoid when he isolated and identfied a Salmonella from pigeons with an enteritis. Moore referred to this organism as a member of the hog-cholera group. The first report of paratyphoid infections 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. Paratyphoid infection in disease outbreaks in man, many species of fowls and various species of domestic animals has been well substantiated.

Etiology: The organisms of this group are serologically related and are Gramnegative, 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 staggering losses.

Severity of infection with each of the various serotypes has not 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 in the Midwest and North Central area, but until recently it rarely has been a problem in the Southwest.

Results of research to date indicate that *all* Salmonella species must be considered as potential pathogens in poultry with outbreak severity depending 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 these 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 others. 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 New York, Minnesota, Iowa, Texas and others have reported recovering one or more species from 20 to 30 percent of the samples from certain materials such as animal byproducts 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 pin-point 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 an 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 successful 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 infection, 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. Although no control program is included in the National Poultry or National Turkey Improvement Plans, Texas now has a voluntary testing program which may be used by producers shipping eggs to such hatcheries. 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 known to aid in paratyphoid control include:

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 to 48 hours after the eggs are placed in the incubator).

Rodent control.

Isolation from other sources of infection such as pigeons, ducks and others.

Serological testing of hatchery supply flocks and following outlined requirements of voluntary state programs.

Treatment: Proper use of drugs may reduce mortality in acute outbreaks of paratyphoid. The choice drug is furazolidone (NF-180), 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 2 pounds NF-180 (100 grams furazolidone) 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 is 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 organism 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. Edwards *et al.* have pointed out the close relationship of this group of the Salmonellas and have suggested that they should be called "parasalmonellas" rather than "paracolons." 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 conditions, these organisms may cause disease in young turkey poults, and thus be of economic importance. For unknown reasons, Texas poultry producers have had fewer disease outbreaks from paracolon infections than are reported for some other sections of the country.

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

OMPHALITIS

Omphalitis is technically an inflammation of the navel. As commonly used, it 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 taken place during the past 3 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 over-heating 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 as being transmitted 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" and in general appear to be of inferior quality and show 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 72 hours but may not occur before the tenth day.

Characteristic lesions are poorly healed navels, 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 are the only sure way 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.

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 egg transmitted.

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 as on shoes or equipment.

Symptoms and lesions: The disease seldom is seen in birds under 4 months of age. 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 resembling those of fowl typhoid or possible bluecomb. Typical lesions include any or all of the following:

Pin-point 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.

Spleen enlarged and congested.

Creamy or solid collection of material in joints.

Cheesy material in the internal ear of birds having twisted necks.

Turkeys may have pneumonia with solidification of one or both lungs.

Diagnosis: A tentative diagnosis may be made based on flock history, symptoms and post mortem 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: Until recently, vaccines for fowl cholera have been useless, particularly the old typhoid-cholera bacterin. Recent research programs have developed a fowl cholera bacterin that appears to produce good results. Field reports indicate that it may be valuable in halting the spread of chronic fowl cholera within affected flocks.

Management practices that aid in preventing fowl cholera are:

Complete depopulation each year between older birds and replacements.

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 drug of choice, using the same levels 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. Sulfamerazine and sulfamethazine also may be used, but they are less effective than sulfaquinoxaline. In all instances, follow the manufacturer's directions carefully.

ERYSIPELAS
(Swine Erysipelas)

Erysipelas is a bacterial disease caused by *Erysipelothrix insidiosa* (formerly *E. rhusiopathiae)* which was once considered to be an important disease only in swine and sheep. The disease in swine frequently was 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 bacteria of the genus Streptococcus, whereas infection of man with *Erysipelothrix insidiosa* is called erysipeloid.

Cause: Erysipelas of fowl is caused by a bacteria, *Erysipelothrix insidiosa (E. rhusiopathiae).* In turkeys, it occurs most often in the fall or winter months and usually affects birds 4 to 7 months old, although any age birds are susceptible. Incidence is higher in males than in females, possibly because in fighting, males receive numerous skin abrasions which may serve as entry portals.

Transmission: Apparently, the primary route of infection is via wounds or skin abrasions which become contaminated with soil. The organism survives for long periods in the soil and most outbreaks are thought to originate from contaminated soil or premises. Sheep and swine may be carriers, but many outbreaks occur on premises that have not been occupied by sheep or swine for many years.

Symptoms and lesions: The first indication of trouble may be the discovery of several dead birds. Usually several sick birds can be found. Symptoms include:

Birds may appear listless with drooping wings and tail.

Many birds may have a yellowish-green diarrhea but the vent feathers usually are not soiled.

Infrequently some birds may be found lame with swollen leg joints.

The snood or carnucle of the toms may present a turgid, swollen, purple appearance.

In breeding flocks there is usually decrease in fertility and hatchability.

Difficult breathing may be evident in advanced cases. Mortality rates vary from 2 to 25 percent.

Typical gross lesions are:

Purple-colored blotches on the skin.

Inflammation of the intestinal tract.

Small or diffuse hemorrhages in almost any tissues or organs.

Enlarged, congested livers and/or spleens.

Thickened or vegetative type growths on heart valves.

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 and treatment: Antiserum prepared in horses is available and effective under certain conditions, but it is expensive. Use of this product is very limited.

Bacterins now available are useful on premises where history indicates that outbreaks may be expected. Three weeks are required to produce a high level of immunity. If vaccinated at 10 or 12 weeks of age, repeat the procedure for birds held over as breeders. The vaccine is relatively expensive. Use according to manufacturer's directions.

Good management practices for preventing erysipelas include:

Avoiding use of ranges previously occupied by swine or sheep.

Debeaking and other measures which prevent injuries from fighting.

Removal of the snoods of the toms.

Removal of sick birds to a hospital pen for treatment and to prevent other birds from killing them, plus moving unaffected birds to clean range may aid in stopping the spread of the disease.

In addition to using bacterin for unaffected birds, 150,000 to 200,000 units of penicillin injected into the leg or breast muscle of visibly sick birds is very effective in decreasing mortality. One injection usually is sufficient, but treatment may be repeated in 3 to 4 days if necessary. Penicillin has no value as a prophylactic measure because of its short period of activity.

BOTULISM
(Limberneck; Food Poisoning)

Botulism is a disease caused by the ingestion of a toxin produced by the bacterial organism, *Clostridium botulinum*. All domestic animals and fowls are susceptible. To some degree, swine are resistant and vultures apparently are immune. 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 toxin of the bacteria *Clostridium botulinum,* an organism common in nature. This organism grows best under conditions of high humidity, relatively high temperature and in an environment containing decaying vegetable or animal matter. Acid conditions are detrimental. Stagnant pools or damp areas containing decaying matter with an alkaline reaction are a danger area.

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 water soluble and relatively heat stable, but may be destroyed by boiling. Fly maggots feeding on decayed tissue may contain enough toxin to cause the disease when ingested by poultry.

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 and lesions: Ingestion of small amounts of toxin may produce a mild form with recovery in 24 to 48 hours. First signs of illness generally are weakness with flaccid paralysis of the legs, neck and wings, followed by prostration and death. Affected birds may have a peculiar trembling of the feathers which are loose in the follicles and generally shed freely during examination of birds. Because of the paralysis, birds are unable to swallow and mucus accumulates in the mouth.

Usually there are no significant lesions evident on dead birds with the possible exception of loose feathers and an excessive amount of mucus and dirt in the mouth. Examining the contents of the crop and intestine may reveal maggots or other material to indicate that the birds 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 post mortem lesions.

As an aid to diagnosis, give sick birds water, keep in a cool environment and treat with antitoxin intravenously. Recovery of a large percentage of birds treated this way would substantiate the diagnosis.

Additional supportive evidence for diagnosis may be obtained by producing the identical illness signs in other birds or laboratory animals by inoculating them with material from the digestive tract of affected birds.

Prevention and treatment: Aim at eliminating the toxin source. For this reason:

Do not feed spoiled canned foods.

Promptly remove all dead animals from houses or pens.

Control fly populations.

Debeak birds to prevent cannabalism.

Do not allow wet feed to accumulate around feeders.

To treat this disease:

Remove all visibly sick birds from the flock, place them in a cool shady area and fill the crop with water twice daily.

Use mild laxatives such as molasses at a rate of 1 pint per 5 gallons of water for birds which have been exposed but do not yet show symptoms.

Antitoxins may be useful in treating affected birds, but are expensive. In turkeys use 2 to 4 cc of polyvalent antitoxin, administered intravenously, intraperitoneally or intramuscularly.

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." From the beginning, this group of diseases has been important.

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 disease 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.

Ornithosis.

Quail bronchitis.

Caused by bacteria:

Chronic respiratory disease—air sac syndrome of chickens and infectious sinusitis of turkeys.

Infectious coryza.

Endemic fowl cholera (roup).

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 had been reported in at least 30 states. This indicates that within a very few years the disease became widespread.

Cause: Newcastle disease is caused by a virus. Different types of 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 adults.

Transmission: Newcastle disease is highly contagious. All birds in the group usually become infected within 3 to 4 days. The virus can be transmitted through contaminated equipment, shoes and clothing of man 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 mucus in the trachea, (3) cloudy air sacs, (4) cast or plugs in the air passages of the lungs and (5) cloudiness of 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 zero 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 shell, off-color 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, this disease cannot be differentiated from infectious bronchitis and some of the other respiratory infections except by laboratory 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. This disease does not

always respect even the best management program, but practices outlined in other sections of this bulletin will help reduce 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 one most successful and widely used is the mild live virus vaccine known as the B_1 type. This vaccine was used originally by dropping it into the nostril or eye. Now, however, the vaccine is applied also in the drinking water, as a dust or as a spray.

In broiler production use two doses of the B_1 type vaccine. The first vaccination usually takes place when birds are between 1 and 5 days of age and is followed by a second dose at about 3 to 4 weeks.

Give chickens to be kept for egg production three applications of the vaccine. The vaccine usually is given when the birds are 1 to 5 days, 3 to 4 weeks and 4 to 5 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_1 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.

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: This infection is confined to the respiratory system. Symptoms are difficult breathing, gasping, sneezing and rales (rattling). 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, soft shell, small, 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 may be applied through drinking water, as a dust, spray or 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 pheasant. It is characterized by respiratory distress, rapid spread and often, 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 material 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. Use the vaccine with care because it is a virulent virus and vaccinated birds can become carriers of the infection. When vaccine is indicated, it is applied to the mucous membrane of the cloaca. 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 confirmed completely.

ORNITHOSIS

Ornithosis, also known as psittacosis, is an acute or chronic virus infection of a number of fowl and other animals, including man. When the disease is in birds of the parrot family, it is known as psittacosis. Ornithosis is the name used when the disease occurs in other species of wild or domestic birds. In domestic poultry production, the disease has been a problem only in turkeys. Its importance is magnified because it is transmissable to man, and in the past, workers in poultry processing plants have become infected from handling turkeys.

Cause: The disease is caused by an agent usually considered to be a large particle virus. Numerous virus types have been recognized. Some produce only a mild disease in turkeys while others may cause significant mortality.

Transmission: How turkey flocks become infected is not known. 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 having 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. Post mortem lesions are suggestive but a definite diagnosis can be made only by isolating the virus 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.

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 the 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.

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 PPLO or Mycoplasma is common to all three conditions. CRD, in a strict interpretation, is caused by a pure PPLO 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 PPLO 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 PPLO infection.

PPLO are widely spread and affect many species of birds. They exist in most, if not all, chicken flocks. They also are widely spread among turkeys, but only about 40 percent of turkey flocks are infected.

Transmission: The primary method by which PPLO are 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 ultimate answer to this problem in both chickens and turkeys must be eradication. To accomplish this, it is necessary to develop PPLO free breeder flocks. This goal can now be reached in turkeys because satisfactory blood tests are available to detect infected flocks. Testing and hatching from noninfected flocks provides a good chance for eradicating infectious sinusitis in turkeys. A voluntary eradication program is available to Texas turkey producers operating under the Texas Turkey Improvement Plan.

Because all chicken flocks are infected, obtaining PPLO free flocks is more difficult. An eradication program has not been well formulated for use in chickens. Such a program should become available as additional information develops.

INFECTIOUS CORYZA

Infectious coryza is a bacterial infection of chickens. It is a slow-spreading, chronic disease that does not affect all birds at one time. It occurs more often in semi-mature or adult birds.

Cause: The cause is a bacterial organism known as *Hemophilus gallinarium.* The organism remains endemic in some areas but is only rarely found in Texas.

Transmission: Transmission is by contact with affected or carrier birds. Individuals which have recovered from the disease may appear normal, yet carry the organism for long periods. Once the infection has been in a flock, each individual must be considered a carrier. Preventing contact be-

tween susceptible and affected or carrier birds usually will prevent spreading.

Symptoms and lesions: Main disease signs are "puffy" or "doughy" swellings around the face and in the wattles. There is a discharge from the nostril, and the sinus under the eye may be swollen. Because of a watery discharge from the eyes, the lids may be stuck together. Vision may be affected because of the swelling around the eye.

Diagnosis: Because the disease is rare in this state, can be confused with other respiratory diseases and is difficult to diagnose, submit suspected cases to a trained diagnostician who has laboratory facilities. Diagnosis can be made only by isolating and identifying the organism. This frequently is confused with endemic or localized fowl cholera which is a much more common disease in Texas.

Treatment and prevention: Infectious coryza usually can be prevented by management programs that prevent contact between susceptible and infected birds. Introduce started pullets or mature birds with extreme care to prevent introduction of the infection onto the premises.

There are effective drugs for treating this condition. The first requirement prior to treatment is a correct diagnosis. The disease can be treated effectively with sulfa drugs such as sulfathiazole, sulfamethazine and sulfaquinoxaline.

Prevention is the only sound approach to control. 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 often are confused even in textbooks on poultry diseases, and pictures of this disease often are mislabeled as infectious coryza. The disease, as it occurs in Texas, 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 fowl cholera may show symptoms and lesions closely resembling this condition. The disease affects chickens primarily, although turkeys can be infected by inoculation.

Cause: The bacterial organism causing this disease is a Pasteurella that cannot be differentiated by ordinary methods from *Pasteurella multocida,* the bacteria causing acute fowl cholera.

Transmission: This disease is transmitted only by direct contact between susceptible and infected birds. Recovered birds remain carriers indefinitely. They will spread it, however, only by direct contact. Separation between susceptible and carrier birds, even by a short distance, will prevent spread.

Symptoms and lesions: This is a chronic disease affecting only a small percentage of birds at one time. It causes a nasal discharge, an inflammation of the eye and a swelling of the sinus under the eye. The sinus becomes filled with hard, "cheesy" type exudate that has a putrid odor. Sometimes the lungs and air sacs are affected. The disease causes little direct mortality, but it does cause reduced egg production, and increased cull birds and in general poor performance.

Diagnosis: A trained diagnostician usually can identify 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 sometimes is difficult and requires a long time. Because of this, diagnosis usually is based on other characteristics of the disease.

Treatment and prevention: As with infectious coryza, this disease can be prevented readily by management. It requires only separating infected or carrier birds from the susceptible population. Fifteen years ago, it was estimated that 80 percent of the chicken flocks in Texas were infected. By management programs, infection has been almost eliminated from commercial operations. Introduce started or adult birds with extreme caution to prevent introducing the infection.

In general, treatment is unsatisfactory. Probably the most effective drug is sulfaquinoxaline. This must be given in the ration at a rate of .033 percent. It has to be given a long time and simply serves to prevent other birds from developing the disease. It does not cure those already affected. This drug level is near the toxic range and must be used with care to prevent causing a drop in egg production. Injection with some antibiotics appears to be useful in treating the affected bird.

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 isn't contagious and does not spread from one bird to another. Most 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 a paralysis or other forms of nervous symptoms. The more chronic form in older birds usually results in the losses 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 changing litter and replacing with new 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, avian lymphomatosis (leucosis), avian encephalomyelitis (epidemic tremor), avian infectious synovitis and bluecomb disease.

AVIAN POX

(Fowl pox, Canker, Avian diphtheria, contagious epithelioma, Sore head)

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. The disease may occur in any age bird at any time during the year; but the greatest seasonal incidence is 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, most 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 infects chickens, turkeys, pheasants, quail and ducks; pigeon pox virus infects pigeons, chickens and turkeys; and canary pox virus infects 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, which is the usual manner this 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 experience difficulty in eating and breathing.

The disease manifests itself in one of, or a combination of, 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 elsewhere 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 usually 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. Two kinds of vaccines are available: pigeon pox and fowl pox vaccines. The pigeon pox vaccine is of questionable value, and its use normally is not recommended. Fowl pox vaccine is an efficient product. Its use varies according to type of operation, but generally the following recommendations apply:

Broilers: Vaccination usually is not required; but in some sections where 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. Market birds not selected and vaccinated within a day or two.

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

AVIAN LYMPHOMATOSIS
(Avian Leucosis Complex)

Avian lymphomatosis is a widespread, transmissible virus disease complex of chickens, turkeys and other fowl characterized by tumor formations which may involve any body structure. The disease is the most devastating condition affecting mature laying chickens and is becoming increasingly important as a cause of losses in broilers, growing birds and turkeys.

Cause and transmission: Avian lymphomatosis is caused by a virus or group of viruses. Some researchers believe that variable host response to a single agent explains its different manifestations; others feel that disease variability is explained best by assuming that several agents or strains of virus are involved.

The disease may be transmitted in a number of ways. The agent(s) of lymphomatosis is eliminated naturally from the body of infected birds via eggs or feces. The virus may be transmitted mechanically from infected birds to susceptibles by blood-sucking parasites (mites, ticks and others) or by man in such procedures as fowl pox vaccination or blood testing.

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

Manifestations of avian lymphomatosis: Five distinct types are recognized: visceral, neural, ocular, osteopetrosis and blood type.

Visceral lymphomatosis (big liver disease) is the most common form and the one which produces the most economic loss. Although visceral lymphomatosis may cause losses in birds as young as 3 to 4 weeks, it is seen most commonly in adults, particularly in the first production year. Severe losses frequently are associated with the onset of production; however, in such flocks, occasional losses will continue as long as the flock is retained.

Visceral lymphomatosis is characterized by the formation of lymphoid tumors, particularly of the visceral organs. Affected birds may die without preliminary symptoms; but the disease usually is chronic in nature with affected birds showing loss of appetite, progressive emaciation and diarrhea. Clinically affected birds invariably die.

Autopsies of affected birds reveal characteristic tumors. Although the liver, spleen, kidneys and ovary most commonly are involved, any organ including muscle and skin may be affected. The neoplastic process may be diffuse involving 100 percent of the affected organ, or it may be a nodular type. Affected structures, the liver in particular, may be greatly enlarged.

Neural lymphomatosis (range paralysis, fowl paralysis) attacks primarily young birds between 2 and 5 months of age; however, it has been seen as early as 3 weeks and as late as 3 years. Losses may run as high as 25 percent but usually are much lower. A flock which has had a neural lymphomatosis problem frequently will become a visceral lymphomatosis problem flock.

Neural lymphomatosis is characterized by a progressive paralysis of the wings, legs and neck. Loss of body weight, anemia, labored respiration and diarrhea are other common symptoms.

When affected birds are autopsied, lesions, if observed in uncomplicated cases are confined to the nerve trunks and nerve plexuses innervating the paralyzed extremities. Affected nerve tissue is swollen due to accumulation of lymphocytes and tissue fluids. Frequently no gross lesions are observed.

Ocular lymphomatosis (grey eye, pearl eye) is responsible for most blindness in chickens. This type usually is seen in early maturity. Morbidity and subsequent mortality usually are low but in some instances approach 15 to 25 percent.

The disorder is characterized by a spotty depigmentation or diffuse greying of the iris of the eye due to 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.

Osteopetrosis is the bone form of the disease. Until recently it was thought to be a disease primarly of older birds, particularly males; however, it is now known as an extremely common disease in young chickens and is one of the more important causes of broiler condemnation.

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 lymphomatosis are diagnosed infrequently.

Treatment and prevention: There is no treatment for lymphomatosis. Although the disease cannot be prevented completely, certain steps can be taken to control infection in a flock. Among these are:

Buy resistant strains of birds. Most reputable breeders have invested a great deal of time and money developing strains which have increased resistance to lymphomatosis.

Brood in isolation. Most lymphomatosis infections are 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.

Incubator sanitation.

Control of blood-sucking parasites.

INFECTIOUS SYNOVITIS
(Infectious Arthritis)

Infectious synovitis is a chronic disease of chickens and turkeys 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 down grading 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 agent producing infectious synovitis has been characterized as a large-particle virus, but recent research indicates that the primary cause may be a PPLO, known as *Mycoplasma synoviae.*

Transmission: Mode of transmission is not well understood. Contact transmission does occur, but the natural route of invasion is not known. Experimentally, the disease can be reproduced by many routes. Egg transmission studies are inconclusive, but some field and experimental evidence of egg transmission does exist.

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 are seen in chickens; however, shank swelling is seen infrequently in turkeys. Rapid loss of condition, dehydration and diarrhea usually are seen.

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 and enlarged spleens.

Diagnosis: A presumptive diagnosis may be based on flock history, symptoms and lesions. Laboratory tests may be necessary to differentiate the disease from staphylococcic 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 antibiotic of choice is aureomycin. 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 serve as a continued infection source.

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

The adoption of a sound sanitation program offers the best hope to prevent introduction of infectious synovitis.

EPIDEMIC TREMOR
(Avian Encephalomyelitis)

Avian encephalomyelitis is a viral infection primarily affecting susceptible chickens of all ages, but usually producing clinical manifestations only in young birds. Signs of infection include incoordination, nervousness, a jerky or irregular gait, falling over on the side with outstretched wing and muscular tremors 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 from several other countries.

The custom of hatcheries to adjust chick losses has led other segments of the poultry industry to regard this disease as of minor importance and significant only to hatcherymen. Such losses are

costly and troublesome, and as they reduce efficiency of all operations including breeder and broiler flocks, the disease 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 it is doubtful that this is of major significance. Additional work is needed before effective controls can be formulated.

Symptoms and lesions: In a small percentage of outbreaks, the disease may be suspected by 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 averages 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 is provided nearby. New cases developing after the fifth or sixth week are rare. Mortality usually is negligible but always remove visibly affected individuals and destroy them.

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, together 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 available aid must be used to give 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.

Prevention depends upon hatching from flocks which have recovered from the disease. Vaccines are used widely but are still in the experimental stage. Eventual control and eradication depend upon continued research. Methods for determining whether flocks are susceptible or immune need to be perfected and simplified. All transmission methods must be determined. Better immunizing methods are needed also and better diagnostic methods would be a great aid.

BLUECOMB DISEASE

(Pullet Disease, Avian Monocystosis of Chickens; Non-specific Enteritis, Transmissible Enteritis and Mud Fever of Turkeys)

Bluecomb is an acute, subacute to chronic, contagious disease of chickens and turkeys characterized by sudden onset, marked depression and severe diarrhea. Death losses may be high, particularly in young turkey poults; but heaviest losses in adults are due to loss of condition or production.

Cause: Bluecomb is presumably caused by a large-particle virus. The agent has not been well characterized. Some evidence exists that the turkey agent and chicken agent are not identical.

Transmission: Method of natural transmission in chickens is not known. In turkeys, the disease spreads by contact with affected birds or infected premises.

The agent from chickens will reproduce the disease in chickens and turkeys, but the turkey agent will not reproduce the disease in chickens.

The disease in chickens occurs most frequently in hot weather, particularly in heavy breeds, recently housed and in a good rate of production.

No specific environmental factors influence occurrence in turkeys.

Symptoms and lesions: Bluecomb disease produces different symptoms and lesions in chickens than in turkeys.

Chickens: The disease appears most often in young adults and is characterized by sudden onset with 25 percent or more of the flock becoming sick within a day's time. Affected birds usually exhibit marked depression, decreased feed consumption, decreased egg production, profuse whitish or watery diarrhea and cyanosis of the head (bluecomb). Early mortality may be high, but flock mortality seldom exceeds 5 percent. The disease usually runs

its course in about 2 weeks, followed by rapid clinical recovery in most instances.

When affected birds are autopsied, the following lesions may appear. The body musculature is dehydrated and may display a degenerative fish-flesh like appearance. Minute hemorrhages may be seen on the viscera and necrotic foci on the liver. The ovary usually undergoes rapid degenerative changes, and many misshapen and ruptured ova may be seen. Kidneys commonly are 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.

Turkeys: 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 drop markedly and poults lose weight rapidly. Morbidity and mortality may approach 100 percent in uncontrolled outbreaks.

Young poults show few lesions other than those asociated 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.

Lesions observed in older turkeys are essentially the same as those for chickens.

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

Treatment and prevention: Flush chickens and older turkeys 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 200-400 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. Then 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. Other than these, apply routine management and sanitation practices for disease prevention.

SECTION 5. DISEASES OF UNDETERMINED CAUSE

The cause 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.

INFECTIOUS HEPATITIS

Infectious 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 rate. 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 infectious hepatitis has not been definitely characterized. Some workers feel the agent is a bacterial organism belonging to the vibrio group, while others feel it may be a large-particle virus.

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 suggestive of 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, infectious hepatitis may be confused with diseases such as pullorum, typhoid, bluecomb, hemorrhagic disease, blackhead and lymphomatosis. 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 (4 pounds of NF-180) per ton. Supply medicated feed for approximately 10 days.

Until more is known about the causative agent of infectious hepatitis, specific prevention recommendations are not possible. Furazolidone as a preventive medicant usually prevents outbreaks, but the use of the drug for this purpose is not recommended. Routine management and sanitation practices for disease prevention offer the most economical and reliable method of control.

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 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 petechial (pin point in size), but they may be diffuse involving large areas. Hemorrhages are often 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.

HEMORRHAGIC ENTERITIS

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

Cause: Unknown. Numerous organisms have been isloated in laboratories from affected birds. Among these have been *E. coli,* paratyphoid species and yeasts; in a few instances, coccidia have been found in the intestinal contents. However, it is believed generally that these organisms are coincidental or secondary invaders, and primary causative agents. Experimental transmission studies have not proved that this is a contagious disease. Toxins, such as might be produced by poisonous plants, have been suspected, but not proved.

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 on range or drylot in 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, 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—the 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 gizzard to 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 is 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; also give them adequate shade from the sun if possible. Remove dead birds from the range promptly.

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 the birds 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 rather 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 is 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 leucosis should be ruled out.

Treatment: Affected birds usually make a spontaneous recovery if placed on the floor or if the cage bottom is covered with newspaper or other such material.

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, 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.

Post mortem 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 pin point 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 abdonimal 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_{12} and 5,000 to 10,000 units of vitamin E per ton of feed. Hold protein level at 17 percent. Feed the modified diet for an indefinite period.

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 important poultry diseases caused by these organisms.

COCCIDIOSIS

Coccidiosis is a highly contagious, 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 species of coccidia in an area and exposure to them.

The species of coccidiosis affecting chickens and turkeys are:

Chickens	Turkeys
*Eimeria tenella**	*Eimeria adenoeides**
*Eimeria necatrix**	*Eimeria meleagrimitis**
*Eimeria acervulina**	*Eimeria meleagridis*
*Eimeria brunetti**	*Eimeria dispersa*
*Eimeria maxima**	*Eimeria gallopavonis*
Eimeria mitis	*Eimeria innocua*
Eimeria hagani	*Eimeria subrotunda*
Eimeria praecox	

*Considered the major causes of clinical outbreaks.

Transmission: Coccidiosis is transmitted by direct or indirect contact with droppings of infected birds. When a bird eats coccidia, the organisms invade the intestinal tract lining where they produce tissue damage while undergoing reproduction. Within a week after eating coccidia, an infected bird will shed descendant coccidia in its droppings. So great is the reproductive potential that a single organism may produce about 1 million descendants. This means infection 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 resistant to environment and may survive for long periods. They are transmitted easily from one house or premise to another by dirty boots, free-flying birds, feed sacks, equipment and others.

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. adenoeides* 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 build up 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* 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 contain 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 disease 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: An outbreak of coccidiosis usually can be controlled by medication with certain sulfonamides. Sodium sulfaquinoxaline (S.Q.) or sodium sulfamethazine (Sulmet) may be used. Use drugs according to directions on the label. In general, they are used as follows:

Chickens:

Sulfaquinoxaline (S.Q.). The commercial liquid preparation contains 3.44 percent sulfaquinoxaline. For the first 2 or 3 days, mix the drug in drinking water at the rate of 3 tablespoons (or 1½ ounces) per gallon, giving a concentration of 1:2500 (0.04 percent). Supply non-medicated water for 3 days and follow with sulfaquinoxaline for 3 days at the rate of 2 tablespoons (or 1 ounce) per gallon, giving a concentration of 1:4000 (0.025 percent). If necessary to continue treatment, supply nonmedicated water for 3 days, after which the 1:4000 (0.025 percent) concentration can be given an additional 2 days.

Sulfamethazine (Sulmet). The commercial liquid preparation contains 12.5 percent sulfamethazine. Mix the drug in drinking water at the rate of 2 tablespoons (or 1 ounce) per gallon, giving a concentration of 1:1000 (0.1 percent). Supply medicated water for 3 days followed by 3 days on nonmedicated water. Repeat treatment for 3 days and follow with nonmedicated water another 3 days. Continue treatment for an additional 1 or 2 days if necessary.

Turkeys:

Sulfaquinoxaline (S.Q.). Mix the drug in drinking water at the rate of 2 tablespoons (1 ounce) per gallon, giving a concentration of 1:4000 (0.025 percent). The medication schedule is:

Medicated water for 3 days; nonmedicated water for 3 days; medicated water for 3 days; nonmedicated water for 3 days; medicated water for an additional 1 or 2 days if necessary.

Sulfamethazine (Sulmet). Administer the drug as outlined for chickens.

When sulfonamides are used in drinking water to treat coccidiosis, do not use feed containing sulfonamides since toxicity problems may result.

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 (depressed weight gain, production and others).

Allow a natural immunity to coccidiosis to develop in the flock if exposure is present.

Be cheap.

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

Many coccidiostats are available on the market. Most are suitable for broilers. However, sulfaquinoxaline, fed at a level of 0.015 percent, is considered the coccidiostat of choice for replacement chickens and at a level of 0.0175 percent, for turkeys.

A "so-called" vaccine to control coccidiosis is available commercially. Use of the product is not encouraged in Texas now.

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 are raised. Blackhead is one of the most important diseases of growing turkeys, causing 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 organism.

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 flood 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 ½ 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 lymphomatosis.

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, microscopic and cultural, may be required.

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 premise. 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 important 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 indefinite carriers.

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 usually 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.

Some research workers have described lesions of trichomoniasis involving the ceca and liver which resemble those of blackhead. Although trichomonads frequently are found in the lower intestinal tract, workers have not found that blackhead-type lesions are associated with the organisms.

Diagnosis: Trichomoniasis is diagnosed on the basis of lesions and demonstration of the causative

organism on slide preparations viewed through the microscope. Other diseases, such as crop capillaria infection or fungus infections of the crop, may produce similar lesions; do not make a diagnosis without 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 vinegar. Mix thoroughly to get into solution. (Never give undiluted stock solution 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, 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 trichonomiasis.

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 was transmitted to young quail, chicks and ducklings, and that of quail and partridges was transmissable to poults. However, poults could not 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 to 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 a 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. Survivors suffer great losses in weight and 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 definitive diagnosis cannot be made unless typical flagellates can be demonstrated in intestinal contents taken from the duodenum area. Most flagellates observed in the ceca area 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, care for the younger group 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 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 dried whey 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 victims' circulatory system where they destroy great numbers of leucocytes (monocytes and macrophages). In certain respects the disease resembles true ma-

laria, but birds are the sole hosts of the genus *Leucocytozoon.*

The disease occurs in many areas of the country, but is 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 dimondi.*

Transmission: The disease is transmitted by several species of the black fly, *Simulium sp.* which breed 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 seldom are detectable.

The most consistant 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 not rearing turkeys near running streams. Segregate breeding and brooding operations since adults may be carriers. Brooding in screened houses will prevent infection of young birds.

Drugs effective against malaria appear to have little if any value in 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 follow by adding it in feed at a level of 0.0175 to 0.025 percent until losses are controlled.

Refer to Texas Agricultural Extension Service publication MP-691, *Texas Guide for Controlling External Parasites of Livestock and Poultry,* for recommended insecticides and methods of use.

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 (Lipeurus 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 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 of young chickens and turkeys, which often become infested within a few hours after hatching by lice from the mother. Heavily infested chicks soon become droopy and weak and may die before they are a month old. When the chickens become faily 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 even 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 important 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 much less important than its relative, the body louse. 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 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 hologaster), 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 (Goniocotes gigas) is a robust, dark, smoky-gray species of striking appearance. It is seldom abundant or of much importance.

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 mite attacks, 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 retared 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 is a night feeder, 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 to 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 to the houses and equipment, not on the birds.

Feather Mite

This mite (*Leponyssus sylviaruns*) is an occasional, but serious pest of chickens. Heavy infestations result in lowered condition of the birds and reduced 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 to birds is necessary to destroy the mites.

Scaly-Leg Mite

This mite (*Cnemidosoptes 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 in 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 (*argus persicas*) 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 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 types of fowls.

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. The female 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, 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. Nor-

mally 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 engaged 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 about the size of lead in an ordinary pencil. 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. Two to 3 weeks are required for the larvae in the egg to reach infective stage. 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. Now the treatment of choice is piperazine. Many forms of piperazine are produced, and all are effective if administered properly. Follow the manufacturer's instructions exactly.

The parasite can be controlled by strict sanitation. If the birds are confined, thoroughly and completely clean the house 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 new group of birds.

CECAL WORMS

This parasite *(Heterakis gallinae)* is found in the ceca of chickens, turkeys and other birds. The worms are small, white and measure ⅜ 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 blackhead 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 any of the 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 or 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.

No effective drugs to remove tapeworms from poultry are known. The tapeworms can be controlled by preventing the birds from eating the infected intermediate hosts.

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 antigen 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.

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, air sacculitis – 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 bacterin 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 occuring here and there; not widely diffused.

Spore – as applied to bacteria and fungi, a reproductive 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) = 38.35 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.
2 milligrams in 1 gallon of water = 1 p.p.m.

Thus, 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.

Thus, if water is to be medicated at a level of 200 p.p.m., add the medicant at the rate of 400 milligrams per gallon.

Dilutions

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

[Blank Page in Original Bulletin]

Texas Agricultural Experiment Station, R. E. Patterson, Director, College Station, Texas