INTRODUCTION

The poultry industry is a dynamic industry which has a major economic impact in those states along the Eastern seaboard and in the Southeast. The veterinary profession has played an integral part in maintaining the viability of this industry by virtue of their expertise in the diagnosis, prevention and control of many of the disease problems facing this industry.
This course is included in the veterinary and poultry science curriculum so that the student can become better acquainted with the housing, management and disease problems commonly encountered in the poultry industry. This manual is offered as an outline of those diseases discussed in courses taught by the department of avian medicine and should be used in conjunction with the visual aid used therein. The information contained in this manual was obtained from textbooks and journals dealing with avian diseases and from personal observations of the authors and is presented so that the student can spend more class time concentrating on the gross and microscopic alterations present during the lecture period.
FACTS ABOUT POULTRY IN GEORGIA

Georgia was first in broiler production in 1998 - $2,386,382,000.
Georgia was first in total poultry income in 1998 - $2,780,732,000.
U.S. poultry income in 1998 - $22,231,320,000.

Income from some poultry products are as follows:

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- Above figures do not include the income of allied industries such as feed companies, drug companies, equipment companies, etc.

- The total state livestock income for 1986 was $1,822,233,000.
  Of this $1,262,601,000. was from the poultry industry or approximately 67.1%.
  Total poultry receipts were 37.1% of the gross farm income which was $3,407,787,000.

- The total loss to diseases in poultry in 1995 was approximately $200,000,000. in the United States, with 4% of the broilers and 17% of the layers lost to disease.
## Per Capita Consumption of Meats

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1. BROILER BREEDERS

Hybrid chickens produced by crossing white Plymouth rock and cornish breeds. These sires and dams have been bred with desired characteristics to produce the final progeny which are broilers or the meat bird that reaches the consumer. The mature breeder hens weigh from 6-9 lbs. And the male may weigh up to 10-13 lbs. These birds come into production around 25-28 wks. of age and peak in egg production at 30-34 wks. of age usually around 80-85%. Hatching eggs are produced by these hens for about 10 months. These birds are expected to produce around 165-180 eggs per bird on a feed ratio of 7-8.5 lbs. of feed per 1 doz. eggs. Feed intake is restricted to around 32-36 lbs./100 hens per day. The usual breeding ratio is 10 hens per rooster. Spent hens are sold as "heavy hens", usually frozen in cry-o-vac bags. Roosters are used in further processed products such as soup. Some genetics companies offer a mini-breeder bird that weighs 5-6 lbs. And produces a broiler comparable to the full size hens at the lower broiler weights (3.65-3.85 lbs.).
2. BROILERS

These birds are the progeny of the breeder described above. The broiler is usually marketed at around 6-7 wks. of age and weigh from 4 to 4.95 lbs. Average feed conversion ratios are 1.8 to 2.5 lbs. of feed to produce 1 pound of live broiler. The meat from these birds is used in all types of poultry products. Fresh icepack poultry and chicken parts are major ways broilers are marketed. Further processing is growing rapidly.

3. COMMERCIAL LAYERS

Usually leghorn hybrids that are used for the purpose of producing infertile eggs for human consumption. The mature hens weigh about 3.0 to 4.0 lbs. These birds come in production at 19-20 wks. of age and peak in egg production at 26-30 wks. of age usually around 90%. The hens are kept for about 14 mos. of lay and are expected to produce from 250 to 310 eggs. No roosters are required. Feed intake is non-restricted and is 22-24 lbs./100 hens per day. The feed conversion ratio is about 3.5-3.9 lbs. of feed per 1 dozen eggs. Spent hens are sold as "stewing hens" or put in soup. Many flocks are molted and brought back into production for 6 to 8 months.
4. **LIGHT BREEDER HENS (COMMERCIAL LAYER BREEDERS)**

   These birds produce the progeny mentioned above. Specific breed lines have been developed with high egg production as the main objective. Breeding ratio is 10 hens per rooster.

5. **POULTS**

   Sexually immature male or female turkeys. Kept in brooder houses 6-8 weeks before being moved to final growout facility. Poults are worth about $2.25 at hatch.

6. **TURKEYS**

   Toms (males) are marketed at around 17-21 wks. of age and weigh from 28 to 30 lbs. at that time. Hens are marketed at 14 wks. of age and weigh from 14 to 16 lbs. Feed ratios are usually 2.8 to 3.2 pounds of feed per 1 lb. of live turkey. Toms and hens are raised separately. They are housed in broiler type houses until they are 8 wks. of age. They are then grown out on ranges or in confinement. Normal mortality for turkeys over 8 wks. of age is 1 per 1000 birds daily.
7. TURKEY BREEDERS

Hens are lighted (day length extended) at 28-30 wks. of age and start producing at around 32 weeks of age. Expect 80-90 poults produced per hen. All commercial turkey breeders are artificially bred and left in production for about 24 weeks before being molted and kept in production another 15 weeks. To ensure good fertility, new hens are artificially inseminated the first time 2-3 days in a row. Afterwards, they are bred once per week. The semen is stored in crypts in the oviduct. Individual straws of semen are used for each hen to prevent venereal transmission of diseases. Broodiness is a problem in turkey hens.
Sue Clanton:
Section I
index goes on slides
9 & 10
Sue Clanton:

Section I

index goes on slides

9 & 10
AVIAN INFLUENZA

- A respiratory infection of chickens and turkeys that is characterized by upper respiratory involvement, mortality and decreased egg production in adults.

- Infects most species of birds.

- 1983 outbreak of virulent AI (Fowl Plague) in chickens in Pennsylvania and surrounding states resulted in $60 million eradication program.
ETIOLOGY

VIRAL - Orthomyxovirus type A
15 Hemagglutinin and
9 Neuraminidase types

- Extremely variable in virulence
- Highly virulent AI viruses cause the disease fowl plague
- $H_5N_2$ - Pennsylvania "break".
  $H_5N_1$ - Hong Kong outbreak – 1997-98
  These viruses are extremely virulent.

- Hens may be found dead on the nest.
INCUBATION PERIOD

- Variable - few hours to days
- Depends on virulence of the virus and the route of exposure
COURSE OF DISEASE

- 1-2 weeks - depends on strain of virus.
METHOD OF SPREAD

1. Contact with infected birds.

2. Water fowl are usually involved in outbreaks.

3. Ethnic slaughter house and distribution also involved.

Live markets are a problem.
MORBIDITY

Variable
MORTALITY

- Usually doesn't exceed 10% unless fowl plague virus.
- Then can reach 80-100% mortality.
CLINICAL SIGNS

NON-SPECIFIC:

1. Decreased feed consumption.
2. Decreased egg production.
3. Mild to severe respiratory rales.
4. Sinusitis.
5. Edema of head and wattles.
6. Diarrhea.
7. Whitens the shell of broiler breeder eggs. Shell color isn't being laid down in the oviduct. Destroy infected eggs.
POSTMORTEM LESIONS

FAIRLY NON-SPECIFIC:
1. Variable depending on strain of virus.
2. Sinusitis with mucopurulent to caseous exudate.
3. Fibrinopurulent pericarditis.
4. Congestive, hemorrhagic and necrotic changes on the skin and the intestinal tract.
5. Hemorrhages in proventriculus and heart.
Differential Diagnosis

1. Mycoplasma
2. Newcastle
3. Ornithosis
DIAGNOSIS

1. Serology
   AGP and ELISA

2. VI - HA

3. Histopathology - non-specific
TREATMENT

None
PREVENTION AND CONTROL

1. Isolation rearing.

2. Depopulate infected flocks.
   All strains are reportable.
   Bury the birds.

3. Killed vaccine are available
   for certain approved areas.
Many strains of similar virus cause signs ranging from mild respiratory signs (pneumotropi) with low mortality to severe neurological (neurotropi) and/or visceral lesions (viscerotropi) with heavy mortality.

- It affects most domestic fowl as well as many wild and pet type birds.
- It causes conjunctivitis in humans.
Paramyxo-virus type 1 = Newcastle disease. There are 9 types of paramyxovirus. Single stranded RNA virus.

The fact that this virus naturally hemagglutinates red blood cells is used in a simple differential diagnostic test. All paramyxo-viruses will hemagglutinate RBC's.

Virus can survive in dust and survives well in organic materials.
ENZOOTIC - virus has strains that differ in pathogenicity. The Mean Death Time of the minimum lethal dose (MDT/MLD) is used to differentiate among the pathotypes.

This is done in embryos and based on how quickly the embryos die.
ETIOLOGY (CONT.)

- **LENTOGENIC** - mild - kills embryos in > 90 hours.
- **MESOGENIC** - moderate - kills embryos in 60-90 hours.
- **VELOGENIC** - highly virulent neuro- or viscerotropie - kills embryos in < 60 hours.
ETIOLOGY (CONT.)

- Lentogenic & mesogenic used as vaccine strains.

  Hitchner: $B_1 - B_1$ - milder
  La Sota: $B_1 - La Sota$ - more virulent.
EXOTIC NEWCASTLE

- Synonyms: VVND, Asiatic Newcastle's, or Doyle's disease
- VVND - Velogenic Viscerotropic Newcastle Disease
- Not in the U.S. or Canada at present.
- Caused very costly outbreak in California in early 1970's which cost $54,000,000 to eradicate.
METHOD OF SPREAD

1. Aerosol from infected bird excretions

2. Mechanical vectors

3. Vaccination is done with mild viruses but this keeps a form of the disease endemic in commercial poultry producing areas.
Exotic Newcastle was brought into this country in pet birds.

This introduction caused the California outbreak.

There have been many other introductions of this virus into this country but fortunately the pet birds have not come in contact with commercial poultry flocks.
There are about 100 quarantine stations where pet birds are held when imported into the U.S.

There has been no recorded incidence where pet birds that were released from these stations caused the dissemination of NCD.

The greatest threat is smuggled pet birds.
MORTALITY

ENZOOTIC NCD

LENTOGENIC & MESOGENIC -
  - Usually negligible but can be complicated with other infections and cause up to 30% mortality.

VELOGENIC -
  - Variable up to 50% in adults and 90% in chicks.
EXOTIC NCD

- VVND -- 90-100% mortality in poultry -- much less in non-gallinaceous birds.
Most problems in commercial poultry in U.S. are caused by vaccinal strains acting as stressors causing secondary infection such as *E. coli* airsacculitis.

Mortality will vary depending on severity and virulence of secondary invaders.
SIGNs

YOUNG CHICKENS
ENZOOTIC NCD

1. Sudden onset with depression & prostration. Respiratory signs such as gasping, coughing, rales and nasal discharge, necrotic comb.

2. CNS signs closely follows respiratory signs. Twisted heads and "Stargazers". Usually less than 25% have CNS signs - opisthotonus. This occurs with either neuro- or pneumotropic strains.

3. Mortality depends on virulence of strain. May be up to 95%. 
SIGNS (CONT.)

ADULT CHICKENS
ENZOOTIC NCD

1. Sudden onset with mild depression and anorexia. Respiratory disease may be mild and mortality is low or absent.

2. CNS signs are few.

3. Layers may cease to produce. Eggs that are laid are low quality and rough or soft shelled - similar to IBV.

Production may or may not return - depending on stage of lay.
EXOTIC NCD
YOUNG AND ADULT CHICKENS

1. Marked dyspnea
2. Violent diarrhea (blood stained)
3. Swollen head and throats
4. Conjunctivitis
5. Paralysis and death in 2 to 3 days
SIGNS (CONT.)

TURKEYS

- Usually mild to moderate respiratory signs.
ENZOOTIC NCD

1. Inflammation of the trachea and airsacs
2. May be no gross lesions
EXOTIC NCD

1. Severe inflammation of trachea and airsacs.
2. Hemorrhagic or necrotic foci in mucosa of gut and cecal tonsils.
4. Pet bird may have mild non-specific lesions and no gross lesions. (Can have severe enteric type).
5. CNS histopathological lesions are present but must be differentiated from AE and MD.
DIFFERENTIAL DIAGNOSIS

1. Infectious bronchitis - respiratory
2. Laryngotracheitis - respiratory
3. Avian encephalomyelitis - neurological
4. Vit. E & selenium def. - neurological
5. Mycotic encephalitis - neurological
6. Avian influenza - variable pathogenicity
DIAGNOSIS

- History
- HA - HI
- VN - with known NCD antisera
- FA test
- ELISA
- Paired HI test on acute and convalescent sera
Isolation and identification of the virus. Hemagglutinating virus. Tracheas are the organ of choice for virus isolation.

Blocking viral growth with NCD antiserum. In cases of mixed viral respiratory infection, NDV will show up in the embryos before IBV.

Reproduce disease in susceptible chickens with virus.
1. None

2. Broad spectrum antibiotics for secondary bacterial involvement.
ENZOOTIC NCD

VACCINATION:

LIVE VIRUS

- $B_1$ - lentogenic
  VG strains (Glisson/ Villegas), vaccine developed at UGA, has reduced vaccinal reaction, viscerotropic.
- La Sota - lentogenic

KILLED VIRUS in oil adjuvant, produced with $B_1$ virus.

Maternal antibodies interferes with active immunity buffering the expected vaccine reaction.
ENZOOTIC - immature birds - broilers, leghorn pullets, etc.
Vaccinated with live vaccines usually at 1 to 4 days and at around 14 to 28 days.

This virus doesn’t replicate in the harderian gland, so lower maternal antibody is desired to prevent vaccine blockage.

BREEDERS AND LEGHORNS - give killed vaccine at around 12-18 wks. of age after being primed with live vaccine.
EXOTIC NCD

- Eradicated in the country by massive slaughter in early 1970's.
- Vaccination not allowed.
- Vaccinated for in foreign countries using more potent vaccines than are used in the U.S. Greater vaccine virulence causes more severe vaccine reactions.
- Requires multiple vaccination programs and success is limited.
INFECTIOUS BRONCHITIS

- An acute, highly contagious respiratory disease of chickens.
- All ages infected; particularly a problem in laying flocks.
- **Chicks** - growth suppression & predisposition to other diseases.
- **Hens** - variable production loss and affects egg quality.
- Turkeys resistant.
ETIOLOGY

- Coronavirus - RNA - heat sensitive.
- Many serotypes and strains with great antigenic variation have been identified.
- Mass.-41 & Conn.-46 are used as vaccine and protect against closely related serotypes.
- Different strains affect different organ systems: respiratory, renal, reproductive.
- Some important field strains are JMK, Ark. 99, Fla. 88, Holland, 072, GA variant, and many others.
INCUBATION PERIOD

- 18-36 hours - used in diagnoses.
- Rapid and highly contagious.
COURSE OF DISEASE

- 1-2 weeks, secondary problems can linger. Predisposes birds to chronic respiratory problems.
METHOD OF SPREAD

1. Airborne aerosol from infected birds.

2. Direct contact with short time carriers (about 1 mo.)

3. Contaminated premises (about 1 mo.)
MORTALITY

- Respiratory IB usually not significant-although tracheal plugs at the bifurcation cause asphyxiation.
- Some serotypes can cause serious airsacculitis.
- Depends on secondary infection such as Mycoplasma.
- Nephrotropic strains may cause high mortality in chicks and layers. Causes urolithiasis.
- Nephrotropic strains include Holt and Gray.
CHICKS -

1. May vary, usually respiratory rales
2. Wet frothy eyes with conjunctivitis - swollen harderian gland.
3. Occasional bird swollen infraorbital sinus
4. Depressed and cold
5. Increased feed conversion
6. Swollen head syndrome - the virus gets into the harderian gland located in the eyelid near the medial canthus. Secondary *E. coli* is involved.

TRT also causes a swollen head syndrome.
LAYER - 1. Respiratory rales - snicks.
2. Seldom have nasal or ocular discharge.
3. EP may drop 20-50%.
4. Soft, misshapen and or rough surfaced shells. Shell problems may persist due to prior oviduct infection.
POSTMORTEM LESIONS

CHICKS AND BROILERS

1. Hyperemia of trachea
2. Serous exudate in trachea
3. Slight airsacculitis - severity varies with serotype of IBV.
   Ark causes airsacculitis.
4. Tracheal plugs at the bifurcation
PULLETS AND LAYERS

1. Hyperemia of trachea
2. Serous & catarrhal exudate of trachea
3. Egg yolk peritonitis
4. Salpingitis & permanent damaged oviduct.  
   2-3 week old pullets infected with IBV will cause infertility, salpingitis, and internal laying.
5. Swollen kidneys with urates
DIFFERENTIAL DIAGNOSIS

1. Newcastle Disease
2. Laryngotracheitis - slow moving
3. Infectious Coryza - swollen head
4. Avian Influenza
DIAGNOSIS

1. History of fast spreading respiratory disease

2. ELISA - uses Mass. antigen but get cross reaction with other serotypes.

3. HI - less cross reaction early in an outbreak but difficult to interpret later.

4. SN - rises in titer between paired serum samples (2 wks. apart)
5. Isolation and identification of virus-embryonating eggs – stunting, curled, and hemorrhagic - vaccine strains are embryo adapted and often affect embryos on the 1st or 2nd passage whereas field strains may require additional passages before lesions appear.

6. Identification of IBV serotype - PCR, monoclonal antibody test, etc.
TREATMENT

1. None against virus

2. Broad spectrum antibiotics for secondary bacterial involvement.
PREVENTION

- Vaccination - complete prevention of IB is difficult because of variation of field strains and the ability of the virus to change. There is little cross protection between serotypes.
PREVENTION (CONT.)

VACCINES:

**LIVE** - Monovalent - usually Mass.
Bivalent - Mass. & Conn.

Other attenuated strains such as Holland, Ark. 99 and Fla 88 are used as vaccines.

It is normal after administration of a live vaccine to have a reaction 5 days later. This reaction should be resolved within 5 days.
PREVENTION (CONT.)

VACCINES:

KILLED - used in breeders and layer pullets to prevent production losses and produce consistently high antibody titers.
Parenteral antibody influences success of vaccination in young birds.

High maternal antibody may block the viremia from the 1-day-old vaccination but the Harderian gland is exposed and produces local protection.

It is often applied in a spray cabinet. Chick will rub eye on vaccine moistened feathers.

Therefore most breeders should have consistent antibody titers. These titer levels can be used as a guide for proper vaccination time in the progeny.
PREVENTION (CONT.)

- This involves a series of live and/or killed vaccines or a live vaccine every other month throughout the lay cycle.

- Progeny can be vaccinated at 2 wks. of age no matter what the parenteral antibody titers are at hatching. Even chicks that had high maternal antibody at 1-day of age will be susceptible. Those who responded to the 1-day vaccination may have a buffered vaccine response.
INFECTIOUS LARYNGOTRACHEITIS (ILT OR LT)

- An acute, highly contagious disease of fowl, characterized by respiratory distress, slow spread and high mortality.
- Airsacculitis in uncomplicated outbreaks is uncommon.
- Doesn’t occur in birds less than 2-weeks-old and most commonly occurs in chicks over five weeks of age.
- Can be a serious problem in roasters.
- Occurs naturally in pheasants.
- Reported from most countries.
ETIOLOGY

HERPESVIRUS - an enveloped virus

The virus is heat labile but can remain viable in cool weather.
POSTMORTEM LESIONS

1. Feather follicles enlarged
2. Tumors on any or all visceral organs
   Some tumors may regress with time.
3. Enlarged edematous nerves
4. Many viremic birds die without showing any visible lesions
**DIFFERENTIAL DIAGNOSIS**

1. Lymphoid leukosis - older birds only
   - MDV more common in younger birds
2. Riboflavin deficiency -
   - causes hindlimb paralysis
3. Various types of chronic hepatitis
   - i.e. blackhead
4. Any disease causing CNS signs –
   - NDV, AI, Encephalomalacia, etc.
DIAGNOSIS

1. Postmortem lesions

2. Histopathology - brain, nerve, spleen, liver, kidney, heart, bursa, skin
   - lymphocytic infiltration
1. **VACCINATION** - doesn't prevent infection - only prevents lesions - given at day 1 by subcutaneous injection in the hatchery or in-ovo at transfer

   (A) HVT Turkey herpes virus - Serotype III

   (B) SB-1* Non pathogenic MD virus - chicken origin - Serotype II

   (C) Modified Live Marek's virus CVI-988/C* - Rispen's chicken origin – Serotype I

   *Use Combined with HVT
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LYMPHOID LEUKOSIS
(LL)

- LL is a bursal dependent neoplastic disease found only in chickens over 14-16 weeks of age.
ETIOLOGY

- A small RNA virus.
- Has envelope proteins classified A through J.
- E is endogenous and already incorporated into the bird's genome. The rest are exogenous.
INCUBATION PERIOD

14-16 weeks for most types

J-virus will cause production problems in broilers.
METHOD OF SPREAD

1. Primarily transovarian tolerant hens shed the virus in nearly all eggs laid.
   - Chicks infected *in ovo* become immunotolerant. These chicks are most likely to shed virus and develop tumors.
   - Other chicks become infected in the hatch trays: Lateral transmission through fecal/oral route.

2. Lateral transmission birds exposed after sexual maturity usually develop antibodies and recover.

Deaths are only seen in chickens over 14-16 weeks old and seldom exceeds more than 10%.

Mortality curve shows low grade, chronic losses.
1. Pale shriveled combs
2. Emaciation
3. Profound depression and death
4. Reduced growth in broilers with J-virus
POSTMORTEM LESIONS

1. Hepatomegaly, frequently filling entire abdominal cavity. "Big liver disease."

2. Enlarged bursa; neoplastic nodules easily palpated. A bursa should not be palpable on an adult chicken.

3. Tumors on the mesentery and other abdominal viscera.

4. Tumors in the bone with J-virus.

5. Neural involvement never seen.
DIFFERENTIAL DIAGNOSIS

Marek's disease

REV
1. HISTOPATHOLOGY
   A homogenous population of lymphoblastic cells seen
2. POSTMORTEM LESIONS
   (A) Bursal enlargement
   (B) Lack of nerve involvement
3. COFAL - For presence of group specific antigen
4. RIF (resistance inducing factor) - For envelope proteins - differentiates subgroup
5. ELISA
PREVENTION

1. Elimination of the virus from breeder hens.
   Test and slaughter.
   Virus is spread to progeny.

2. Genetics
Considerable effort has been spent attempting to get COFAL negative flocks for vaccine production and MD research.
Exogenous Virus
Horizontal
Infectious Virus
Transient Viremia
Immunity to exogenous Virus
Lymphoid leukosis rare

Endogenous Virus
Congenital Genetic
Viral DNA Integrated in gamete DNA
Viremia or antigen expressed
Immune tolerance to endogenous Virus
Lymphoid leukosis very rare

Horizontal and vertical transmission of exogenous LLV and genetic transmission of endogenous virus. (Crittenden, Avian Patho )
AVIAN ENCEPHALOMYELITIS (AE)
(EPIDEMIC TREMORS)

- A viral infection of young chicks and laying hens, characterized by ataxia, tremors of head and neck and paralysis of young chicks and a sudden drop in egg production for 4-5 days in laying hens.

- Disease in turkeys milder than in chickens.

- Can also occur in quail.
ETIOLOGY

PICORNAVIRUS

- Transmitted through eggs laid by infected hens for up to 1 month.
- Lateral transmission also occurs in chicks.
INCUBATION PERIOD

9 to 21 days

Embryos may be infected

COURSE OF DISEASE

- Signs may be at hatch time or delayed for 2 to 3 weeks.
- Most commonly appear at 7-10 days.
METHOD OF SPREAD

(1) Primarily by egg transmission
(2) Contact with infected birds
(3) Vaccination - can't vaccinate birds in lay with this live vaccine because it will be passed in the eggs and affect the chicks.
SIGN

(A) CHICKS

(1) Occurs in first 3 weeks of life
(2) Ataxia, birds fall from side to side
(3) Tremors of head and neck, especially when excited
(4) Lateral recumbency and paralysis
(5) Opacity of the lens
Develops in 8-10 weeks
(B) HENS

(1) Transient (4-5 days) drop in egg production

(2) Egg production drops of 10-20% on the average but may be as great as 40%.

(3) Virus is shed in eggs during this time.
MORTALITY

(A) CHICKS
5-10% in most cases:
may exceed 50%

(B) LAYING HENS
Negligible
POSTMORTEM LESIONS

NVL - lesions are microscopic
DIAGNOSIS

(1) History

(2) Histopathology

- Brain, proventriculus, gizzard, and pancreas - lesions may not be visible in acute cases. Hold the birds until they are 2 weeks of age to allow lesion development. The most diagnostic lesions occur in the brain with central chromatolysis and perivascular cuffing.
DIAGNOSIS (CONT.)

(3) Isolation - AE antibody free eggs. The virus may need to become adapted to eggs, so pass it 3-5 times. Lesions consist of stunted, curled embryos, and atrophy of leg muscles.

(4) Serology – ELISA, SN – test hens prior to egg production.

(5) Embryo susceptibility study
DIFFERENTIAL DIAGNOSIS

(1) Vit E - Selenium deficiency - CNS signs.
    Usually occurs in birds over 3 weeks of age.

(2) Marek's - CNS signs.
    Usually seen in birds over 3 weeks of age.

(3) Newcastle - CNS signs.

(4) Rickets - inability to move around.

(5) Vit. B₁ And B₂ - Thiamine deficiency - stargazing

(6) Mycotic Encephalitis - CNS signs.
    Occurs after 3 weeks of age generally.
TREATMENTS

None
PREVENTION

1. Vaccination of breeder hens or commercial layers. Passive immunity prevents disease in baby chicks.
   (A) Killed vaccine
   (B) Modified live vaccine - before onset of production
2. Don't hatch eggs from viremic hens.
3. Isolation of affected flocks.
4. Vaccinate birds after 7 wks. of age-often given with pox at 10 weeks of age.
1. Vaccinate laying hens with only killed vaccines.
2. Don't expose chicks under 3 weeks of age.
3. 80-90% of all hens are immune to AE at housing with no symptoms visible.
CHICKEN INFECTIOUS ANEMIA

- First identified in Japan in 1979. It was called the chicken anemia agent (CAA) and later referred to as chicken anemia virus (CAV).

- The disease is characterized by aplastic anemia, generalized lymphoid atrophy with concomitant immunosuppression, and subcutaneous hemorrhage.

- Primarily causes T-cells suppression
CHICKEN INFECTIOUS ANEMIA

- It is often complicated by secondary viral, bacterial, or fungal infections.
- The disease produced in young chickens most frequently involves severe bone marrow depletion with a reduction in hematocrit values.
- It plays a major role in a number of multifactorial diseases associated with hemorrhagic syndrome and aplastic anemia.
SYNONYMS

- Hemorrhagic syndrome, anemia-dermatitis, blue-wing disease.
ETIOLOGY

- Circovirus - a single stranded, circular DNA virus.
- There is only one serotype but there are variations in pathogenicity.
METHOD OF SPREAD

- Primarily vertical transmission from breeder flocks which become infected during lay.
- The virus is not transmitted from hens which have cleared the infection during the rearing period.
- Lateral spread usually results in subclinical disease.
PERIOD OF INCUBATION

- Ten to 14 days in antibody negative hens.

- In natural outbreaks, peak mortality in broilers is commonly observed between 17 to 24 days followed by a second wave of increased mortality between 30 to 34 days of age.
Clinical CAV rarely occurs, but occasional outbreaks are generally devastating.

Mortality is usually 5 to 10%, although it has been reported up to 60% in some cases.
Anemia is the only specific sign with hematocrit values ranging from 6-27%.

Normal hematocrit value is 35%.

Depression, paleness, and anorexia are often seen.

Lesions on the wing (blue-wing) result from secondary bacterial infections leading to gangrenous dermatitis.
POSTMORTEM LESIONS

- Hemorrhages can be observed in the skin and musculature. The bone marrow is pink to yellow in color.
- Thymic atrophy is obvious. Changes in the bursa of Fabricius are less obvious.
HISTOPATHOLOGY

- Bone marrow depletion; erythrocytes, thrombocytes, and granulocytes are replaced by adipose tissue.

- Other lymphoid tissues including spleen and bursa of Fabricius are also atrophic but to a lesser degree and for a shorter duration.
DIAGNOSIS

- Flock performance history, signs, postmortem lesions, and the presence of other related diseases such as gangrenous dermatitis, and hemorrhagic syndrome.
- Low hematocrit values.
DIAGNOSIS

- Virus isolation from infected livers. Inoculate the virus into susceptible day-old chicks.

- Follow with virus neutralization using MSB1 cell culture to detect seroconversion.

- Indirect FA or ELISA tests are also available.
PREVENTION AND CONTROL

- Infection and seroconversion of breeder flocks during the rearing phase.

- Monitor for the presence of antibodies at 10-12 weeks of age.
PREVENTION AND CONTROL

- Artificial exposure is accomplished by the transfer of contaminated litter.

- A commercial vaccine has recently been approved for use in the U.S.
This vaccine is given via the wing web stab between 10 and 18 weeks of age. It should not be administered later than 6 weeks before the onset of egg production.
INFECTIOUS BURSAL DISEASE
(GUMBORO OR IBD)

CLASSIC:

- Acute, highly contagious viral disease of young chickens characterized by edema and swelling of the bursa of Fabricius followed by hemorrhage with vent picking, diarrhea, ataxia and mortality in 3 to 6 wk. old birds.

- This form can result in high mortality.

- Infection prior to 3 wks. of age results in immunosuppression and bursal atrophy due to destruction of undifferentiated lymphocytes.
INFECTIONOUS BURSAL DISEASE
(GUMBORO OR IBD)

VARIANT:

- Causes no obvious clinical disease but produces severe immunosuppression with bursal atrophy regardless of age of infection.
ETIOLOGY

- IBD virus is a birnavirus - a double stranded RNA virus.
- Variant viruses have been described in Delmarva and other broiler production areas. These variants do offer some cross protection with classic viruses, but not visa versa.
- IBD is highly contagious and very difficult to remove from a house. It tends to reoccur on the same farm.
- Virus is very hardy and resistant. It survives for long periods (at least 6 mo.) in poultry houses even where thorough cleaning and disinfection procedures are followed.
ETIOLOGY (CONT.)

- The bursa of Fabricius is an immune organ that produces B lymphocytes which migrate to secondary immune organs (cecal tonsil, Harderian gland, etc.) for the purpose of antibody production.

- Embryos are immunocompetent by 18 days and B-cells begin to leave the bursa at this time. However, full B-cell production peaks at 3 weeks and this is when the birds are most susceptible to the virus and classic IBD infection.
INCUBATION PERIOD

48-72 hours

- Hemorrhaging in the bursa is evident within 3 days of infection.

COURSE OF DISEASE

5-7 days
MORTALITY

Broilers -- 0-20%
Leghorns -- 5-50%

METHODS OF SPREAD

Contaminated feed, water, servicemen and trucks
**SIGNS**

**CLASSIC:**
1. Occurs in broilers at 3-6 weeks of age
2. Depression, ataxia and tremors
3. Vent picking
4. Diarrhea and dehydration
5. Mortality

**VARIANT:**
1. Inapparent infection
2. A variety of other disease problems
COMMENT

- The classic form of this disease is rarely seen today. Birds infected with variant viruses and birds infected previous to 3 weeks of age may not show clinical signs.
POSTMORTEM LESIONS

CLASSIC:
1. Enlarged edematous bursa of Fabricius followed by bursal atrophy.
2. A gelatinous film covers the exterior of the bursa.
3. A cheesy core may be found in the bursa.
4. Kidneys are swollen and filled with urates.
POSTMORTEM LESIONS (CONT.)

5. Extensive intramuscular hemorrhage on the medial surface of the thigh.

6. A rachitic-like condition sometimes seen.

VARIANT:

1. Small, atrophic bursa of Fabricius.
Birds infected with variant viruses and birds infected previous to 3 weeks of age may have bursal atrophy without other lesions.

The variant virus is different from the classic IBD. It causes bursal atrophy at any age but the later the infection occurs, the less immunosuppression occurs. Atrophy may be transient because many B-cells may have already migrated to populate secondary lymphoid organs.
DIAGNOSIS

1. Postmortem lesions
2. Histopathology
3. Viral isolation - bursa, cecal tonsils, spleen
4. Serology - despite the destruction of antibody producing cells, titers to IBD will be high. This test detects antibody for the classic IBD so won’t be as helpful to detect infection with a variant virus.
PREVENTION

1. **VACCINATION** - once on farm, the disease tends to reoccur. It cannot be eradicated. To determine the effectiveness of the vaccine program, evaluate bursas at 21-28 days for the degree of atrophy.

**LIVE ATTENUATED** - in broilers and as a primer for killed vaccine. Live vaccines come in mild, intermediate, and hot forms. The vaccine of intermediate virulence is most commonly used. Bursal derived vaccines are very expensive ($100/1000 doses compared to $40/1000 doses) but they have high antigen titers. The virus is harvested from infected bursas.

**KILLED IN OIL** - Breeders at 12-18 weeks of age.
2. Isolation and strict sanitation. Organic iodine disinfectants help. This disease is usually less of a problem in breeders due improved biosecurity and sanitation.

3. Maternal antibody from the breeder hen will protect the chick for the 1st 2 weeks of life. This antibody protects more thoroughly against disease than do many others. The half-life of the passive antibody is 4-5 days so will be completely gone by 3 wks. of age.
First described in 1963 concurrent with a severe respiratory outbreak in broilers.

1970 - CANADA
DELMARVA
INDIANA
TENNESSEE
ETIOLOGY

VIRUS

- An adenovirus will reproduce the disease in immunosuppressed birds
- Gumboro virus plays a role in the field problem. Lack of maternal antibody will result in an early gumboro infection which leads to immunosuppression.
INCUBATION PERIOD

3-5 days

COURSE OF DISEASE

- Usually seen in birds 3-5 weeks of age and lasts up to 3 weeks.
- Excessive cull rate for 5-6 weeks.

METHOD OF SPREAD

Lateral
CLINICAL SIGNS

1. Birds usually found dead

2. Shanks and comb become very pale

3. Reduced growth rate
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1. Livers swollen, mottled with subcapsular stellate hemorrhage.

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(A) Kidney
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2. VIRAL ISOLATION

   Need SPAFAS eggs. Some embryos will have liver lesions grossly and microscopically.
TREATMENT

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   (4) Inclusions surrounded by a clear halo

2. VIRAL ISOLATION

   Need SPAFAS eggs. Some embryos will have liver lesions grossly and microscopically.
TREATMENT

- Broad spectrum antibiotics and vitamin fortification will diminish the cull rate.
CONTROL

A. Vaccination
   ▸ Attenuated vaccines for Gumboro

B. Decontamination of premise
HEMORRHAGIC ENTERITIS

- An acute highly contagious disease of turkeys 7-15 weeks of age that is characterized by massive hemorrhaging into the intestinal tract and sudden death.

- It most often affects turkeys around 10 weeks of age.
ETIOLOGY

- Virus
- An adenovirus related to marble spleen disease of pheasants.
- Marble spleen disease virus is less pathogenic to turkeys and may be used as a vaccine virus.
INCUBATION PERIOD
5-6 days

COURSE OF THE DISEASE
10-14 days

METHOD OF SPREAD
Unknown, possibly through the litter
SIGNS

1. Short term depression

2. Anorexia

3. Birds die acutely and are in good flesh

4. Causes immunosuppression with secondary *E. coli*.
POSTMORTEM LESIONS

1. Massive hemorrhaging into the lumen of the intestinal tract.
   Blood is very dark in color.
   Birds appear pale due to anemia.

2. Petechial and ecchymotic hemorrhages in the liver, spleen, heart and abdominal viscera.

3. Birds usually well fleshted.
DIAGNOSIS

- Lesions are pathognomonic - intranuclear inclusions in the spleen, kidney, or liver on histopathology
TREATMENTS

(1) Hyperimmune sera

(2) Broad spectrum antibiotics
PREVENTION

- Vaccinate with field attenuated virus at 4 weeks
- Create an autogenous vaccine from spleens or use commercially available vaccines.
POULT ENTERITIS

- A highly contagious disease of turkeys that is characterized by profuse diarrhea and high mortality in young poults.

- A variation called "spiking mortality " has become a major problem in GA and NC adding 2-3 weeks to the growth period.
ETIOLOGY

1. VIRUS

- A coronavirus has been isolated that will reproduce the disease.
- Many other viruses appear to be involved - reovirus, rotavirus and others.
INCUBATION PERIOD

2-3 days. Spreads very rapidly.
COURSE OF DISEASE

2 weeks

MORTALITY

Poults - 10-50%
METHODS OF SPREAD

1. Contamination of litter, feed and water with infected fecal material.

2. Carriers do develop and serve as a source of infection for young poults.
SIGNS

A. POULTS

(1) Restlessness, increased noise
(2) Depression
(3) Anorexia - prominent keel
(4) Brownish diarrhea - flush between 8-10 weeks of age losing 1-2 lbs.
(5) Unevenness, feathering problem
(6) High mortality
POSTMORTEM LESIONS

A. POULTS

(1) Watery gaseous distention of the intestinal tract
(2) Emaciation
(3) Severe enteritis
(4) Spleen reduced in size
(5) Litter in gizzards
(6) Thymus atrophy
TREATMENT

1. Broad spectrum antibiotics (i.e. penicillin)
   Reduce mortality by controlling secondary infections.

2. Electrolytes

3. Increase in environmental heat
PREVENTION

1. Isolation rearing of young poults
2. Strict sanitation
3. Depopulation and clean up to break cycle
4. Vaccine available for reovirus immunization of breeder hens.

Immunity is passed to poults resulting in lower mortality.
VIRAL ARTHRITIS (VA)

- A widespread viral infection of poultry that affects the synovial membrane, tendon sheaths, tendons and myocardium of meat-type chickens, and occasionally turkeys.

- Infection occurs early in life (less than 2 weeks of age) but hens break down when placed in breeder house (usually slatted floors).

- Roasters - over 5 lbs.
COMMENT

- Signs and lesions are most often seen involving the tendons of the hocks and legs because these are weight bearing limbs.

- Many times Staphylococcus becomes involved as a secondary infection of tendon sheaths.
ETIOLOGY

- Reovirus
- First recognized in 1957
INCUBATION PERIOD

- 3-5 DAYS - but usually musculoskeletal disease presents much later in life.
- Enteritis can start to cause nonuniformity of the flock early in life.
- Maternal antibody is important in determining the incubation period. This can prevent the disease if MAT is high for the 1st 3 weeks of the broiler's life.
COURSE OF DISEASE

Chronic
METHOD OF SPREAD

- Via respiratory or digestive routes.
- Transovarian. This occurs when the hens are viremic.
- For a period of 2-4 weeks, there may be poor chick quality and high mortality in the progeny.
MORBIDITY

Frequently 100%

MORTALITY

Usually low - chronic problem
CLINICAL SIGNS

1. Swelling of the tendon sheaths of the digital flexor and metatarsal extensor. A knot is palpable in the tendon above the hock.

2. Affected birds tend to sit on their hocks and are reluctant to move.

3. Rupture of the gastrocnemius tendon. This occurs when birds start to jump on the slats. A large bruise may be visible above the hock.

4. Reovirus induced enteritis will cause diarrhea, feed passage, and poor growth and feed conversion.
POSTMORTEM LESIONS

1. Edema of tendons.
2. Excessive fluid in the hock joint.
3. Rupture of the gastrocnemius tendon and hemorrhaging dorsal to the hock.
4. Erosion of articular cartilage.
5. Staph will often invade afterwards causing purulent arthritis.
DIFFERENTIAL DIAGNOSIS

1. Mycoplasma synovitis.
2. Staphylococci
3. Trauma
DIAGNOSIS

1. **Serology** - 
   AGP & ELISA - interpretation unclear because there are many reoviruses in chickens that do not cause this disease. Reovirus does not produce a strong antibody response.

2. **Virus isolation** - CAM of eggs. Virus infects the birds early in life but lesions develop much later so virus is not usually still present.

3. **Histopathology** - no lesions associated with viral invasion may be visible 10 weeks post infection. Staph comes in and confuses the picture.

4. Best is to use history of company problem and clinical signs.
TREATMENT

TLC
PREVENTION AND CONTROL

1. Depopulate farm and decontaminate.
2. Breeder hens are vaccinated.
3. Both attenuated and killed vaccine are available.
4. Must use live vaccine primer before killed vaccine. Problem breeder flocks may receive 2 live followed by 2 killed vaccines. This is very expensive.
5. Under certain conditions broilers are vaccinated with live vaccines. Can be given with MDV at 1 day of age S.Q.
EASTERN EQUINE ENCEPHALITIS

- Has been described as a "sleeping sickness" in pheasants, chickens, ducks and turkeys.
- Caused by an Arbovirus.
- Transmitted by mosquito vectors.
- Chickens over 3 wks. of age have no clinical signs.
- Diagnosed in embryonated chicken eggs. Public health significance.
EASTERN EQUINE ENCEPHALITIS

- Gross lesions in ratites: diffuse hemorrhagic enteritis - contagious through the digestive tract in birds.

- This is a major problem in emus.
DUCK VIRUS ENTERITIS
(DUCK PLAGUE)

- Acute, contagious herpesvirus infection in ducks, geese and swans characterized by vascular damage with tissue hemorrhage and free blood in the gut and body cavity, with ulcerative intestinal lesion on intestinal mucosa. Liver may be mottled.

- Severe outbreak in Long Island duck raising area of N.Y. in 1967. Diagnosis, typical lesions. Do VN in embryonated duck eggs.
DUCK VIRUS HEPATITIS

- Probably caused by picornavirus. Highly fatal, rapidly spreading viral infection of young ducklings.
- Characterized primarily by hepatitis. Usually die before 3 wks. of age.
- Morbidity 100%, mortality may reach 95% in 1-3 wk. old ducklings.
- Diagnosis - chick embryos using VN test with specific immune sera.
DIFFERENTIAL DIAGNOSIS

Pasteurella anatipestifer
TREATMENT AND PREVENTION

- Serum therapy is highly successful. 0.5 ml antiserum per bird. Can prevent by strict isolation for first 5 wks. (Difficult)

- Immunization: 3 ways
  1. Antiserum
  2. Vaccination of breeders with attenuated strain.
  3. Vaccinate ducklings with avirulent strain of DHV.
PULLORUM DISEASE

- Was once the most important disease in poultry, "Bacillary white diarrhea."

- Regulatory programs for control were developed and administered by National Poultry Improvement Plan: Chickens, 1935, Turkeys, 1943.

- Cooperative state federal program.
Still prevalent and important in certain countries throughout the world. Occasionally in backyard flocks in U.S. and Canada.

In 1986, an outbreak occurred in Missouri when a mail order hatchery bought eggs from backyard flocks.

Pullorum also entered commercial flocks in NC and LA in the early 1990's.
PULLORUM DISEASE

- Controlled by test and slaughter. Eradicated in commercial poultry in USA and Canada.
- Reportable disease.
CAUSATIVE AGENT

SALMONELLA PULLORUM

Non motile - GM (-) rod

Bacteria location

1. Chicks and poults - internal organs, yolk sac and blood stream.

INCUBATION PERIOD
7 to 10 days

COURSE OF DISEASE
2 to 3 weeks

MORTALITY
In chicks and pouls less than 2 weeks old, up to 100%
METHOD OF SPREAD

1. "Carrier" layers - transovarian. This allows eradication.

2. Infected hatchers - automated incubators allowed pooling of eggs and lateral dissemination of pullorum.

3. "Backyard" flocks largest threat in U.S. and Canada.
INFECTED EGGS

- Dead or moribund chicks in hatcher or dead in the shell
- Chick quality problems - related to breeder/ hatchery contaminations.
- MATURE BIRDS - seldom die.
CLINICAL SIGNS

CHICKS AND POULTS

1. Some chicks may be moribund or dead soon after hatch - clinical presentation appears the same whether transovarian or hatchery transmission.

   ▶ Mortality starts at 5-10 days old and peaks at 2-3 weeks of life.
CLINICAL SIGNS (CONT.)

2. Appear cold, anorexia, whitish diarrhea that causes pasted vent. Painful defecation.

  Use caution as heat stress also causes pasty vents.
ADULTS

- Usually without signs
- Fertility and hatchability reduced
POSTMORTEM LESIONS

CHICKS AND POULTS

**Peracute** - lesions absent, rapid mortality.

**Acute**
1. Liver - enlarged, congested, yellow streaks with hemorrhages. Use caution with interpretation because a yellowish tinge to liver is normal in young chicks.
POSTMORTEM LESIONS (CONT.)

2. Omphalitis - solidified yolk. This occurs because the bacteria digests the carbohydrates in the yolk.
   - This produces acid which coagulates the protein.
   - So young birds have problems absorbing the yolk.
3. White nodules in heart, liver, lungs, ceca, large intestines, and gizzard muscle.

4. Kidneys congested and urate filled.

5. Swollen hock and wing joints filled with exudate.

6. Caseous cecal cores.
ADULTS

1. Misshapen, discolored, caseous ova.
2. Nodular pericarditis.
3. Peritonitis with internal ovulation.
4. Testicular abscesses.
DIFFERENTIAL DIAGNOSIS

- Chilling or overheating.
- Omphalitis.
- Other Salmonellas and *E. coli*.
- In adults similar to other septicemic diseases.
DIAGNOSIS

1. **Suggestive diagnosis** - high mortality in chicks and poults during first two weeks of life plus lesions. Look for cecal cores.

2. **Positive diagnosis** - isolation and identification of causative agent. Culture the yolk sac and gut.

3. **Agglutination blood test** - indicates infected breeder flocks.
CULTURE METHODS

See Page 125.
CONTROL PROGRAM

- Voluntary regulatory program (+) reactors must be disposed of under supervision of state regulatory agency.
- Flock usually destroyed.
- Premises decontaminated as per the NPIP.
- Several cases found since 1986 originating from "mail order hatchery" in the mid-west.
SEROLOGICAL TESTING

- Stained antigen whole-blood test accepted by NPIP for chickens, not turkeys.
- Tube agglutination test done after 16 weeks.
- Usually kill infected flock.
CONTROL

- Establish and maintain Pullorum-free breeders.

- Serological testing - stained antigen whole blood test.

- Purchase chicks and poults from hatcheries that participate in NPIP.

- Organism in hatchery can be killed by formaldehyde fumigation.
Non-pullorum reactors (false +) can occur on testing. This problem is overcome by careful bacteriologic exam. of suspicious reactors.

The false positives are usually caused by common cross-reactive antigens possessed by other bacteria.

Salmonella enteritidis has a similar antigen to S. gallinarum and S. pullorum.
Sue Clanton: Insert Salmonella and Arizona Culture Methods
TREATMENT I

- Birds usually destroyed in U.S. and Canada.
Drugs will not eliminate infection from a treated flock, and will perpetuate the carrier state.

MORTALITY CAN BE CONTROLLED WITH:

- **SULFONAMIDES**: i.e. Sulfamerazine. Can't use sulfa in egg hens.

- **ANTIBIOTICS**: tetracyclines, gentomycin, and spectinomycin.

- **NITROFURANS**: effective but illegal in U.S.A.
FOWL TYPHOID

- A septicemia, seen primarily in chickens, also in turkeys, and other avian species. It looks very similar to pullorum disease.

- This is common internationally due to lack of eradication programs.

- This disease can continue for months and can be seen in young adults or mature fowl, due to the stress of coming into production.

- Dramatic drop in outbreaks since NPIP. (1954)

- Reportable.
CAUSATIVE AGENT

1. Bacteria, *Salmonella gallinarum*

2. Closely related to *S. pullorum* but biochemically different

3. *S. pullorum* & *S. gallinarum*
   Antigenically identical (complete cross agglutination) (*S. enteritidis* also cross-agglutinates).
INCUBATION PERIOD
4 to 5 days

COURSE OF DISEASE
5 days in acute cases.
Losses may extend over 2-3 wks. period.
May recover.

MORTALITY
If acute may range up to 50%.
Variable.
METHOD OF SPREAD

1. Lateral from carriers

2. Vertical from carriers: Transovarian

3. Rats and feral birds (buzzards)

4. Human traffic
CLINICAL SIGNS

CHICKS AND POULTS - Similar to pullorum

1. Infected eggs yield moribund and dead chicks
2. Whitish pasty vents
3. Anorexia
4. Labored breathing
CLINICAL SIGNS

GROWING AND MATURE BIRDS

1. Drop in feed consumption
2. Depressed and pale
3. High fever (up to 112°F) - normal body temperature - 104°F
4. Greenish diarrhea - catarrhal enteritis
5. Death in 4 to 10 days after exposure
POSTMORTEM LESIONS

- Same as pullorum with a tendency for enlarged (2-3x) dark spleens in acute cases and mahogany or greenish-bronze livers in subacute and chronic cases.

- Focal areas of necrosis in the heart and grayish lungs are also apparent.
COMMENT

- This disease is usually very acute, and dead birds will be found on the nest and on the floor.

- These birds may not have lesions.
DIFFERENTIAL DIAGNOSIS

- Abscesses in lungs and viscera in chicks unique to typhoid and pullorum.
- In adults similar to other septicemic diseases.
- Reaction to agglutination test helpful in chronic cases.
1. **SUGGESTIVE DIAGNOSIS** - high mortality in hens, showing described lesions.

2. **POSITIVE DIAGNOSIS** - isolation and identification of the causative agent from liver and/or spleen.

3. A positive agglutination test to pullorum antigen will aid in the diagnosis.
CULTURE METHODS

See Page 125.
CONTROL PROGRAM

- Voluntary regulatory program (+) reactors must be disposed of under supervision of state regulatory agency.
- Flock usually destroyed.
- Premises decontaminated as per the NPIP.
Same as *S. pullorum* treatment I (destroy) & II (treat to control mortality).

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PREVENTION

- An attenuated vaccine strain 9R of *S. gallinarum* is being used with success in some foreign countries that do not have successful eradication programs.

- This is not licensed in the U.S.
AVIAN ARIZONOSIS

- Similar to other Salmonella infections, seen mainly in turkeys less than 3 wks. of age.

- Problem has essentially been eliminated in foundation breeders through the use of antibiotics and bacterins.
CAUSATIVE AGENT

Salmonella arizona

- This organism differs from other salmonellas biochemically and serologically.
- It is motile.
INCUBATION PERIOD

4 TO 5 days

COURSE OF DISEASE

- 5 days in acute cases with losses extending over 2-3 weeks.
MORTALITY

- 5-10% in young birds.
- Occasionally up to 60% if exposed at hatching time.
- No mortality in adult birds, they become carriers.
METHOD OF SPREAD

1. Egg shell contamination
2. Transovarian proven in turkeys
3. Hatchery contamination - lateral transmission.
4. Carrier birds, fecal contamination
5. Biological vectors - rats and mice
1. Similar to other Salmonellas - diarrhea, listless, huddling, mortality.

2. Often blindness and nervous signs are seen in poults

3. Opacity of anterior chamber of eye in poults

4. None in adult carriers.
POSTMORTEM LESIONS

- Same as other Salmonellas
- Eye lesions (opacity) in pouls very common.
- Omphalitis
- Cecal cores
DIAGNOSIS

- Isolation and serotyping.
- There is no diagnostic antigen available.
CULTURE METHODS

See Page 125.
TREATMENT

1. POULTS
   (1) Nicholas formula: 1-day-old injection with antibiotics (gentamicin or spectinomycin) + Vit. B complex.
   (2) Same as *S. pullorum* Treatment II.
       Page 126.

2. CHICKS
   Same as (2) above.
PREVENTION

- Use only *S. arizona* negative breeders.
- Concentrate on egg sanitation.
- Pre-incubation fumigation.
- Three times amount of fumigant used for paratyphoids, but this can decrease hatchability.
CONTROL

- Controlled by egg-dipping (Gentamicin).
- Turkey breeders vaccinated at 20-28 wks. Use autogenous bacterins.
- Bacterins - not USDA approved but O.K.*
  *Must be made by owner.
Reinfection may take place at commercial breeder or farm level.
PARATYPHOID INFECTIONS

- All Salmonella infections in poultry other than *S. pullorum, S. gallinarum,* and *S. arizona*.

- They are not host specific and infect many types of animals.

- Public health significance is more important than bird health - these cause foodborne illness.
CAUSATIVE AGENT

1. Many species of Salmonella.
2. Have somatic and flagellar antigens.
3. These Salmonella are motile.
4. Survive and multiply in most environments.
5. *S. typhimurium* most frequently isolated from young poultry. This is more common in turkeys than in chickens.
1. Salmonella paratyphoid: approximately 200 serotypes found in poultry.

2. 10 or 12 serotypes cause most problems.

3. 75% of all poultry infected at some time in life with 1 or more serotypes.

4. Important problem in hatchery sanitation. It serves as a barometer of egg shell and chick quality problems. These organisms are motile so are more common.

   But *E. coli* is a more common chick quality problem than Salmonella.
COMMENT

- *S. enteritidis* important in human outbreaks. It is most severe in old, very young, and immuno-compromised people. Large percentage of the outbreaks related to consuming eggs.

- This organism is found contaminating chicken ovaries. Rats and mice strongly involved in epidemiology.

- *S. enteritidis, S. pullorum, and S. gallinarum* are all type D Salmonella, so testing protocol for pullorum and typhoid also detects *S. enteritidis*. 
ESCHERICHIA COLI INFECTIONS
(E. COLI OR COLIBACILLOSIS)

- Generally a secondary bacterial infection causing many different signs and lesions in chickens and turkeys.

- *E. coli* from avian sources do not cause problems in mammals.

- It is not known whether chickens can carry *E. coli* that are pathogenic to humans. Widespread distribution because it is an intestinal inhabitant.
ETIOLOGY

- The bacterium *Escherichia coli* which is in the Enterobacteriaceae family meaning it is found in the intestine: This organism is coliform, gram negative, and motile.

- Most problems in poultry are caused by somatic antigen serotypes 1, 2, and 78.
E. coli is not part of "normal flora" of seed eating birds. It is more often seen in birds that eat protein of animal origin like poultry.

E. coli causes major economic losses in the poultry industry.
TYPES OF INFECTION

Depend on where primary problem occurs.

1. Respiratory - airsacculitis along with mycoplasma or virus infection. Virus may be vaccinal. Without *E. coli*, the mycoplasma infections would not be significant.

2. Omphalitis - (embryo & early chick mortality) egg transmitted due to penetration from contact with contaminated environment (dirty nest, floor eggs, egg washing, Sweating after refrigeration, and dirty hatching equipment). This is a major chick quality problem.

3. Gastrointestinal - this is seen in turkeys and is usually primary but predisposed by contaminated feed, water or crowding.
OTHER E. COLI CAUSED CONDITIONS

ACUTE OR CHRONIC SEPTICEMIA INVOLVES MOST ANY BODY TISSUE

1. Bordetella infection in turkey coryza occurring at 2-4 wks. of age results in secondary invasion by *E. coli* with increased mortality at 6-8 wks. of age.

2. Coligranuloma - usually liver, spleen and intestines.

3. Blackhead - secondary to *histomonas*.

4. Arthritic infections.

5. After HE in turkeys.
METHOD OF SPREAD

- Very ubiquitous organism.
- From contaminated environment.
- Mostly through fecal contamination.
INCUBATION PERIOD

- 72 hrs. experimentally in 1 day old chicks.
- Under field conditions variable depending on the primary infection.
MORTALITY

- Variable: Usually high in acute conditions but negligible in chronic problems.

- Causes the mortality in turkey coryza (Turkey bordetellosis).
COURSE OF DISEASE

- Omphalitis in chicks and poults and enteritis and hepatitis in turkeys are usually acute.
- Other conditions such as infectious process, salpingitis, coligranuloma, and air sac disease are usually chronic.
SIGNS

- Variable depending on system infected.
- Cyanosis in cases of respiratory disease.
POSTMORTEM LESIONS

1. Omphalitis
2. Airsacculitis, peritonitis, perihepatitis, and pericarditis
3. Enteritis
4. Coligranuloma - looks like blackhead
5. Salpingitis
6. Arthritis and synovitis
7. Infectious process
**E. COLI**

DIFFERENTIAL DIAGNOSIS

1. Mycoplasma
2. Respiratory virus - NCD, IB
3. Staph infection
4. Fowl Cholera
5. Blackhead
6. Erysipelas
7. Salmonella
8. Other bacterial septicemias
E. COLI IN TURKEYS

Usually brought on by stress such as crowding and heat. Usually from 3 to 12 wks of age.

LESIONS -
1. Enteritis - dark duodenum with purple spleen in dead birds.
2. Pericarditis
3. Turkey coryza
   Bordetella + E. coli.

Can occur secondary to hemorrhagic enteritis.
TREATMENT

Don’t treat chicks with omphalitis - most *E. coli* are resistant to everything.

Sulfa drugs and antibiotics.

Run sensitivity study.

*E. coli* resistant to most drugs available for use in poultry.
1. Good egg sanitation - decreases omphalitis and I.P. (pre-incubation fumigation).

2. Don't use floor or dirty hatching eggs.

3. Use M.G. clean stock.

4. Good, low stressful management.

5. Continuous medication - not used much today as it can cause other problems.
STAPHYLOCOCCUS INFECTIONS

These infections are usually seen in poultry as "navel ill" (omphalitis), arthritis, and "bumblefoot".

- A differential diagnosis for this would be *E. coli* or Salmonella.
- Not highly contagious, causes problem as chronic condition. Secondary infections occur due to poor management.
- High mortality seen with omphalitis from contaminated hatchers.
- Prevalent problem in replacement broiler breeders and turkeys, as arthritis.
CAUSATIVE AGENT

Bacterium, *Staphylococcus aureus*

- Gram pos. (+) cocci occurring in clusters
- Most pathogenic staphs are coagulase positive (+).
- Very ubiquitous organism that is an opportunist.
- Infection usually follows traumatic or biological injury.

i.e.: genetic stress in turkeys
(stress of large breast size on hock joints causing “Cow hocked” stance); restrictive feeding; coccidial outbreaks; wing web inoculation; viral arthritis; moving and handling.
Replacement broiler breeders between 8-18 weeks, due to every-other-day feeding.

- Staph problems often occur following a coccidiosis outbreak, and again between 20-24 weeks after birds are moved to laying quarters and onto slated floors due to trauma.

- Replacement broiler breeder males which are put onto slats and fed 12% protein restrictive rations.
INCUBATION PERIOD

Three to four days in omphalitis.

Two to three weeks with bumblefoot and arthritis.
COURSE OF DISEASE

One week to several weeks in chronic cases.
METHOD OF SPREAD

1. Primarily through injuries which allow entrance of organism.

2. Egg shell contamination in breeder house.

3. Contaminated hatchers - failure to fumigate properly.

4. Oral entrance possibly.

5. Turkeys - entrance through respiratory system.
MORTALITY

1. Suppurative synovitis/arthritis in replacement broiler breeder males may reach 4% per week between 20-24 wks.

2. Bumblefoot: usually low 3-6%. Debilitation is the problem.

3. Omphalitis: 30-40% mortality within 3 to 4 days after hatching. 100% of the affected birds will die.

4. 10-15% in laying hens acutely infected with septicemic "staph".
CLINICAL SIGNS

IN CHICKS

1. High mortality in 72 hours.
2. Large scab on navel.
4. Very putrid odor.
CLINICAL SIGNS

ADULT CHICKENS

1. "Bumblefoot" - injured footpad with severe swelling (abscess).
2. Arthritis and synovitis: In slatted houses.
3. Occasionally systemic (septicemia) but this is rare.

TURKEYS - Usually in growing toms

1. Birds reluctant to move and are crippled.
2. Swollen joints, synovial sheaths and bursas.
POSTMORTEM LESIONS

1. "Bumblefoot" - usually there is a wound with caseous suppuration in an organized abscess. Common in backyard flocks.

2. Omphalitis - retained yolk in emaciated baby chicks or poults. The infection has putrid odor.

3. Arthritis - suppuration in the joints and also, around synovial sheaths,
   e.g., tendon sheaths of the hocks, gastrocnemius tendon, stifle, and wing joints.
4. Breast blisters (synovial bursa over sternum) filled with suppurative exudate. Associated with coarse litter.

5. Septicemic "staph" lesions are similar to other bacterial diseases but usually have dark brown or greenish edges on the liver lobes. This has been related to tibial dyschondroplasia and turkey osteomyelitis complex.
DIAGNOSIS

SUGGESTIVE -

1. Swelling in joints or foot pads with suppuration present. (White & creamy occasionally with flocculence)

2. High mortality in chicks with swollen abdomens and retained yolk sac (putrid odor) (second most common after *E. coli*), swollen foot pads and joints.

3. Breast blisters are filled with creamy pus.
4. Osteomyelitis in tibial and femoral growth plates. Discrete 1-2 mm abscesses may be found in marrow cavity. Related to TD.

- TD will resolve with time but often associated with green livers.

5. In septicemia, "staph" lesions are similar to those seen in fowl cholera or any other septicemic disease. This is rare.

**POSITIVE** - Isolation and identification of the causative agent.
DIFFERENTIAL DIAGNOSIS

- The symptoms and lesions of arthritis are very similar to those found in infectious synovitis (MS) and chronic fowl cholera as well as other bacterial diseases.

- Must culture to differentiate.
CULTURE METHODS

STAPHYLOCOCCUS AUREUS

→

Visceral organs (liver, spleen, etc.)
and hock exudate (swab)

→

Blood agar
White to yellow colonies
Hemolytic variable

→

Gram (+) cocci

→

Coagulase test (+) or (staphyloslide* test)
Staphtrac™ - biochemical test strips
Sensitivity tests should dictate drug being used. Penicillin is usually effective.

Drugs of choice: Novobiocin (expensive), Penicillin

Others used: Chlortetracycline, Oxytetracycline, Erythromycin

Use broad spectrum antibiotics for omphalitis at the rate of 1-2 gms per gal of water for 7-10 days.
REPLACEMENTS ONLY:

- Potassium penicillin G water soluble powder must be used for 7-10 days in replacements.

- Retreatment may be necessary before placement in laying houses.  
  (No chicken clearance)
PREVENTION

1. Remove sharp objects from the litter and premises. Correct rough turkey ranges.

2. Cleanliness and fumigation of incubators.
3. In turkeys, treatment of arthritis is actually a prevention method.

4. No vaccine available.

5. Anticoccidial & restrictive feeding programs in replacements can have impact on stress and resultant staph. infection.
FOWL CHOLERA

- All species of fowl affected.
- Both acute (primarily in turkeys) and chronic (primarily in chickens) infections occur.
- Young adults and mature birds usually affected.
- This rarely occurs in broilers but when it does, it is usually associated with a rodent control problem.
CAUSATIVE AGENT

- *Pasteurella multocida*
- Bipolar staining gram (-) rod.
- The most important bacterial infection in turkeys.
- Will survive 3 months in carcass.
- Found on membranes of healthy birds.
Different strains occur.

MOST COMMON SEROTYPES:
X-73 = serotype 1 - chickens and turkeys
P1662 = serotype 4 - chickens and turkeys
P1059 = serotype 3 - turkeys
CU (Clemson university) vaccine = Crossing strain - 3 x 4

Pathogenic serotype most prevalent (80%) at present - non-vaccinal 3 x 4 - chickens and turkeys
Pasteurella anatipestifer (ducks),

*P. haemolytica* (colonies have β-hemolysis),

*P. gallinarum* (in mixed chronic respiratory infections) are also pathogens and may cause disease in poultry and waterfowl.
INCUBATION PERIOD

Four to nine days.

COURSE OF DISEASE

- Acute about two weeks, becomes chronic and may kill birds for several months.

- During the acute phase, mortality may be quite high.
MORTALITY

- Acute - up to 50%
- Chronic - 1-2% - develop swollen wattles and purulent joints.
- Death rate usually higher in turkeys than in chickens.
- Cholera usually occurs in older birds: >10 weeks in turkeys, and in broiler breeders.
METHOD OF SPREAD

1. Exact method unknown. Stress will exacerbate.

2. Biological vector suspected to be main method. (Cats and rodents)


4. Once established in flock respiratory route & water contamination. Large numbers of organisms are shed.

5. Cannibalism in turkeys.
CLINICAL SIGNS

PERACUTE:

(1) May be found under roost or dead in the nest with no symptoms.

(2) Rapidly mounting mortality.

(3) Cyanosis of comb and wattles or whole head of turkey.
CLINICAL SIGNS (CONT.)

ACUTE:

(1) Depression, anorexia, and fever. Sick turkeys on range will hide along a fence or near trees.

(2) Catarrhal nasal discharge and respiratory rales.

(3) Greenish or yellow diarrhea.

(4) Cyanosis.

(5) CNS signs occasionally in turkeys and chickens.
CLINICAL SIGNS (CONT.)

CHRONIC:

(1) Usually occurs in chickens after acute outbreak.

(2) Edematous wattles — later become caseous.

(3) Swollen joints with yellow caseous pus.

(4) Torticollis — localizes in inner ear.
POSTMORTEM LESIONS

PERACUTE:

- May be no lesions, or increased incidence of internal layers and egg yolk peritonitis in laying hens.

- This is a good place to culture the causative organism.
POSTMORTEM LESIONS (CONT.)

ACUTE:

1. Small hemorrhages on heart surface, gizzard, and abdominal fat.
2. Severe congestion of duodenal mucosa.
4. Liver - congested, brown or yellow brown with or without pin point necrotic areas.
5. Tenacious mucous in mouth and nasal passage, especially in turkeys.
6. After 1 or 2 days, consolidation of lungs in turkeys. This is almost pathognomonic in turkeys but rare in chickens.
POSTMORTEM LESIONS (CONT.)

CHRONIC:

1. Caseous exudate in swollen wattles in chickens.

2. Suppurative exudate in swollen joints and tendon sheaths. (May become caseous).

DIAGNOSIS

SUGGESTIVE - Sudden rapidly mounting mortality, hemorrhages of heart, gizzard and abdominal fat, consolidated lungs, purulent wattles, and septic livers.
DIAGNOSIS

**POSITIVE** - Isolation and identification of causative agent. Brain and bone marrow are the best places to isolate the organism.
1. Fowl cholera: *Pasteurella multocida*

Brain, bone marrow, liver, spleen, and lungs. Bone marrow in DOA’s - THIS IS THE LAST PLACE FOR POSTMORTEM CONTAMINATION TO OCCUR.

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<thead>
<tr>
<th>Sugar</th>
<th>Mannitol</th>
<th>Lactose</th>
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DIFFERENTIAL DIAGNOSIS

Dependent on bacterial cultural method.

Must differentiate from:

**ACUTE**: Fowl typhoid, Colibacillosis, systemic Staphylococcus, Psittacosis and Erysipelas (in turkeys).

**CHRONIC**: Infectious synovitis, localized Staphylococcus, Salmonella in joints, Psittacosis, etc.
TREATMENT

- Treat in the water if possible.
- Drug of choice - Sulfadimethoxine (Albon) use as directed by drug company. Preslaughter withdrawal 5 days.
- Sulfaquinoxaline is effective but toxic - withdrawal 10 days.
Sulfa drugs cannot be used in laying hens so they need to be well protected.

Sulfa drugs will reduce production in laying birds.

Also, will reduce hatchability in eggs from breeding birds.
Other sulfa drugs such as Sulmet, triple sulfa. Use as directed.

- Antibiotics - choice terramycin or chlortetracycline 200-400 gm per ton of feed for five days; 400-500 mg/gal of water for five days.

- Can also use K penicillin in the water.

- Erythromycin, follow label recommendations.

May also be injected.
INJECTIONS:

Penicillin - Streptomycin combination 25 mg
Dihydrostreptomycin and 500 units Procaine
Penicillin G for each pound of body weight.

CAUTION - never exceed 75 mg Strep and 1500 units Pen in a single dose for chickens or 500 mg Strep in turkeys.
PREVENTION / TREATMENT

- If the turkeys are 12 weeks of age or less, vaccinate with live vaccine and treat in 4 or 5 days.

- Birds should not be treated 72 hours previous to vaccination or for 4 days post vaccination.

- Do not treat birds just prior to going to market.
PREVENTION

- Live field attenuated strain - CU strain good results in turkeys and chickens.

- Vaccinate turkeys in drinking water at 6-7 wks. prior to movement into the growout facility.

- Then every 3-5 weeks (once a month). Make sure watering systems are free from chlorine which will inactivate the vaccine.

- No. of vaccinations depends on individual farm status.
M-9 - attenuated CU - used in turkeys and chickens. So mild it is not recommended.

Less immunity than CU but also less side effects.

All live vaccines must be injected in chickens. (Wing web route). Oral vaccines given in the water are used in turkeys.

PM-1 vaccine now available. This is a temperature sensitive mutant which is less virulent than CU, but more virulent than M-9. Use this vaccine in poults with pre-existing conditions. Follow up with a more virulent booster.
PREVENTION (CONT.)

- Killed vaccine - limited success in turkeys. Inoculated I/M or S/Q at 10 wks. or older and boost from 3 to 10 wks. later.
- Both aqueous and oil emulsion products available. Usually used in layer or breeder chickens.
- Better results are obtained if a live and a killed vaccine are given in either order than if two live or two killed are used.
- AUTOGENOUS BACTERINS
Live vaccines give a broader spectrum of protection. Vaccinate hens with live vaccine by the wing web "stick" method. Use PM-1 or CU strain vaccine and pox inoculation needle.

CU vaccine will slightly increase the number of cull birds due to the development of joint problems in hens.

Good plan to use CU along with a bacterin in heavy breeders and commercial layers.
PREVENTION (CONT.)

- Break cycle of infection with depopulation, follow by thorough cleaning and disinfection of the house and equipment.

- PROPER RODENT CONTROL

- Allow at least 30 days between flocks.
ERYSIPELAS INFECTION

- Important in commercial turkeys on range.
- It is less of a problem in turkeys reared in confinement unless there is a paddock area. The organism gets into the soil.
- Same organism as in swine.
- Septicemic disease.
- Usually seen in young adult tom turkeys in wet weather.
- Zoonosis.
CAUSATIVE AGENT

- Bacterium, *Erysipelothrix rhusiopathiae*

- Small gram (+) rod that grows in pinpoint colonies on artificial media.

- Alpha hemolytic – 2 or 3 days.

- Can be stained in blood smear of infected birds.
INCUBATION PERIOD
Two to five days.

COURSE OF DISEASE
Two to three weeks.

MORTALITY
From 2-25%.
METHODS OF SPREAD

1. Contaminated soil primary source.
   This is a problem for flocks raised on the range.

2. Other domestic animals.

3. **Fighting among males is the principal means of spread in a flock.**

4. Biological and mechanical vector such as flies, rats, etc.

5. Artificial insemination in turkey hens.
CLINICAL SIGNS

1. Sudden onset with depression and mortality.

2. Turkeys from 4 to 7 months usually.

3. Usually in males. Females can get it through artificial insemination.

4. Yellow or greenish diarrhea.

5. Cyanosis of head.
6. Swollen reddish snood is quite characteristic.

7. Swollen hock joints occasionally.

8. Thick nasal exudate is common.

9. "Brush mark" hemorrhages on head and face.

10. Hens die 4-5 days after AI.
Erysipelas is primarily a wound infection that becomes septicemic.

Probably spread through the digestive tract or AI in breeders in epidemic cases in turkey hens.
COMMENT

- Erysipelas will infect humans and cause an Erysipeloid.

- The disease is also thought to be related to arthritis in humans.

- WEAR GLOVES TO NECROPSY TURKEYS!
POSTMORTEM LESIONS

1. Hemorrhages in muscles and on the heart, lungs, spleen, gizzard and small intestines.

POSTMORTEM LESIONS (CONT.)

3. Liver congested, engorged with blood and friable, focal necrosis occasionally. May see bacteria in Kupfer cells with impression smear of liver.

4. Spleen - congested and dark purple.

5. Congestion of duodenum.

6. Endocarditis -- especially in vaccinated turkeys.
PRESUMPTIVE DIAGNOSIS

- Gram-positive pleomorphic rods in stained smears of the liver, spleen, heart blood or bone marrow.
DIAGNOSIS

SUGGESTIVE -

1. Flat mortality curve in toms.
2. Swollen snood in males.
3. Yellow to green diarrhea.
4. Extensive hemorrhage.

Must differentiate from Fowl cholera.
DIAGNOSIS

DEATH CURVE

CHOLERA

ERYSIPELAS

2-3 birds per 1000 per day mortality.
Normal mortality 1/1000.
DIAGNOSIS

- POSITIVE - Isolation and identification of the causative agent.
ERYSIPELAS: *Erysipelothrix rhusiopathiae*

Visceral organs and bone marrow (DOA)  
(liver, spleen, etc.)

Bl. agar

Pinpoint colonies after 2-3 day  
Alpha hemolytic

GRAM (+) ROD

TSI

$H_2S$ along line of stab in 6-24 hours. Acid in surrounding media. Non-motile.
TREATMENT

1. 10,000 unit K or Na penicillin IM or SC in visibly sick birds — hospital pen.

2. 1,000,000 - 1,500,000 units pen/gal in drinking water for 4 or 5 days -
   Most effective and legal.

   ▶ Bacterin + No. 1 above in sick birds.

   ▶ Move to new range.
TREATMENT (CONT.)

- **PENICILLIN** - 200 gm/ton for 2-3 weeks less effective than injections.

- **TETRACYCLINES** - 200 gm/ton 4-5 days.

- **ERYTHROMYCIN** - 2 gm/gal; 100 gm/ton for 5 days. Can also inject.
PREVENTION

‣ De-snood tom poults in hatchery may or may not be helpful.

‣ Do not use infected range. Try to wait 2 or more years between the use of ranges.

‣ Confinement rearing.

‣ Vaccinate – in high risk areas.
PREVENTION

- Bacterin available for use in high risk areas.
- Vaccinate market toms 1 time at 12 to 16 weeks. Toms are usually marketed at 18-19 weeks of age.
- Breeders - hens and toms 2 times — first time at 16 to 20 weeks. A second done 4 to 8 weeks later.
- Live vaccine now available.
- Recommended vaccinate 3 times in water 4 weeks apart.
- Expensive at $0.35/dose
CHLAMYDIOSIS

- Called ornithosis in nonpsittacine birds, psittacosis or parrot fever in psittacine birds and man.
- **Turkeys** - most commonly affected poultry. Important in psittacine birds, parrots, cockatiels, etc.
- **Man** - turkey processing plant workers in the picking room or on the evisceration line. Also cage birds such as parakeets.
CAUSATIVE AGENT

- **CHLAMYDIA PSITTACI** - specialized bacteria (Rickettsia) that is an obligate intracellular parasite. Lacks own enzyme for reproduction.
- Size: 0.3 to 1.5 microns in diameter.
- Psittacine bird = high toxigenic.
- Turkeys = low toxigenic or high toxigenic.
- Usually in combination with another disease condition - Pasteurellosis.
COMMENT

- Can be seen with light microscopy. If impression smears of infected tissue (airsac, liver, spleen) are stained with Giemsa, Machiavello, or Gimenez method.

- Appear as multiple intracellular elementary bodies in the cytoplasm of host cells with stains & FA conjugate - the most common method of detection.
INCUBATION PERIOD

- Variable - according to strain, species and age.
- Turkeys - high virulence = 6-8 days
- Psittacine - 3-29 days experimental
  41-106 days natural.
- Man - 5 to 16 days.
COURSE OF DISEASE

- Variable depending on virulence of organism. May be silent infections that become acute upon stress to host.
- Can be acute or chronic.
- Will persist for long periods in convalescent host which may be carrier host.
MORTALITY

- Variable - depending on host, strain of organism and particularly the age of the host. The younger the birds, the greater the mortality.

- In turkeys, may range from 10-30% in toxigenic strain -- 1-4% in less toxigenic which is more common.
METHOD OF SPREAD

- Not known for sure - possible carrier host.
- Problem has been related to migratory shore and wading birds (seagulls). Surface water frequented by such birds.
- Airborne in dried excreta.
- Contaminated premises.
CLINICAL SIGNS

Depends on virulence of organism.

1. Depressed, off feed, with emerald greenish diarrhea due to the presence of bile.
2. May take 2-8 weeks to produce signs in large numbers of birds.
3. At peak, 50-80% flock show clinical signs with toxigenic strains - 5-20% show signs with less toxigenic strains.
4. Birds may have characteristic “resting” attitude - raised tail, resting on breast.
POSTMORTEM LESIONS

ACUTE:

1. Pericardial membrane is thickened, congested, and coated with fibrinous exudate.
2. Heart may be enlarged and covered with thick yellowish flocculent exudate.
3. Lungs are diffusely congested and pleural cavity may contain fibrinous exudate.
POSTMORTEM LESIONS (CONT.)

4. The liver is enlarged and congested and may be covered with a pseudo-membrane.

5. There is usually severe airsacculitis mainly in thoracic area.

6. The spleen may be enlarged, 2 to 4 times, dark and soft or have gray-white spots.
In less severe infections and chronic cases: Lesions less pronounced

1. Enlargement of liver and spleen.
2. White focal areas in liver and spleen.

In parakeets - airsacculitis in thorax
   - enlarged liver and enlarged mottled spleen
   - emaciated but eating
DIFFERENTIAL DIAGNOSIS

- *E. coli*
- Cholera - without lung lesions
- Mycoplasma
**DIAGNOSIS**

**SUGGESTIVE** - lesions with antibody titer of 1:64 or more.

**POSITIVE** - the organism can’t grow on media. It needs a host’s enzyme system to grow and multiply. Isolation & identification usually done in embryonated eggs or mice, or upon demonstration of a four-fold rise in titer between acute and convalescent serum.

- Visualization of elementary bodies by special stain on impression smear.
This is a reportable disease.

If chlamydiosis is suspected, you should immediately contact the state health department. If you see psittacine birds in your practice that you are suspicious of, contact the state health department. They will tell you what to do.

Always wear gloves and a mask when “posting” suspect birds.
TREATMENT IN TURKEYS

- Diagnosis suggestive of chlamydiosis - use 400 G. chlortetracycline/ton until confirmed if high toxigenic or low toxigenic.
  - High = 400-800 gm/ton for 3 weeks
  - Low = 200-400 gm/ton for 3 weeks
- Must sterilize infection before processing.
- USDA involved with determining treatment in turkeys.
TREATMENT (CONT.)

PSITTACINE BIRDS

- 70% for sale have been treated. To sterilize unknown population use chlortetracycline - impregnated millet, 0.5 mg/gm for 15 to 45 days.
- Sick bird - suspect psittacosis.
- Give doxycycline by gavage 8-12 mg/lb. twice daily for 5 days, then CTC in feed for 30-40 days.
CONTROL

- Place new turkeys on clean premise - free of excreta contaminated by previous infected flocks.
- Separate diseased birds & excreta from well birds.
- Isolate sick birds.
- Treat sick bird promptly & thoroughly.
CONTROL (CONT.)

- Serologically test flock close to infected flocks.
- Prevent wild & feral bird exposure.
- Imported birds should be quarantined and fed tetracycline impregnated feed (can’t get high enough levels in the water).
- Treat birds an additional 15 days after release from quarantine.
PREVENTION

TURKEYS
  ▸ Vaccination not effective as yet.
  ▸ Research in progress.

PSITTACINE BIRDS
  ▸ Described under treatment.
SYNONYMS:  "Alcaligenes"  
Alcaligenes Rhinotracheitis  
Turkey Bordetellosis

- Mild contagious upper respiratory disease of young turkeys (1 to 6 wks old; possible up to 20 weeks).
CAUSATIVE AGENT

*BORDETELLA AVIUM*

Related agents include:

*Bordetella avium*-like
*Bordetella bronchiseptica*
*Alcaligenes faecalis*
CAUSATIVE AGENT (CONT.)

- Small gram (-) motile rod that grows in pinpoint grey smooth colonies.
- Similar to the organisms which cause atrophic rhinitis in pigs
- Kennel cough in dogs
- The bacteria produces toxins which degrade the cartilage
INCUBATION PERIOD: 7 to 9 days

COURSE OF DISEASE: 12 weeks
(peak 2-3 weeks after onset)

MORBIDITY: Up to 100%

MORTALITY: Uncomplicated = 1-5%

With *E. coli* or *Pasteurella* = up to 60-75%
severity increases with environmental stress also.
METHODS OF SPREAD

1) Direct contact - nasal and ocular secretions
2) Contamination of water. Most turkeys still use trough waterers, although some are now using nipples in brood period which should decrease the problem.
3) Recovered birds can carry infection (important means of spread on multiage farms). This is common on turkey farms.
4) Human traffic - from older to younger birds.
5) Wild birds may act as reservoirs
COMMENT

- *B. avium* will damage the thymus and alter immune function resulting in immunosuppression.
- Birds that have been infected will respond poorly to subsequent vaccination.
- Use of *Pasteurella multocida*, NCD, or HE vaccine in infected poults will result in mortality in response to the vaccine & susceptibility to subsequent challenge.
CLINICAL SIGNS

Non specific respiratory signs:

1. Voice change (loss of voice)
2. Snicking, flicking of head
3. Accumulation of mucus around nares and dirty shoulders
4. Huddling and depression, reduction in appetite, decreased weight gain. There can be severe variation in size of survivors which causes problems at processing.
CLINICAL SIGNS (CONT.)

5. Swollen sinuses, submandibular swelling
6. Conjunctivitis (foamy)
7. Dyspnea - acute
8. Rales - only sign in older birds
POSTMORTEM LESIONS

1) Catarrhal sinusitis and tracheitis (upper 1/3)

2) Trachea may collapse (weaken tracheal cartilage due to toxins)

3) Rarely tracheal plugs - due to ciliary malfunction

4) Submandibular edema

5) Secondary *E. coli* infections with pericarditis, perihepatitis, airsacculitis will cause mortality 4-5 weeks later
DIAGNOSIS

SUGGESTIVE

1. History: primarily on multi-age continuous confinement operations. Will spread from older to younger birds.

2. Clinical signs & postmortem lesions
SEROLOGY - May be useful (paired sera)
   ELISA not widely used

MUST DIFFERENTIATE FROM
   Cholera, NCD, Cryptosporidiosis
   MG, AI, Aspergillosis & Colibacillosis
DIAGNOSIS (CONT.)

POSITIVE

- Isolation and identification of causative agent from tracheas, sinuses, tracheal swabs (live birds)
- Will grow on MacConkey’s media.
# Differentiation of Causative Agents

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<th>Malonate</th>
<th>Urease</th>
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<td>B. avium</td>
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<td>B. bronchiseptica</td>
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<td>A. faecalis</td>
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<td>B. avium-like</td>
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PREVENTION

1) Clean out, wash & fumigate house with formaldehyde
2) Sanitize waterlines and water supply as soon as turkeys are moved
3) Litter management - remove wet spots, control dust
4) Maintain good ventilation
5) Control human traffic (young to old turkeys always)
POULTRY HOUSE
WATER SANITATION
6) Prevent contact with wild birds - screen them out
7) Cull sick birds in convalescent flocks
8) Vaccination - won’t work well in presence of virulent strain, so clean up house.
   Artvax™ temperature sensitive mutant - so only grows in the upper respiratory tract - NC strain
   → 1d intraocular or spray - use
       Flashlight and do at night.
   → 14d water
   Effectiveness questionable
9) Monitor and control darkling beetle populations
TREATMENT

1. Conventional antibiotic therapy **not** effective (organism on surface of mucosa)
2. Tetracyclines & sulfas may help reduce losses due to secondary infections
3. Remove birds from infected house if possible
4. Improve air quality - ↑ Ventilation
   ↑ Temperature
   ↓ Crowding
INFECTIOUS CORYZA

- Rapidly spreading respiratory disease found primarily in chickens.
- All affected birds will be showing signs by the third day.
- Modern management methods have reduced the incidence, still a problem in congested poultry populations.
- All-in/all-out management practices are recommended.
- Avoid multiple-age farms if possible.
CAUSATIVE AGENT

- Bacterium *Hemophilus paragallinarum*.
- This organism is quite fragile and requires "carrier" birds to transmit the disease under field conditions.
- Surviving hens will remain carriers.
This disease is quite important the world over in tropical climates.

In the U.S., it is found mainly in California and Florida in commercial flocks, but may be found most anywhere in backyard flocks.

It has occurred in South Georgia.
Infectious coryza occurs in broilers in many tropical countries such as Central and South America, Southeast Asia, and Africa. It usually occurs after 4 wks. of age and cannot be prevented by vaccination. Weight loss and low feed conversion are the results of infection. These birds appear sleepy and sit around the walls. Sinuses do not swell much in broilers. It may cycle through gamecocks.
One problem with this disease is the amount of debilitation that results in a flock.

The cull rate with this condition may run as high as 20%.

Very costly in started pullets.

Pullets are usually affected when they come into production. This will decrease egg production.
COMMENT

- If laying hens become infected 40-50% productions losses are common.
- Foreign countries where infectious coryza occurs usually have Mycoplasma infections also.
- Together these conditions are quite debilitating and cause severe economic losses.
INCUBATION PERIOD

One to three days.

COURSE OF DISEASE

- One to three weeks or may become chronic and persist for several months.
- Aggravated by cold, damp weather.
- Secondary infection causes chronic condition.
MORTALITY

- Usually low, but poor management and unsanitary condition as well as secondary infection will increase mortality.
METHOD OF SPREAD

1. Carrier birds.
2. Direct contact and airborne droplets.
3. Contaminated water spreads the disease once it is established in the flock.
CLINICAL SIGNS

1. Clear nasal discharge that becomes thick and purulent.
2. Severe unilateral or bilateral swelling of infraorbital sinuses, with eyes completely closed. Foam may accumulate in the corner of the eye due to blocked ducts. The turbinates are normal. Some swell so severely they appear bruised.
3. Yellowish dry crust around nasal opening. Secondary bacterial infections in the sinus is common.
4. Dyspnea. (Difficult breathing).

5. Offensive odor of the nasal discharge.

6. Broilers get very sleepy and may lose about 1 wk. of growth and have high condemnation rates at processing.
POSTMORTEM LESIONS

1. Tenacious, white to yellow pus and mucus accumulation in the infraorbital sinus.

2. Airsacculitis in chronic cases.
DIAGNOSIS

- **SUGGESTIVE** - Fast moving respiratory disease that produces swollen faces.

- **POSITIVE** - Isolation and identification of causative organism. Can inject sinus exudate in susceptible chicks and get typical lesions in two or three days. It is easier to culture the organism in these birds just beginning to show signs, before other bacteria invade.
Infectious Coryza; *Hemophilus paragallinarum*

Sinus exudate - best to culture birds that have just come down with the disease before secondary bacteria can invade.

Bl. Agar
Cross streak with staphylococcus  Provides “V” factor or NAD

Incubate - 10% co$_2$ tension

For 48 hours

Satellite phenomenon - small colonies next to staph streak

Gram (-) pleomorphic rod  catalase - negative

*H. avium* is catalase positive but is non-pathogenic.

To improve the chance of isolating the bacteria, flush the affected sinuses with saline and inject the rinsate into sinuses of healthy susceptible leghorns. In 3-4 days, sinus swelling should be visible. Culture these sinuses to get pure coryza.
TREATMENT (CONT.)

- Most commonly used sulfa drug at present is Sulfadimethoxine (Agribon) in the water and/or Rofenaid™ in the feed.

- Cannot use sulfa drugs in birds laying eggs for human consumption.
TREATMENT (CONT.)

- INJECT - Dihydrostreptomycin sulfate
  0.2 grams per hen.
- Sulfa drug mixtures in water.
- Erythromycin in the water at the rate of 2 grams per gallon or injected as directed on label. This drug cuts down on spread when used in the water. This allows development of immunity but still get egg production drops and carrier birds.
- Cannot use these drugs on hens laying eggs for human consumption.
1. Complete segregation of pullet stock from mature birds.

   - All in all out farming breaks the cycle.
   - If a farm is infected, you must depopulate to eradicate the disease.
PREVENTION (CONT.)

- California method - multiple age farms.
- Procure pullets at 16 wks, vaccinate with yolk bacterin, allow to become infected, treat with erythromycin or sulfa drugs, cull before they come into production. This is a common practice in the Caribbean.
- By exposing the birds as pullets, production drops associated with infection during lay can be avoided.
PREVENTION (CONT.)

- A bacterin is available, Coryza-o-vac, Solvay Laboratories, developed at UGA.
- This works well in older birds.
- Given 2 to 3 times depending on exposure situation.
- Immunity is very short lived due to adaptability of the organism.
- Japanese also produce a bacterin.
- Local bacterins produced in many countries.
ULCERATIVE ENTERITIS

- Has been limiting disease to the commercial bobwhite quail industry.
- Occurs in ground raised birds.
- Japanese quail (coturnix) are resistant.
- Occasionally seen in white leghorn pullets and turkeys secondary to other conditions such as coccidiosis.
- This disease is not seen in broilers.
ETIOLOGY

- **QUAIL** - *Clostridium colinum*
  Anaerobe sporeformer

- **CHICKENS** - *C. colinum* + intestinal stress such as coccidiosis. Quail usually on ground or reared on contaminated wire floors. UE usually a secondary infection to *Eimeria brunetti*, *E. tenella* or *E. necatrix* in chickens. These forms of coccidiosis disrupt the lamina propria and the deep lesions result in bloody feces.
COMMENT

- Ulcerative enteritis seldom seen in broilers because of the good coccidiosis control program usually used in broilers (coccidiostats).

- Most often seen in leghorn pullets because of weak coccidiostat program.

- If quail are left in contaminated flight pens for 2 weeks, they will start dying.
INCUBATION PERIOD

- **QUAIL** - as soon as 18 hours usually 1-3 days.

- **CHICKENS** - 48 hours after inoculation with UE in birds previously infected with coccidiosis for 6 days. Usually seen in chickens about 1-2 weeks after "c cocci".
COURSE OF DISEASE

- **QUAIL** - 2-3 weeks. Acute may become chronic and have birds dying over long periods of time.

- **CHICKENS** - 2-3 weeks. Not as severe as quail. Acute.
MORTALITY

- QUAIL - Up to 100%
- CHICKENS - Seldom over 10%
METHOD OF SPREAD

- Probably "carrier" birds. If one of these birds is introduced into a clean flock, it can cause disease.
- Bacteria builds up in the soil.
- Carried over in infected ground and equipment.
- Spore continues to infect for years.
CLINICAL SIGNS

QUAIL
1. Usually seen first in 6-10 week old birds.
2. Develop diarrhea (watery).
3. Sudden mortality in heavy birds.
4. Infected birds thirsty - birds huddle around drinkers.
CLINICAL SIGNS (CONT.)

QUAIL

5. Can become chronic and birds emaciate
6. Any age may be affected
7. Usually seen in birds raised on the ground
8. Occasionally occurs on wire
9. Huddled up, ruffled feathers
SIGNS

CHICKENS
1. Often occurs following a coccidiosis "break"
2. Typical "sick bird"
3. Birds develop diarrhea
4. Thirsty birds
5. Mortality
6. Usually in white leghorn pullets 6-14 weeks old. Immunity to coccidiosis is usually developed by this age.
COMMENT

- Has been confused with severe *E. brunetti* - grossly
POSTMORTEM LESIONS

QUAIL
1. First seen in heavy, fat birds
2. Crop full of water, feed may be present indicating sudden death.
3. Yellow necrotic areas in liver
4. May be hemorrhagic infarcts in liver - this may be a good place to isolate the organism.
QUAIL
5. Discrete ulcers in small intestine and ceca (the lower 1/2 of the gut). This is usually the only lesion seen.
6. Birds that live more than 10 days become emaciated.
7. In peracute cases, may see liver lesion without gut lesions.
POSTMORTEM LESIONS

CHICKENS

- Same as quail, but lesions are usually where previous coccidiosis lesions were.
- Intestinal lesions not as distinct as quail. Liver lesions alone frequently.
- Seldom chronic.
- Secondary airsacculitis may result from septicemia occurring after gut damage.
DIAGNOSIS

- Lesions are unique
- May have to differentiate from necrotic enteritis in chickens
- QUAIL - Used as a biological indicator
- LIVER SMEAR - Typical organism
TREATMENT

QUAIL

- FEED: Bacitracin - 100-200 gm/ton for 7-10 days
- WATER: Bacitracin - 1/4 to 1/2 gm/gal 7-10 days

CHICKENS:

- Same as quail.
- Feed or water; pen. or strep.
PREVENTION

**QUAIL:** When birds are raised or held on the ground or exposed to a contaminated environment, use bacitracin at 50-100 gm/ton (1/2 the treatment dose) in the feed continuously.

**CHICKENS:** Bacitracin: 50-100 gm/ton of feed continuously. Also low levels of lincomycin or virginiamycin.
A problem in broilers.
Has become prevalent in all broiler producing areas since about 1966.
The introduction of ionophore coccidiostats has almost eliminated the disease.
ETIOLOGY

- Unknown for sure
- High energy feeds (broilers)
- Probably a combination of events including sub-clinical coccidiosis and *Clostridium perfringens*
- Maybe gut stasis leading to an anaerobic environment.
Cl. welchii
+
Opiates
(gut stasis)
+
Chalk
(bulking agents)

Lesions of
Necrotic Enteritis
COURSE OF DISEASE

- Affected birds die quickly
- If untreated, several weeks
- After treatment, usually abates in 12-48 hours.
- May re-occur up to 3 times in one growout
MORTALITY

- If untreated maybe up to 20%.
- If treated early less than 2%.
METHOD OF SPREAD

- Probably by humans, or may be normal soil organism that has flourished in built-up litter.
- Will re-occur in infected house.
CLINICAL SIGNS

1. Ataxia, "creepers"

2. Water filled crop

3. Birds show intoxication
   Usually seen first time at
   3 weeks of age.
POSTMORTEM LESIONS

1. Extreme dehydration
2. Dark congested liver
3. Entire mid and lower small intestine have "cooked" appearance. Mucosa looks like a "dirty turkish towel". No liver lesions as seen with ulcerative enteritis.
BIOLOGICAL TEST

QUAIL
Used for biological testing of this pathogen.

- Homogenized suspect gut. Results in 72 hours.
  50% suspension per OS

- NECROTIC ENTERITIS + Quail = 0

- ULCERATIVE ENTERITIS + Quail = disease
TREATMENT

TREAT IN DRINKING WATER

- Bacitracin - 1/4 - 1/2 gm/gal - 4-5 days
- Pen. Strep. - high level - 4-5 days
- Streptomycin (strep-sol) works best
- Response to treatment can be used as a diagnostic tool.
TREAT IN FEED

CHICKENS:

- Bacitracin only mildly effective.
- Tylosin works well but is expensive.
PREVENTION

Low continuous levels of antibiotics:

- Bacitracin - 50-100 g/ton in feed for chickens, continuously
- Lincomycin
- Virginiamycin
- Ionophores such as monensin seems to have some effect against N.E.
- Disease much reduced in occurrence since Coban (Monensin) became a commonly used coccidiostat.
- Apply salt to flooring at 60 lbs./1000 sq. ft.
GANGRENOUS DERMATITIS (GD)

- A disease mainly seen in 3 to 8 week old broilers characterized by massive necrosis of the skin with mortality.

- The disease is strongly related to immunosuppressed chickens often associated with an earlier IBD challenge.
ETIOLOGY

- Usually seen in broilers that are progeny of hens with no immunity to Gumboro (IBD). IBD destroys the bursa and then causes immunosuppression.
- Affected chickens usually have Gumboro (IBD) before 2 weeks of age. The earlier the infection of the bursa, the more severe the immunosuppression.
- Wound infection.
- Staph usually present.
- *Clostricium perfringens* type A and *C. septicum*. 
INCUBATION PERIOD

(A) Natural Outbreaks
    3-5 weeks

(B) Experimental
    24-36 hours
COURSE OF DISEASE
10-14 days

MORTALITY
2-10%

METHOD OF SPREAD
Built up litter
CLINICAL SIGNS

1. Usually in 3-8 week old birds
2. Mortality increases
3. Dead birds well-fleshed
4. Large reddish-green necrotic areas on skin
5. Gas on palpation - crepitation
6. Follows certain breeder flocks that had poor immunity to IBD
7. Dead birds decompose rapidly
POSTMORTEM LESIONS

1. Massive hemorrhaging under skin along back, thigh, and hips
2. Large brown liver, frequently with gas
3. Hemorrhages in the mesentery
4. Retained yolk sacs - poor chick quality
5. Anemia, pale bone marrow
DIAGNOSIS

(1) Typical postmortem lesions

(2) Staph and Clostridia isolation on bacteriology
TREATMENT

1. Penicillin
2. Erythromycin
3. Tetracycline
PREVENTION

- Good IBD immunization program in breeder hens to prevent it in the broilers.
- Then vaccinate the broilers at the “right” time.
- Clean out built-up litter if history of GD.
- Reduce injury - overcrowding.
This is a major problem in wild ducks in the western U.S.
It also affects all domestic avian species.
Problem caused by powerful toxin.
Infection not present in affected bird.
Condition referred to as "Limber neck" in chickens.
**CAUSATIVE AGENT**

- Exotoxin produced by bacterium *Clostridium botulinum*.
- Type A and C usually seen in domestic birds.
- Western duck sickness is caused by type C. *Cl. botulinum*, a large gram (+) spore forming rod that grows anaerobically.
- 0.00012 mg of toxin will kill adult guinea pig.
Botulism has occurred in broiler flocks in the last few years that resembles an infectious disease with mortality in consecutive flocks in the same house.
In ducks - most losses due to dehydration. A supply of fresh water decreases mortality.

Buzzards withstand as much as 300,000 guinea pig lethal doses.
INCUBATION PERIOD - 24 to 48 hours after toxin is consumed.

COURSE OF DISEASE - acutely affected bird seldom survives. In mild case, birds may recover in 2-3 days.

MORTALITY - 1 to 100%.
METHOD OF SPREAD IN CHICKENS AND PHEASANTS

- In backyard flocks and floor layers - decomposing dead animals and fly maggots feeding on dead animals.

- 1 g. fly larvae may contain 180,000 mouse LD-50’s
METHOD OF SPREAD
(DUCKS)

- Western duck sickness - shallow alkaline lakes with high content of botulism in mud --> decay of vegetation --> anaerobic conditions --> insect larvae die contaminated with botulism --> ingested by ducks --> ducks die of toxin --> ingested by blowflies --> flies die in water --> ingested by ducks --> perpetuation of outbreak.
CLINICAL SIGNS

1. Dullness, leg weakness, and progressive paralysis.
2. Complete paralysis of neck and wing muscles, head rests on ground. Neck is extended (limberneck).
4. Feathers are loose and easily plucked. Erector pili muscles is affected.
5. Half open eyes in broilers.
COMMENT

- Very sick, septic birds can look the same way.
POSTMORTEM LESIONS

- No diagnostic lesions.
- Maggots may be present in the crop.
DIAGNOSIS

- Suggestive - usually good enough typical symptoms.
- Positive - demonstration of the toxin intraperitoneal inoculation of susceptible mice with serum, or feed material from crop and intestine to susceptible mice. Control mice protected with antiserum should also be inoculated.
TREATMENT

1. Treating visibly affected birds is of little value.

2. Epsom salts in the feed 1 lb. per 75 to 100 birds for 4 to 5 hours. Will purge the digestive tract.

   Molasses in the water 1 pt/5 gal for one day.
TREATMENT (CONT.)

- In broiler house, with history of botulism problem, change the pH of the soil.
- Treat with antibiotics effective against gram (+) bacteria, such as bacitracin, as soon as diarrhea develops.
- Anti-oxidant effect of selenium also helps problem by clearing free radicles.
For valuable birds you may use specific antitoxin 2-4 mls in large ducks.

Cost approximately $2500/500 ml.
PREVENTION

- Feed fresh wholesome ration.
- Make sure all dead birds are removed from pens each day.
- Antibiotics in broilers (bacitracin).
- Vaccinate valuable birds with two injections of type A and C toxoid 3-4 weeks apart.
PREVENTION IN BROILER HOUSE

- Clean out affected house.
- Put in new litter and "top dress" with litter from "good house".
- Acidifying soil and litter in affected houses. (Sulphur)
- Apply salt (60 lbs. NaCl/1000 sq.ft.) to floors. This will go right into the litter. But the salt can damage metal equipment.
AVIAN VIBRIONIC HEPATITIS

- Probably not a clinical disease of chickens.
- Described as a chronic, contagious disease of laying hens. Occasionally seen in pullets.
- The organism is found in the gall bladder and a common gut inhabitant. It is an opportunist and secondary infection.
- This condition usually occurs concurrent with other problems such as internal parasites or "chronic" coccidiosis.
- Public health problem in dressed chickens.
- Commonly found in GI tract of poultry with no apparent disease.
Campylobacter jejuni is microaerophilic, gram-negative, motile, comma or s-shaped rod. Usually identified by staining with methylene blue and observing for characteristic shape. May be cultured from gall bladders of normal chickens.
METHOD OF SPREAD

- Under natural conditions, fecal contamination.
POSTMORTEM LESIONS

- The gall bladder is usually about 2x and the bile is thick and tenacious.
- But, the gall bladder gets large when bird has been off feed.
Acute cases have been described with hemorrhages and necrosis of the liver.
DIAGNOSIS

- **POSITIVE** - isolation and identification of causative agent.

- Difficult to culture special agars available.
CULTURE METHODS

Campylobacter jejuni

Gall bladder (scrape mucosa)

Bl. agar

Incubate - 48 hr. - 10% co₂ tension

Colonies are tan

Oxidase (+) blue

Gram (-); "s" shaped rods (chains)
AVIAN TUBERCULOSIS

- This is a disease of older birds (5-6 years) in old contaminated chicken yards.
- Due to modern methods of poultry management this disease is not considered important at present.
- Spreading infected poultry litter onto cattle pastures will cause a positive tuberculin test in cattle.
CAUSATIVE AGENT

- *Mycobacterium avium*
- Can be tested for with tuberculin
- Injected I/D in comb.
- This is rarely done today.
- Major lesions in liver, spleen, intestines, ceca, and bone.
- Humans - resistant to infection.
- Swine - can be readily infected.
CLASSIFICATION OF AVIAN MYCOPLASMAS

** M. galllisepticum
  M. gallinarum
  M. iners
  M. anatis
*M. synoviae
*M. meleagridis
  M. pullorum
  M. gallinaceum
  M. gallapavonis
*M. iowae
  M. columbinasale (pigeon)
Others
Mycoplasma gallisepticum

- A respiratory disease primarily of chickens and turkeys, characterized by respiratory signs and lesions and a prolonged course in the flock.
- MG is one of the more costly diseases of commercial poultry.
- In chickens the disease was originally known as PPLO - pleuropneumonia-like organism.
- It is also called CRD or chronic respiratory disease in chickens and infectious sinusitis in turkeys where it is manifested predominantly by swelling of the infraorbital sinuses.
ETIOLOGY

- The problem is caused by *Mycoplasma gallisepticum*. The organism is coccoid and 0.25 - 0.5 microns in diameter. Mycoplasmas have no cell wall.

- MG alone only causes mild lesions in chickens. However, this organism is often associated with one or more of the following agents and pathogenicity is enhanced by association with infectious bronchitis, Newcastle disease, *E. coli*, dust, or ammonia.
INCUBATION PERIOD

- Four to 21 days for complicated disease. Longer (10-21 days) for uncomplicated disease.
COURSE OF THE DISEASE

- Weeks to months. Becomes chronic.
MORTALITY

- Negligible in adult birds.
- Broilers up to 30% in complicated cases.
- Often the mortality rate may be low but the cull rate is high due to severe respiratory disease.
METHOD OF SPREAD

- MG is transmitted in some of the eggs (transovarian transmission) laid by inapparent carriers.
- The shed rate of the bacterium is highest (about 30%) during the acute phase of infection but decreases (to about 5%) during the chronic phase of the disease.
- Chickens will remain infected after clinical signs have disappeared. Infected progeny then transmit the agent laterally, probably through infectious aerosol coughed into the air.
METHOD OF SPREAD

- MG seldom survives for more than a few days outside of the host.
- Although MG is not highly infectious like respiratory viruses, outbreaks in MG clean flocks have been traced back to humans as a mechanical vector.
Infected eggs from infected breeders have been dipped in antibiotic solutions or super heated to destroy the bacteria prior to setting the eggs. This decreases hatchability as well as transmission.

Infected eggs from infected breeders should be set separately from eggs from uninfected flocks to prevent lateral transmission of the organism in the hatchery.
CLINICAL SIGNS

1. Nasal discharge - usually 1st sign.
2. Conjunctivitis.
3. Respiratory rales.
4. Often starts at 10 days to 2 weeks after viral vaccination.
7. Lower egg production in hens.
8. Swollen sinuses in turkeys.
1. Problems in broilers can often be traced back to a single breeder flock.

2. Pullets are often raised MG free and become infected after being placed in laying houses on multiple-age farms.
1. Mucoid or catarrhal exudate in nasal passages, trachea, bronchi, and thoracic air sacs.

2. Early cloudiness of the air sacs is caused by infiltration of mononuclear cells and hyperplasia of epithelial cells. Early in the infection air sac exudate has the appearance of "soap suds".
3. Later this material organizes and becomes caseous and yellow. Pericarditis and perihepatitis are often a sequelae of the disease when it becomes complicated with E. coli. High condemnations at processing will result.

4. In infectious sinusitis of turkeys, lesions may be restricted to swelling of the infraorbital sinuses. Conversely, sinusitis may be absent although rhinitis, tracheitis, and airsacculitis occur and there may be fibrinous pneumonia.
# Differential Diagnosis

<table>
<thead>
<tr>
<th>Turkeys</th>
<th>Chickens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newcastle disease</td>
<td>Newcastle disease</td>
</tr>
<tr>
<td>Avian influenza</td>
<td>Infectious bronchitis</td>
</tr>
<tr>
<td><em>E. coli</em></td>
<td><em>E. coli</em></td>
</tr>
<tr>
<td>Ornithosis</td>
<td>Infectious coryza</td>
</tr>
<tr>
<td>Fowl cholera</td>
<td></td>
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<tr>
<td>Bordetella</td>
<td></td>
</tr>
</tbody>
</table>
DIAGNOSIS

1. Suggestive - history of chronic respiratory disease accompanied by lowered feed consumption, poor gains, or lowered egg production with typical gross lesions. Positive plate agglutination reaction of suspect birds.

2. Confirmation - Hemagglutination inhibition (HI) test may be delayed more than two weeks after a positive plate reaction.
Isolation and identification of the agent is difficult and time consuming but is important if depopulation of an expensive flock is being considered.

A commercial DNA probe specific for MG is available.
COMMENT

- False positive plate agglutination reactions to both MG and MS occur in chickens vaccinated with oil-emulsion vaccines or if the serum has been frozen.

- The vaccine reaction is transitory and will resolve in 4-6 weeks. In the case of false positive after the use of killed vaccines, retest the flock.

- The HI test will eliminate the false positive reactions.
1. Marketing broilers with a low incidence of disease may be more economical than treatment because treatment can be very expensive.

2. Improving management is important in infected flocks. This involves improving air quality to reduce levels of dust and ammonia which can exacerbate the respiratory disease. Also, withholding respiratory vaccinations should be considered to avoid complicating the MG.
3. Many broad spectrum antibiotics have been used and will suppress losses. However, relapses occur when treatment is discontinued. Most antibiotics are given in the feed or water, preferably in the water. The drugs of choice are tylosin or fluoroquinolones which are expensive. Tylosin is usually used in the drinking water at a rate of 2-3 g/gal. for 3-5 days. If given early the treatment works well.
When breeders are shedding, progeny are often inoculated at 1 day of age with 5 mg of spectinomycin alone or in combination with lincomycin or naxel.

Tetracyclines have been used at 200-400 g/ton of feed. In order to best utilize this drug the calcium should be removed from the feed. This can be done for 5-7 days in broilers but not for more than 3 days in layers. It is less expensive than tylosin or the fluoroquinolones.
PREVENTION

1. Depopulation of infected premises and thorough cleaning and disinfection should precede the introduction of a "clean" flock.

2. Test foundation breeders and eliminate reactors.
3. Quarantine measures must be strictly enforced and good management and sanitation must be practiced to keep a flock free of infection.

4. Bacterins and F strain live MG are being used on multiple-age commercial layer farms with great success. These are being used in pullets where it eventually replaces pathogenic MG. This can increase egg production by 7 eggs/hen. F strain is pathogenic in turkeys.
COMMENT

Program for Eradication of MG (used 20 years ago)

1. Test foundation breeder stock and remove MG reactors.
2. Dipping eggs in antibiotics, erythromycin or tylosin. This is done by placing hot eggs in cold solution.
3. Inject breeder progeny with tylosin in oil 4 times at 5 day intervals.

▷ If no reactors after 2 generations, considered MG clean.
Mycoplasma synoviae (MS)

- A mild respiratory disease which also results in inflammation of the synovial sheaths.
- This disease is known as infectious synovitis and occurs in both chickens and turkeys. It is primarily seen in young birds from 4 to 12 weeks of age.
- It is less virulent but spreads faster than MG. It is common in commercial laying flocks.
**ETIOLOGY**

*Mycoplasma synoviae*

- This is a fastidious organism which requires nicotinamide adenine dinucleotide for growth.
- There appears to be only one serotype although isolates vary in pathogenicity.
INCUBATION PERIOD

8 to 10 days.

COURSE OF THE DISEASE

Several weeks. Becomes chronic.
MORTALITY

- 1-10%. Morbidity can reach 75%.
- The severity depends on the virulence of the infecting strain.
METHOD OF SPREAD

- Vertical (egg) transmission and lateral (direct contact) transmission.
1. Lameness in pale combed birds showing retarded growth. Hock joints and foot pads become swollen and breast blisters appear. Affected birds are reluctant to move. When made to move they will run and sit down as soon as possible.

2. The feces of affected birds are often green and wet. Eventually many birds become dehydrated and thin.
3. The respiratory form of this condition is very similar to the signs seen with MG but not as severe. They usually occur as a continuation of a viral vaccine reaction or are started by some other stress condition.

4. There may be no clinical signs with an MS outbreak, only positive plate agglutination reactions with positive HI reactions.
POSTMORTEM LESIONS

1. In the early stages a viscous, creamy to gray exudate is seen in the hock and/or stifle joint, over the keel bone, and in the foot pads. As the disease progresses in individual birds, this exudate becomes caseous and may be yellow or orange with time.

2. The viscous creamy exudate is frequently seen in the wing joints and is one cause of condemnations in processing.
3. The liver, spleen, and kidneys are frequently enlarged. The enlarged livers are usually mottled and greenish to dark red in color.

4. Erosions on the articular surface may occur.

5. Respiratory lesions may be absent or consist of a mild mucoid tracheitis, airsacculitis, or sinusitis.
DIFFERENTIAL DIAGNOSIS

- Staphylococci
- Fowl typhoid
- Viral arthritis
DIAGNOSIS

1. Suggestive - Typical signs and gross lesions with a positive plate agglutination test on sera from a few birds in the flock.

2. Confirmation - Positive HI test. This reaction may be delayed a few weeks after the positive plate reaction.
   - Isolation and identification of the organism can be done in 5 days in a well equipped laboratory. The isolated Mycoplasma can be identified by direct immunofluorescent antibody techniques.
Serology is less reliable than with MG.
There are more problems with cross reactions.
TREATMENT

- Treatment of lame birds with well established synovitis is usually not very satisfactory.
- Potentiated tetracyclines at 200-400 g/ton with the calcium removed from the diet for synovitis.
- Tylosin in the water at 2-3 g/gal. at the time of vaccine reaction.
- Day-old injection of infected progeny with spectinomycin or gentamicin. Infected eggs should be hatched in a separate incubator.
PREVENTION

1. In breeders, test and segregate as with MG.

2. Start with chicks or pouls that were hatched from MS clean flocks and raise the birds in quarantine, under all-in all-out conditions.
3. Synovitis can usually be prevented by continuous administration of 75-100 g/ton chlortetracycline. This is expensive and rarely done.

4. An oil-emulsion bacterin is commercially available and is of some benefit where the risk of infection is high. The product is expensive and requires handling of each bird because the vaccine must be injected.
**Mycoplasma meleagridis (MM)**

- A disease of turkeys characterized by an inapparent venereal infection in breeder turkeys and airsacculitis in embryos or recently hatched poults.
- Most large turkey breeder flocks are free of MM infection due to testing and eradication efforts.
ETIOLOGY

Mycoplasma meleagridis
COURSE OF THE DISEASE

- Air sac lesions usually resolve by 12 weeks.
- Some birds may develop bone deformities in which case crooked necks may persist.
MORTALITY

- Very low. Culling is the more serious problem.
TRANSMISSION

- The disease is spread venereally from tom to hen.
- Egg transmission leads to infection of the embryo.
- Lateral spread within a flock via the respiratory route.
Most of the signs are mild or inapparent on casual examination and go unobserved.

1. Often there is impaired hatchability of eggs from infected flocks. Embryo mortality is highest after eggs are transferred to the hatcher.

2. Reduced growth and feed efficiency.
3. Stunted birds with skeletal deformities especially of the neck and legs. This lesion is rare.
4. Respiratory infection is present in young poults but usually regresses by 7-9 weeks of age unless predisposed by other primary pathogen or secondary *E. coli*.
POSTMORTEM LESIONS

1. Air sac lesions in 1-day-old poults. The incidence will peak at 4-6 weeks of age. It will be inapparent in older birds.

2. Poults with wryneck may have osteomyelitis of adjacent vertebra.
DIAGNOSIS

1. Suggestive - Air sac lesions in 1-day-old poults. Positive plate agglutination test.

2. Confirmation - Positive HI test or isolation of the organism.
TREATMENT

- No practical treatment in affected flocks. Use of lincomycin/ spectinomycin in the drinking water at 2 g/gal. for the first 5-10 days of life may reduce the airsacculitis and improve weight gains.

- However, the clinical signs are transient and antibiotics will not clear the infection.

- Antibiotic use will also have no effect on reducing egg transmission.
PREVENTION

1. Egg dipping in 3000 ppm of tylosin reduces but does not eliminate infection. Eggs are heated and placed in a cold water bath or antibiotic solution under a vacuum. Eggs can also be injected with 0.6 mg gentamicin sulfate and 2.4 mg of tylosin in a 0.2 ml volume.
2. Begin with MM free pouls obtained from clean breeder flocks.
3. Serologic testing and culturing has not been satisfactory.
<table>
<thead>
<tr>
<th>Name(s) of the Disease</th>
<th>Etiologic Agent</th>
<th>Type Bird Affected</th>
<th>Nature of the Disease</th>
<th>Major Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic respiratory disease</td>
<td><em>Mycoplasma gallisepticum</em></td>
<td>Chicken</td>
<td>A respiratory disease.</td>
<td>Airsacculitis, adhesive pericarditis, fibrinous perihepatitis. Occasionally causes synovitis or salpingitis.</td>
</tr>
<tr>
<td>Infectious sinusitis</td>
<td><em>Mycoplasma gallisepticum</em></td>
<td>Turkey</td>
<td>Unilateral or bilateral sinusitis. May spread to or occur initially in the lower respiratory system.</td>
<td>Swollen infraorbital sinus(es) may or may not be followed by airsacculitis.</td>
</tr>
<tr>
<td>Infectious synovitis</td>
<td><em>Mycoplasma synoviae</em></td>
<td>Chicken &amp; turkey</td>
<td>Involves synovial lining of joints, tendon sheaths. Results in lameness, debility.</td>
<td>Swollen joints and tendon sheaths. Feet, shanks, hocks more obviously affected. Occasionally causes airsacculitis in broilers and turkeys.</td>
</tr>
<tr>
<td><em>Mycoplasma meleagridis</em> infection; MM infection</td>
<td><em>Mycoplasma meleagridis</em></td>
<td>Turkey</td>
<td>A venereal infection of turkeys, usually transmitted by infected pooled semen. Produces airsacculitis in many progeny.</td>
<td>Airsacculitis in nonhatching or newly hatched poults. May spread laterally to other young poults as an airsacculitis. May lead to airsacculitis in market birds.</td>
</tr>
</tbody>
</table>

^ More than 20 serotypes of *Mycoplasma* have been identified in chickens, turkeys, and ducks. These three are the most significant pathogens.
ASPERGILLOSIS
(BROODER PNEUMONIA)

- Affects most avian species
- Important in poult's, chicks, and quail.
- Is a serious problem in penguins in zoos.
ETIOLOGY

Aspergillus fumigatus

- Spores from contaminated incubators, feed, litter can penetrate broken shells.
- Large dose to infect
- Probably not bird to bird.
INCUBATION PERIOD

8 to 12 days \textit{in vivo}
48 hrs \textit{in vitro}

COURSE OF DISEASE

2 to 3 weeks
may linger
MORTALITY

- Depends on age
- Up to 100% under brooder
- Other diseases aggravate
METHOD OF SPREAD

- Inhalation of spores -
  1. Contaminated eggs in incubator
  2. Dusty litter and ranges.  
     Spreads readily under brooder

If clinical signs occur after 7 days of age, consider farm rather than hatchery exposure.
COMMENT

- Usually a young bird disease:

- Exposure in the hatchery or because they are so close to new litter material, they inhale spores.
CLINICAL SIGNS

1. Rapid gasping respiration (without rales)

2. "Starve outs", emaciated

3. Blepharoconjunctivitis yellow caseous pellet in eye

4. Encephalitis common in turkey poults but rare in chickens
POSTMORTEM FINDINGS

1. Round yellow caseous granulomas on air sac, throughout lungs.
   “Pearl disease”
2. Occlusion of trachea
3. Occasionally green mold growth on airsacs or in lungs
DIAGNOSIS

- Typical lesions
- Culture on Sabouraud's dextrose agar w/added chloramphenicol.
- Identify and classify fungal growth (Light microscope).
DIAGNOSIS (CON’T.)

- Histopathology to demonstrate fungal hyphae in tissue.
- Hyphae brownish and have outgrowths in "y" branches.
TREATMENT

- None specific
- Stress prevention
PREVENTION

- Remove source of infection
  - Use high quality litter with little bark
  - Collect eggs often to prevent cracks
  - Keep clean litter in nests
  - Do not set cracked eggs
- Fumigate incubators and hatchers
- Add antifungal agents to feed
- Treat litter w/ fungistatic/cidal agents (TBZ (Folatech), propronic acid, Clinafarm in the hatchery)
- General clean up - disinfection between grow out.
CROP MYCOSIS (THRUSH)

- Most common mycotic diseases of poultry
- Most important in young poults and occasionally cage layers
ETIOLOGY

*Candida albicans*

- Very ubiquitous in normal flora
- Follows debilitating condition
- Broad spectrum antibiotics favor development by disruption of normal flora balance.
- Seen in “spiking mortality” poult due to antibiotic use.
INCUBATION PERIOD

- Experimentally about 30 days, but has been found in 7-14 day old poult's
- Induced by giving sugar water to poult's.
COURSE OF DISEASE

- 3 to 4 weeks
- May become chronic, then several weeks
MORTALITY

- Poults and chicks - up to 20 to 30%
- Older birds - after 4 weeks usually low
METHOD OF SPREAD

1. Drinking water
   a) associated with unsanitary, over-crowded condition
   b) slimy water founts, wet litter

2. Infected birds source of contamination

3. Cages due to decreased bacterial competition
   No contact with feces.
SYMPTOMS

- Poor growth
- "Sick chick attitude"
- In layers - decrease egg production 15 to 20%
- Birds vomit in feed trough
POSTMORTEM LESIONS

- Non-inflammatory - thickened crop and/or mouth
- White, circular, raised ulcer-like formation
- Pseudomembrane
- Proventriculitis
  - Gizzard erosion
- Can occasionally be cultured from the liver.
- May cause splotchy hemorrhages in the digestive tract.
DIAGNOSIS

- Suggestive - typical lesions, non-inflammatory

- Positive - isolation and identification
  - Special medias used:
    - Corn meal agar
    - Biggy agar - brown colony with white halo
TREATMENT

Mycostatin (nystatin)
100 gm/ton 7-10 days

CuSO$_4$
1:2000 in water, 1-3 #/ton

Chlorine in the drinking water at 5 ppm.

Clean up
PREVENTION

- Maintain sanitation
- Avoid prolonged use of broad spectrum antibiotics
- Mycostatin (nystatin) in feed 50 gm/ton
- Ca++ or Na+ propionate in feed 3-5 #/ton
- Propionic acid antifungals in feed 1-4 #/ton. (Depends on concentration of active ingredient).
DACTYLARIOSIS

- Encephalitis of turkeys and chickens
- Caused by the thermophilic (likes heat) fungus
CAUSATIVE AGENT

- *Dactylaria gallopava*
- Fungus contaminated hardwood litter and sawdust.
- The litter often has been stored in such a way as to generate heat which enhances fungal growth. Thermophile.
INCUBATION PERIOD

- Six to ten days experimentally.
- In field outbreaks -- from 1 week to 6 weeks of age generally.
COURSE OF DISEASE

- Acute to fatal.
- Can affect humans also but not by infection from bird. Humans get exposed same way as birds - by inhalation of spores from environment.
- No immunity.
MORTALITY

- Due to encephalitis:
  - Poult's -- up to 30% in Georgia
  - Broilers -- 3-10%
METHOD OF SPREAD

- Spores in contaminated litter inhaled.
- Not commuted from bird to bird.
CLINICAL SIGNS

- CNS signs, incoordination followed by torticollis, leg paralysis.
- Down on sides.
- Weak, ataxia, head tremors
POSTMORTEM LESIONS

TURKEYS AND CHICKENS

- Lesions, granulomatous in character
- Cerebral, cerebellar or optic lobe
- Brains mushy with grayish walled-off lesions.
- Opacity in eye.
- No airsac problems.
DIFFERENTIAL DIAGNOSIS

- Avian encephalomyelitis
- Aspergillosis
- Newcastle disease
- Vitamin E deficiency
1. Demonstration of hyphal elements in the brain.
2. Culture mold from lungs and brains.
3. Micromorphology of mold.
HISTOPATHOLOGY

- Granulomatous areas of brain and lung have massive inflammatory cellular infiltration.

- Centers - coagulative necrosis and giant cell formation.

- Small, delicate hyphal elements demonstrated.
CULTURE TECHNIQUES

- SDA with added chloramphenicol - 42° C Thermophile. Warmer than normal incubation temperatures of 36-37°.

- Dark brown -- dematiaceous fungus with small oval two celled brown spores. Conidia are cuneiform in shape.

- Attached to hyphae by short simple conidiophores. Spores attached to hyphae or simple conidiophores by delicate stalks. Underside of fungal growth turns agar port wine color.
NOTE

- *Dactylaria* sp. is also a human pathogen which is not treatable.
- Use extreme caution when handling diagnostic cultures in the laboratory.
TREATMENT

- None, only supportive therapy
PREVENTION

Treatment of litter with antifungal compounds:

- Propionates, TBZ, clean out and disinfection.
- Do not use hard wood litter or litter that is damaged by composting.
MYCOTOXICOSIS

- Disease related to one or more mycotoxins
- **Mycotoxin** - "mycos" - mold, toxic metabolite
- Produced by filamentous fungi while growing on substrate such as feedstuffs and litter.
- **Pathology** - results from ingestion of mycotoxin, not active infection in affected animal. Over 300 mycotoxins known.
- Many antibiotics are mycotoxins.
DISCOVERY AND ISOLATION

- In poultry, 1969 in England, X disease in turkey poults, killed 100,000.
- Turned out to be aflatoxin in peanut meal.
- Previous concern - food deterioration.
- Now feel the effect on production efficiency may be most costly.
MAIN MYCOTOXINS PRESENTLY CONSIDERED PATHOGENIC

1. Aflatoxin - most common.
2. Fusarium toxins (trichotheccenes)
   - T-2 toxin DAS, HT2, deoxynivalenol
   - (Don) "Vomitoxin", Zearalenone (F-2)
   - "Estrogenic toxins", Fumonisins (B₁).
3. Ochratoxin
4. Oosporein
5. Cyclopiazonic acid (CPA).
SEVERITY OF MYCOTOXICOSIS MAY VARY WIDELY INFLUENCED BY THE FOLLOWING PARAMETERS

- Level in substrate (feed or litter)
- Age
- Sex
- Genetic complement
- Environment, temperature, etc.
- State or plane of nutrition
- State of production (laying birds)
- Presence of other disease
AFLATOXICOSIS

- Received most attention in last few years.

- Mainly due to feed (corn, milo, canola) contamination in poultry.

- Responsible for poor feed conversion and loss of egg production.
AFLATOXICOSIS

- Acute (less common) and chronic toxicity
- In chickens, duck and turkeys.
- Same etiology as "moldy" corn in swine.
ETIOLOGY

AFLATOXINS

- Type B₁ (most important in poultry)
- B₂, G₁, G₂, M₁*, M₂*, and others
  - *Not an avian problem

Known toxin producers:

- *Aspergillus flavus, A. parasiticus, and some species of Penicillia.*
- Substrate - corn, barley, millet, peanuts, peas, rice, sorghum, wheat, coastal hay(s), oats.
CHARACTERISTICS OF AFLATOXIN

- Ubiquitous in nature.
- Carcinogenic, teratogenic.
- Aflatoxin is the most potent mycotoxin, and is responsible for severe economic loss.
DOSE EFFECT

- 400 PPB - weight depression in 10 days to 2 weeks
- 500 PPB - kill poults in 2 weeks
- 1 PPM = 1000 PPB
CLINICAL SIGNS

1. Depressed growth and feed conversion.
   Pale and off feed with increase in mortality - common.

2. Impaired immune system with increased susceptibility to other conditions.

3. Reduced fertility and hatchability.
   Egg production loss depending on dose.
POSTMORTEM LESIONS

1. Pale yellow fatty livers & distended gall bladder

2. Ecchymotic hemorrhage in muscles & petechial hemorrhages in viscera

3. Regressed bursa of Fabricius

4. Congested kidneys (usually enlarged)
5. Enlarged spleen & pancreas
6. Hydropericardium and ascites
7. Pale bone marrow
8. Bile is dilute and lime to fluorescent green in color
IMPLICATIONS

- Some mycotoxins are carcinogenic.
- Documented synergism with coccidiosis, Salmonella, crop mycosis, nutritional deficiency, and infectious bursal disease.
- Impairment of immune ability of affected animal may occur.
- Specifically impairs protein synthesis (building block protein as well as globulins).
- Increased capillary fragility causing bruising and condemnations.
The target organ is the liver and the most sensitive metabolic systems are protein synthesis, lipid synthesis, and lipid transport.
Extraction & quantification of toxins from feed with chromatography, monoclonal antibody technology.

Gross pathology - swollen fatty livers -non-specific.

Histopathology
HISTOPATHOLOGY

- Fatty changes of the liver with focal necrosis.
- Progressive bile duct proliferation with portal cirrhosis.
- Acinar degeneration of the pancreas.
Detection of metabolites in tissue.
  - This is rarely used.
  - The half-life is extremely short, i.e. 1-2 days.
BIOLOGICAL TEST

- Feed suspect feed to 1 day old ducklings
- Age susceptibility - young more susceptible

  Susceptibility to toxin

  Ducks > Turkey > Chicken
Allowed level for feedstuffs in interstate commerce is no greater than 20 PPB (Delaney Clause, FDA).

This number was arbitrarily chosen.

This was the level they were able to detect with technology present at that time.
PREVENTION

1. Establish an ingredient assay program. Kojic acid causes this mold to glow under a black light.
   But the presence of mold does not mean aflatoxin is present.

2. Reject contaminated feed or ingredients. Aflatoxin is a warm weather problem.

3. Reduce feed storage time. (Keep under 9 days)
   This is dependent on price and storage capacity.
PREVENTION (CONT.)

4. Keep feed bins and handling equipment clean. Use chlorox, rotate bins.

5. Use low moisture ingredients and don't allow it to become damp.
TREATMENT

- Discontinue use of contaminated feed.
- Increase protein, fat, vitamin, and electrolyte level of diet. This is to oversupply the systems affected by aflatoxin.
- Fat soluble vitamins have a sparing effect.
TRICHOTHECENE MYCOTOXICOSIS

50 related compounds from genus *Fusarium*.

E.G.: Type A - T-2, HT-2, DAS
     (Diacetoxyscirpenol)
     Type B - nivalenol, deoxynivalenol
     (Don)

*Fusaria* are cold weather molds. Contamination of overwintered wheat and stored corn, and soybeans is common.
EFFECTS

- Immunosuppression - decreased spleen size, decreased cell function, and thymus atrophy.
- Depressed - WBC and RBC numbers.
- Hemorrhages in musculature and viscera.
- Oral, proventricular, ventricular lesions/ulcers.
- Decreased feed intake.
- Depressed weight gains and conversions.
- Depressed egg production.
- Poor egg shell quality.
- More egg breakage.
- Depressed hatch.
- Enteritis with necrosis.
EFFECTS (CONT.)

- Lymphoid depletion in "galt", "balt".
- Bone lesions like "TD", rachitic lesions.
- Neural abnormalities.
- Footpad lesions.
- Stunting.
- Depressed hemoglobin.
- Lowered grain quality due to mold contamination.
- Feed refusal factors.
- Depressed fertility.
- Anti-thiamine activity (*F. Moniliforme*).
DIAGNOSIS

- Detection of toxin
  - Thin layer chromatography
  - HPLC, gas chromatography
TREATMENT

- Remove offending feed.

- Supportive - Increase vitamins
  - Increase protein
  - Increase copper
OCHRATOXICOsis

- A disease caused by a very potent Mycotoxin produced by Aspergillus spp. and Penicillium spp.
- Nephrotoxic in all classes of poultry.
- Most nephrotoxic mycotoxin.
ETIOLOGY

- Ochratoxin A & B
- Produced by *Aspergillus ochraceus*;
  
  *Aspergillus melleus* and *Penicillium viridicatum*.
  
  Poor grades of corn important
  
  This is a warm weather mold.
MORBIDITY

- Variable; depends on the age of the bird and level of ochratoxin consumed.
MORTALITY

- Variable; depends on amount of ochratoxin consumed, and the age of the bird.
1. Diarrhea, urate content very high (Polyuria)
2. Listlessness
3. Decreased feed consumption
4. Rickets
5. Decreased egg production
6. Decreased weights
7. Increase in egg stains in caged birds
POSTMORTEM LESIONS

1. Kidneys enlarged and pale - renal disease is permanent
2. Reduction in bursal size due to depletion of lymphoid elements
3. Suppression of hematopoietic activity in bone marrow
4. Decreased bone strength in the legs
5. Cachexia in advanced cases
DIAGNOSIS

1. Enlarged kidneys suggestive, but IBV can also cause kidney lesions.

2. Feed assays conclusive.

3. Histopathologic changes on H&E.
   Histo: coagulation necrosis of proximal tubules of kidney.
TREATMENT

1. Remove suspect feed

2. Treat secondary problems
PREVENTION

1. Purchase good quality corn

2. Establish a quality control program
OOSPOREIN

- Toxic metabolite of *Chaetomium trilaterale* and other mold species found in animal feeds and cereal grains.
- Will kill chickens at 200-400 ppm of feed in 4 days.
- Uric acid secretion is impaired.
- Birds become lethargic and show lesions of visceral and articular gout.
- Broiler chickens are 4 times more susceptible than poulets.
- Chickens may have pseudomembranes on the mucosa of the proventriculus lining and Gizzard erosions.
- Fairly uncommon.
The toxin cyclopiazonic acid is produced by several Aspergillus and Penicillium species when they grow on corn, mixed poultry feeds, peanuts, and other substrates.

100 ppm of CPA reduced growth by one-half and produced high mortality (60%) when fed from 1 day old to 7 wks. of age in chickens.

Levels above 50 ppm cause mucosal erosion, hyperemia and flaccid dilatation of the proventriculus on gross examination, ulcerative proventriculitis, mucosal necrosis of the gizzard, hepatic and splenic necrosis, and inflammation on histopathological examination.

Fairly uncommon.

Have increased ingesta in carcasses at processing.
TOXICITIES
Before diagnosing a coccidiostat toxicity, ask which coccidiostat they are on.

It is difficult to have a toxicity to something that is not there!
Coban

- Monensin, an ionophore, has been on the market since the early 1970's.
- It is generally not a problem if the birds are started on the compound at day 1; but if it is the second compound in a shuttle program, toxicity can occur even at the recommended levels of 90-110 ppm.
Coban

- Toxicity is characterized by paralysis with legs extended behind the bird in sternal recumbency.
- Birds are alert but cannot move. This is reversible in chickens but many may die.
- Turkeys are especially sensitive and can result in 75% mortality.
Coban

- The use of this compound tends to reduce feed consumption and weight gain.
- This is addressed by increasing the salt in the diet.
- Normal salt levels are 0.3% and are increased to 0.4-0.5% to improve weight gains.
- However, this can result in tibial dyschondroplasia.
Avatec

- Lasalocid: 75-125 ppm.
- This is also an ionophore but is the biological opposite of coban.
- It is used in withdrawal feed.
Avatec

- When using avatec the salt content of the diet must be reduced to levels of 0.25-0.27% or will result in wet litter.

- NaCl deficiency can result with poor weight gains and flock nonuniformity.
Avatec

• The most marked sign of toxicity is birds walking on their toes. This resolves when the feed is removed.

• This is used primarily in the summer because it enhances water consumption.
Nicarbazine

- This chemical is used primarily in the winter and is a very effective product at 125 ppm.

- The compound affects the broilers ability to regulate body temperature and can cause heat prostration if used during warm weather.
Nicarbazaine

- The compound is electrostatic, so adheres to the side of the feed mill.

- If even a small amount (20-30 ppm) gets into breeder feed, it will cause a loss of pigmentation of the egg shells - brown eggs turn white, fertility will decrease by one-half and cause a reduction in hatchability.
Nicarbazine

• It will take 4-6 weeks for the fertility to return.

• Because of this many companies will mix hen feed on one day and dedicate a truck to delivery.
Zoalene

- This is only used in pullets (at 40-125 ppm) because it is a weak coccidiostat which allows immunity to develop.
Zoalene

- When toxicity occurs the birds behave as if they hear a far-off noise by cocking their heads sideways.

- The birds may circle and fall forward.

- Eating and drinking will be diminished.
Zoalene

- The birds are very excitable and tend to pile up.
- The birds will recover within hours of removal of the feed.
MINERALS
Calcium

- Increased calcium levels in the diet will reduce growth. Excessive levels can result in tetany and convulsions.

- High calcium levels can cause a relative phosphorous deficiency.

- Normal levels in broiler rations is 0.8-0.9%.
Calcium

- High levels will affect the kidneys causing a severe nephrosis.

- Severe dehydration will result causing high mortality.

- This results in uric acid accumulation which fills the ureters and deposits on the surface of the viscera.
Dicalcium phosphate is commonly added to the feed. It is grey in color and so birds will pick it out of the feed.

Limestone is another source of phosphorous.
Phosphorous

- Calcium and phosphorus must be in the ration in a balanced ratio.
- An excess of phosphorus can result in a relative calcium deficiency.
Phosphorus

- It is similar to calcium toxicity since both are eliminated through the kidneys in a balanced manner.

- Rickets can result and birds become stunted.
Sodium Chloride

- Salt toxicity is the most common toxicity seen due to feed mill errors.
- Normal salt levels are 0.3-0.4% of the ration.
- If the level reaches 1.5-2% clinical signs will occur.
Sodium Chloride

The clinical presentation is stunted, wet birds with profuse diarrhea. They tend to huddle together.
Sodium Chloride

- On postmortem examination, vesiculated or cystic testes is a diagnostic lesion.

- Eventually ascites may develop.
FEED ADDITIVES
Copper Sulfate

- This is an astringent used to treat mycotic problems. It is commonly used in broiler feed and occasionally in pullet feed.
Copper Sulfate

- It comes in two forms; powder and flake/granular.
- If the flake (granular) is used, gastrointestinal burns will result especially in the proventriculus and gizzard producing a green color in the gizzard and dark tarry feces.
Copper Sulfate

- The level will be toxic if it is over 2 lbs./ton of feed.

- It is commonly used at 1-1.5 lbs/ton. The water soluble form is not as caustic.
3-Nitro

- This is a universal broiler feed ingredient used at 25-33 g/ton for growth and pigment enhancement and has mild coccidiostat activity.

- It is commonly used with Coban.
3-Nitro

- Toxicity to this product is common.
- Most feeds are mixed for 3 minutes which is not enough to prevent "hot spots".
3-Nitro

- Generally, the chickens will present with cervical paralysis, usually on the first cool morning of the season.
- If the birds are left alone and able to drink water, generally, the problem will resolve itself.
The mortality is usually low unless the weather becomes hot and the birds can't get to the water to drink.

Another, milder presentation is the birds will "duck walk" early in the morning.
FEED CONTAMINANTS
Ammonium Nitrate

- This gets into the feed from contaminated freight cars.
- The birds will drink excessively and have severe diarrhea.
- The combs may appear cyanotic.
This is a seed corn treatment to prevent mold growth. It turns the kernels pink.

Toxicity occurs when seed corn is diverted into feed.
Arasan

- At levels over 10 ppm it prevents calcification of the bone resulting in slowed skeletal development and it causes breeders to lay eggs without shells.

- Chicks that hatch from eggs laid by hens ingesting arasan have small skulls and so exophthalmos occurs.
PCB's

- These compounds are used in electrical transformers.
- It has gotten into fish meal through transformer explosions.
PCB's

- It causes decreased growth rates, ascites, cirrhosis of the liver, and increased mortality.

- It can be passed in the eggs of commercial egg layers. The compound is stored in the fat.
Histamine

• This is a biogenic amine caused by the breakdown of fish meal and other animal byproducts used in feed.

• It occurs when animal by-products degrade, so it is a problem if poor quality product is used.
Histamine

- These protein sources may be used at 1-5% because they are inexpensive.

- This disease is common in South America where it is called vomito negro or black vomit.
Histamine

- The compound causes vascular damage in the gastrointestinal tract and causes the birds to bleed to death.
Rapeseed

- Also known as canola. There is a substance in canola that is supposed to be bred out of the grain, which causes ruptured livers.
ENVIRONMENTAL CONTAMINANTS
Diazinon

- This product is used to control fire ants.
- Use inside the chicken house is not legal.
- The pellets are yellow and will attract the birds.
Diazinon

• It is an organophosphate so toxicity results in diarrhea and lacrimation.

• On postmortem you may find yellow pellets in the crop and gizzard.

• The compound is rapidly metabolized so residues will not be present in the tissues.
Formaldehyde

- Companies used to use formalgen crystals in hen nests to control bacterial contamination on egg shells in an effort to improve chick quality.

- These flakes, when exposed to moisture will produce gas.
Formaldehyde

• But the chickens will eat the flakes which will burn holes in the lining of the gastrointestinal tract.

• On postmortem you may still smell the formaldehyde odor.
Quaternary Ammonia

- This is a commonly used disinfectant.
- Turkeys are particularly sensitive to this product.
- It will cause oral lesions that look like T-2 when used in drinking water.
- Levels of 100 ppm will cause weight depression in broilers but will kill turkeys.
Sulfur

- This product is commonly used to change the pH of the soil which decreases the survival of some pathogens and improves bird performance.
Sulfur

• The sulfur is placed on the floor at a rate of 300 lbs./16,000 sq.ft.

• It should but wetted in order to get it down into the soil.

• Then clean shavings should be applied.
Sulfur

• If the sulfur is left in powder form, the birds can scratch down to it through the litter.

• If the powder gets on the birds and contacts any moisture it will produce sulfuric acid which causes skin burns and blindness.

• Birds may die as a result.
ANTIMICROBIALS
Sulphonamides

- Toxicity will produce a hemorrhagic syndrome.

- Birds are depressed, pale, and underweight.
This condition occurs when sulphas are used to treat disease problems because the therapeutic level in poultry is close to the toxic level.
Sulphonamides

- Sulphonamides are difficult to mix evenly in feed. Both feed and water administration is used.

- Toxicity can occur when no allowances are made for increased consumption to meet metabolic needs or for high environmental temperature.
Sulphonamides

- Egg layers have decreased production and shell quality with decrease in brown pigmentation.
- Grossly, hemorrhages in skin, muscle, and internal organs are the most consistent lesion.
- Normal dark-red bone marrow in growing birds changes to pink in mild cases and yellow in severe cases.
- The intestinal tract may be spotted with petechial and ecchymotic hemorrhages.
NUTRITIONAL PROBLEMS

1. Human error
2. Improper formulation
3. Ingredient variation
4. Malabsorption
5. Overproduction -- under consumption
6. Feed separation - especially with calcium in chain feeders
7. Ingredient antagonism
8. Oxidation of ingredients
Most companies run their own analytical chemical lab tests to test the feed ingredients.

Changing genetics of the birds requires constant nutritional changes.
MOST COMMON PROBLEMS

1. Ca/P. Imbalance
   (A) Rickets
   (B) Osteomalacia

2. Sel./Vit. E deficiencies - this is still seen especially with corn from the Midwest. The soils there are selenium deficient. Therefore supplements are used.
   
   Add 0.1 ppm to chicken feed
   Add 0.2 ppm to turkey feed

This is not high enough to meet requirements which are 0.2-0.3 ppm but these levels are legal.
RICKETS AND OSTEOPEROSIS

- Soft bones in bird due to a deficiency or improper balance of Ca, P & Vit. D₃.
- Chickens need Vit. D₃ to absorb calcium.
- Approximate proper levels:
  - Broilers and pullets: 0.8-1% Ca-0.6% Phosphate. The ratio of Ca and Phos. is just as critical as the total amount. An excess of one results in an effective deficiency in the other.
  - Pre-lay diet: 2% Ca++
  - Layers: 3% Ca-0.6% Phos.
RICKETS AND OSTEOPEROSIS

- Problem usually caused by human error or miscalculation.
- Frequently seen as a sequelae to the malabsorption syndrome.
- The fastest producing broilers are on the edge of rickets all the time due to increased growth with low calcium levels.
CLINICAL SIGNS

Osteoperosis (medullary bone)

2. Layers go down in their cages and stop laying eggs.
4. Many times birds appear to have spontaneous fractures of wings and legs.
5. Weight bearing bones are brittle and easily broken.
6. Poor producing individuals not affected.
7. This has almost been eliminated with the use of pre-lay diets.
CLINICAL SIGNS

Rickets

1. Poultry 3 to 6 weeks of age often affected.
2. Crippled birds that squat.
1. If you kill the bird by pulling its head you may also break its legs.

2. Bones break quite easily.

3. Ova may be beginning to show regression.

4. Rib joints may fold - richetic rosary.

5. Malformation of the sternum.
TREATMENT AND PREVENTION

- Maintain proper amounts and proper ratio of Ca, Phos., and Vit. D$_3$.

- Top dress feed with dicalcium phosphate.
COMMENT

- Caged layers may be on NRC recommended Ca, Phos. & Vit. D$_3$ and still get “cage fatigue.”

- Usually this is a low Phos. problem caused by chickens being denied access to their own droppings.

- Increasing the Phos. to 0.8% will alleviate this problem.
VITAMIN E. DEFICIENCY

Encephalomalacia  
(Crazy chick syndrome)

- Fat soluble vitamin, easily destroyed by oxidation.
- Required for proper maintenance of brain tissue, particularly in growing birds.
- Required for proper muscle development in gizzard of turkeys; selenium is involved in Vit. E metabolism.
ETIOLOGY

- Oxidative destruction of fat (rancid).
- Vegetable oils are abundant in alpha Tocopheral.
- Vitamin E required for proper embryo development and proper brain development in young chickens.
1. Usually occurs between 15th and 30th day of age. Has been seen up to 56th day. Usually occurs at 3 weeks of age.

2. Chicks appear depressed.

3. Assume fixed posture. Motor paralysis due to decreased cerebellar function.

4. Intense excitement when stimulated. Birds flap wings and sit up on their tails and fall backward.
POSTMORTEM LESIONS

Gross

- Chicks: hemorrhage in cerebellum & soft area in any part of the brain
- Pouls: require both Vit. E & selenium deficiency to have Encephalomalacia.
- Myopathy in chickens rare because cystine and selenium deficiency along with Vitamin E deficiency required.
POSTMORTEM LESIONS

HISTOPATH.  - Encephalomalacia.
  Must differentiate from A.E.
  especially in chicks.

DIAGNOSIS  - Histopathology - typical microscopic
  lesions of Encephalomalacia:
    necrosis, hemorrhage.
TREATMENT

- Addition of 50 mg/lb or 10,000 units of Vit. E/ton of feed.

- For best results, make sure selenium is added to the feed at 0.1 ppm for chickens and 0.2 ppm for turkeys.
DO NOT USE RANCID OILS OR FEED PRODUCTS IN POULTRY FEED.

DO NOT STORE FEED OVER 3 TO 4 WEEKS. MAKE SURE BREEDERS ARE GETTING PROPER VIT. E IN DIET.

USE ANTI-OXIDANTS SUCH AS SANTOQUIN OR BHT IN ALL POULTRY FEEDS.
COMMENT

- Vitamin E is not required for egg production or fertility in male birds.
- But Vitamin E for the 1st 7 days of life comes from the yolk.
SELENIUM

- Minute amounts of this trace mineral are needed for proper metabolism. Closely involved with Vit. E metabolism.

- FDA passed regulations to allow for addition of the pure form of this mineral to feeds.
ETIOLOGY

- Feeds (grain) from soils deficient in Selenium.
- Certain sulfur containing amino acids may be tied to Selenium deficiency. The amino acids are probably abundant in fish-meal.
- Deficiency interferes with peroxidase production.
CLINICAL SIGNS

1. First notice pale staggering birds with deformed wing feather, stargazers.
2. Usually seen in 3 to 8 wk. old birds.
3. Fluid noted over keel bone and under wing, and intermandibular edema.
4. Chicks with encephalomalacia may also have selenium deficiency.
5. Poults will be unthrifty with above normal mortality.
POSTMORTEM LESIONS

1. Subcutaneous edema - exudative diathesis.
2. Areas of weeping dermatitis with scabbing - usually caused by secondary bacterial invasion.
This condition was misdiagnosed when it was first observed. It was thought to be a subcutaneous clostridium infection.

Clostridium can be cultured frequently, but probably is a secondary invader.
DIAGNOSIS

- Depends on response to addition of selenium along with symptoms and lesions. Affected flocks usually respond to treatment in about 3 days.

- Selenium toxicity in breeders will cause blind chicks with crooked beaks.
PREVENTION AND TREATMENT

- Selenium at 0.1 ppm for chickens up to 16 wks. of age and 0.2 ppm in turkeys.

- Cannot be used in chickens that lay eggs for human consumption.
RIBOFLAVIN - VIT. B\textsubscript{2}

- **Chicks & poults** - early mortality, poor growth & curled toe paralysis.

- **Breeders** - decreased egg production, high embryonic mortality, decreased hatchability.

- **P.M. Lesion** - pronounced swelling of sciatic nerves. These chicks will be too young to have Marek’s disease.
BIOTIN

- Usually seen as dermatitis of the bottom of the feet in turkeys.

- A deficiency prevents the healing of sores. Supplementation costs about $0.35/ton.
FOLIC ACID

- Occasionally seen in turkeys. They are affected by poor growth, poor feathering, anemia & perosis (slipped tendon).

- Early signs may be seen in the form of neck extension, as birds are too weak to lift their heads.
VITAMIN A

- May appear as pustule-like lesions in the pharynx and esophagus.
METABOLIC DISORDERS
ASCITES

- Water belly
- Pulmonary hypertension
- Right ventricular hypertrophy.
ASCITES

- This condition is characterized by the accumulation of fluid in the abdomen which is caused by a cascade of events related to the need to supply high levels of oxygen to the tissues.

- The condition is most prevalent in fast growing male broilers maintained at high altitudes and when there is some cold stress. Incidence is commonly seen at 5-12% but can reach 25% in extreme cases.
ASCITES

- Ascites is essentially caused by hypoxia. The broiler chicken has a high demand for oxygen necessary to fuel metabolic processes.
- When such demand is increased by very fast growth rate, or cold conditions, the lungs must oxygenate increased quantities of blood.
- At high altitudes the situation is made worse by low oxygen tension in the inhaled air.
ASCITES

- In order to meet the demands for metabolism, the bird attempts to pump more blood through the lungs and so this places extreme stress on the right ventricle of the heart.

- Under normal conditions, the right ventricle is relatively small, but in the case of ascites, this ventricle becomes greatly dilated.
ASCITES

- The weakened ventricle creates back pressure to the various supply systems, a consequence of which is leakage of plasma from the liver, commonly referred to as ascitic fluid (water belly).

- Birds dying of ascites are invariably smaller than non-affected birds, although the condition is undoubtedly precipitated initially by superior growth characteristics.
ASCITÉS

- Ascitic birds often seem to be panting even though there is no apparent heat stress.

- Older birds may show cyanosis, especially around the combs and wattles and death seems to occur spontaneously, especially when birds are excited.
Any infection or condition that affects lung or heart condition is likely to affect the incidence of ascites.

While severe viral infection may damage lung structure there is often a concomitant reduction in growth rate.

Under commercial conditions, Aspergillus infection is the most problematic because it can damage lung structure without a major depression in growth rate.
Ascites can be prevented by decreasing growth rate and reducing oxygen demand.

Under commercial conditions mortality due to ascites can be minimized by providing quality air flow across the birds and preventing night time cold stress.
ROUND HEART and
AORTIC RUPTURE - Dissecting Aneurysm

- These are two unrelated conditions occurring in fast growing turkeys.
ROUND HEART

- Round heart is most prevalent in 2-4 week old toms where high mortality is associated with enlargement and rounding of the apex of the ventricles.

- Affected birds usually show dilation of the right ventricle alone. Round heart is sometimes associated with ascites.
AORTIC RUPTURE - Dissecting Aneurysm

- Aortic rupture most commonly affects older turkeys (>12 weeks) and again males seem to be more susceptible.
- Affected birds appear normal immediately prior to death, the onset of which is very sudden.
- At necropsy, the musculature is often pale, presumably due to loss of blood, while the body cavity contains massive hemorrhage.
- The aorta will have a split in it. This condition can be induced by a copper deficient diet.
PREVENTION

- The etiology of both conditions is unclear and there is no good treatment available.
SUDDEN DEATH SYNDROME

- SDS, flip over
- This is a condition affecting fast growing broiler chickens, usually males.
- The normal incidence is from 1.5 to 2.5% of the flock and peaks between 21-28 days of age.
- Afflicted birds appear healthy, are well fleshted, and have feed in their digestive tracts.
- Death occurs within 1-2 minutes and the birds are usually found on their backs.
SUDDEN DEATH SYNDROME

- There are few postmortem lesions (blood clots inside the heart, edematous lungs) and diagnosis is based on signalment, history, and exclusion of other diseases.

- The condition is precipitated by fast growth rate so can be prevented through nutrient restriction.

- The condition seems to be prevalent when ionophore anticoccidials are used.
The condition can best be prevented or reduced in incidence by inducing a period of initial slow growth. This is usually achieved by a reduction in day length.
FATTY LIVER HEMORRHAGIC SYNDROME

- Most often seen in young adult cage layers. Occasionally in older caged hens and floor layers.

- Environmental temperatures and geographic location influences occurrence. Worldwide incidence.
ETIOLOGY
(NOT COMPLETELY KNOWN)

Theories:

1. Simple corn soy diet
2. Estrogenic hormones
3. Over-consumption of feed (positive energy balance) secondary to a marginal calcium content of the diet. Pullets being left on low calcium too long after production starts. Need to use a pre-layer diet. Hens will eat to supply their calcium requirement.
4. Difference in susceptibility among genetic strains of birds.
5. Higher energy ration and lack of exercise.
6. Infectious organism - ruled out?
7. Nutrient deficiency (selenium +?)
## ETIOLOGY

Effect of corn or fish meal on the liver in hens on isocaloric diets

<table>
<thead>
<tr>
<th>Diet (% Prot.)</th>
<th>Weight (G/kg BW)</th>
<th>Fat (% of DM)</th>
<th>Hemorrhages (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn-soy (12.5)</td>
<td>38</td>
<td>43</td>
<td>58</td>
</tr>
<tr>
<td>Corn-fish (12.5)</td>
<td>26</td>
<td>26</td>
<td>16</td>
</tr>
<tr>
<td>Corn-soy (16.5)</td>
<td>40</td>
<td>36</td>
<td>67</td>
</tr>
</tbody>
</table>

Jensen (1979)
ETIOLOGY

Estrogenization of young female chicks

<table>
<thead>
<tr>
<th>Estrogen Injections (5X) (MG/KG)</th>
<th>Liver fat (% of DM)</th>
<th>Livers with hemorrhages</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>14.4</td>
<td>10</td>
</tr>
<tr>
<td>2.5</td>
<td>19.3</td>
<td>20</td>
</tr>
<tr>
<td>5.0</td>
<td>32.4</td>
<td>60</td>
</tr>
<tr>
<td>10.0</td>
<td>29.3</td>
<td>100</td>
</tr>
</tbody>
</table>

Pearsons and Butler (1978)
SIGNS

1. Hens look too good - oily feathers

2. Primary economic loss - increased mortality

3. Low egg production peak - drops too quickly

4. Egg production may not be affected
LESIONS

1. Hemorrhages present on surface of liver.

2. Bright yellow liver with fat droplets.

3. Liver rupture with free blood in abdominal cavity.

4. Heavy abdominal fat 1-1/2" thick (variable).
DIAGNOSIS

- From lesions and history
LABORATORY FINDINGS

- **Normal liver** - 5-10% fat on wet basis and 20-30% fat on dry basis.

- **Affected liver** - 15-30% fat on wet basis and 40-70% fat on dry basis.
PREVENTION

- Use diets with ingredients that contribute selenium + unidentified nutrient: wheat, alfalfa meal, fish meal, etc.

- Do not use plain corn-soy diets without additives.
FATTY LIVER AND KIDNEY SYNDROME

- Seen where non-corn base diets are used. Occurs in 2-5 week old broilers.

- Usually on wheat or barley base diets.

- Biotin supplementation prevents condition.
MORTALITY

- Usually 1-2%. Occasionally up to 30%.

SIGNS

- CNS signs similar to "crazy chick".

POSTMORTEM LESIONS

- Pale swollen liver with hemorrhages.
Lipid content of liver and kidney is doubled.

From 2-3% to 5-6% or more
Kidney dysfunction often leads to either visceral or articular gout, or urolithiasis.

In all cases, increased substrate load to the kidneys eventually leads to precipitation of insoluble products within the kidney or in other regions of the body.

This often occurs with dehydration.
Gout describes the condition in which the plasma uric acid leads to precipitation of monosodium urates either in the synovial fluid and tendon sheaths of various joints or the serous surface of various visceral organs.

Visceral and articular gout are distinct forms and must be considered to be of different etiology.
GOUT AND UROLITHIASIS

- Rarely are the two forms seen together.

- In articular gout, urate crystals appear in the joints accompanied by an inflammatory response while visceral gout is characterized by white urate deposits on the surface of visceral organs and membranes.
GOUT AND UROLITHIASIS

- Articular gout is most commonly seen in birds fed excess levels of protein.

- Urolithiasis is most commonly seen in Leghorn hens and is induced by feeding high levels of calcium for a long time prior to sexual maturity.
DIFFERENTIAL DIAGNOSIS

- Oosporein, ochratoxin, infectious bronchitis.
PREVENTION

- Urolithiasis - control calcium levels in diet, avoid exposure to IBV and nephrotrophic mycotoxins.

- Gout - avoid excess levels of dietary protein and assure adequate water supply.
TIBIAL DYSCHONDROPLASIA - TD

- This condition involves incomplete ossification at the active growth plate and occurs in fast growing meat birds but is most common in broiler chickens usually at 21-35 days of age.

- The exact cause is unknown but the unusual development of the cartilage plug at the proximal growth plate of the tibia can be induced by a number of factors.
TIBIAL DYSCHONDROPLASIA - TD

- The incidence can be greatly increased by metabolic acidosis induced by feeding products such as NH$_4$Cl. This can cause a disturbance in Ca metabolism.

- It seems as though TD occurs more frequently when the diet contains an excess of sodium relative to potassium when chloride levels are very high.
PREVENTION

- Adjustment of dietary levels of Ca:K and consideration of dietary electrolyte balance, and reduced growth rate.
COMMENT

- The incidence of most metabolic disorders can be greatly reduced when birds grow more slowly.

- This can be best accomplished by modification to the lighting program.
# Reduced day length programs for broilers

<table>
<thead>
<tr>
<th>Age of bird (days)</th>
<th>Blackout housing</th>
<th>Open sided housing</th>
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<tr>
<td></td>
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<td>15-18</td>
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<td>19-23</td>
<td>18h</td>
<td>23h</td>
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<tr>
<td>24+</td>
<td>23h</td>
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</table>
AVIAN COCCIDIOSIS

- One of the most potentially destructive diseases in domestic poultry production.
- Most costly of all poultry diseases.
- Strictly a gut infection in chickens and turkeys.
- All avian species affected.
- Host specific.
ETIOLOGY

- Protozoa - genus *Eimeria*.
- Direct short life cycle, sexual and asexual phases with high reproductive potential.
- 1 oocyst produces 1,500,000 oocysts.
- Self limiting - severity of infection, dosage dependent.
ETIOLOGY (CONT.)

- Modern poultry production methods encourage severe infection. Mixed infections are common in chickens.
- Poultry raised on the floor are highly susceptible throughout their life.
- Pullets raised on wire away from fecal contamination have a low chance of infection but remain susceptible.
- Oocyst are resistant to adverse environmental conditions.
- Exposure related immunity develops.
SPECIES AFFECTING CHICKENS

*Eimeria necatrix*

- "tenella"
- "brunetti"
- "maxima"
- "acervulina"
- "mivati"
- "mitis"
- "praecox"
- "hagani"

- "bloody"
- shallow
- ? pathogenic
Sue Clanton:
Add in pages showing areas of gut infected with different species
Pages 386-387
INCUBATION PERIOD

- Depend on species of coccidia 7-8 days to complete life cycle.

- **Mortality** - depends on coccidia species and dosage. Usually 5 to 8 days after infection.

- **Blood appears** - 4-5 days depending on coccidia species.
COURSE OF DISEASE

- 1-3 wk. on a flock basis.
- Depends on species of coccidia.
- Immunity develops.
ECONOMIC EFFECT

- Mortality - variable.
- Poor feed conversion and weight gains.
- Reduction of desired pigmentation.
- The lack of uniformity in pullets.
- Above depends on species of coccidia, dosage, condition of overall health and genetics of the chicken.
- Every ton of feed has $5/ton of coccidiostat.
METHOD OF SPREAD

- The natural behavioral traits are conducive to ingestion of sporulated oocyst.

- Starts in a few birds and dosage is built up over one or two passages and whole flock may be exposed.
SIGNS

1. Typical "sick bird" - depressed and ruffled feathers.
2. May or may not have bloody diarrhea depending on species of Coccidia.
3. Mortality - may be first thing noticed. Species dependent.
4. Loss in egg production - rare because usually exposed and immune before the start of lay.
POSTMORTEM LESIONS

- *Eimeria tenella* - cecal coccidiosis.
- Erosions of cecal wall with free blood and bloody cores in ceca.
- Caseous cores (old cases).
- **Micro** - presence of ovoid oocyst in cecal scraping from sub-epithelium.
- Oocysts are double walled.
POSTMORTEM LESIONS

- *Eimeria necatrix* - mid gut.
- Ballooning in mid gut. Parasite in sub-epithelium.
- **Serosa** - white spots and petechial hem.
- **Open gut** - mucoid blood-filled exudate.
- **Micro** - gut has large schizonts (single wall with little definition) but no oocyst. Life stages moves to ceca where oocysts are found.
- Ceca-oblong ovoid oocyst with no wall erosion.
- Less likely to be a mixed infection.
POSTMORTEM LESIONS

- *Eimeria brunetti* - mainly in lower SI and rectum. Some infection in mid SI and ceca.
- Serosa - ecchymotic white areas with thickening of lower SI and rectal walls.
- Open gut - coagulative necrosis, mucoid bloody enteritis in lower gut.
- Micro - large ovoid oocyst in coagulative material. Infection in sub-epithelium.
POSTMORTEM LESIONS

- *Eimeria maxima* - midgut - “salt and pepper” lesions.
- **Serosa** - faint hemorrhages and wall ballooning.
- **Open gut** - blood-tinged mucus sometimes orange exudate.
- **Micro** - large golden color oocyst in gut is diagnostic. 30 x 20 microns.
POSTMORTEM LESIONS

- *Eimeria acervulina* - usually in duodenal area.
- **Serosa** - white plaques that may be elongated and have a tendency to be "Ladder like" (horizontally) or in severe infections coalesced plaques and wall thickening. The ladder lesions are becoming less common.
- **Open gut** - only the epithelium is affected. May be whitish petechiae to coalesced lesions with a milky appearance (oocyst) in severe infection. Pathology depends on dosage.
- **Micro** - small (18 x 14 microns) ovoid oocyst from gut epithelium.
POSTMORTEM LESIONS

- *Eimeria mivati* - some disagreement over the actual authenticity of this being a separate species from *E. acervulina*.
- Occurs in the epithelium of the upper gut.
- **Serosa** - similar to *E. acervulina* but round lesions and more gut thickening.
- **Open gut** - depending on dosage from individual plaques to coalesced large infected areas that appear milky.
- **Micro** - similar to *E. acervulina* with more macrogametocytes present mixed with oocyst.
POSTMORTEM LESIONS

- *Eimeria mitis* - low in pathogenicity lower third of the SI.
- Serosa - difficult to see any lesions.
- Open gut - slight mucoid appearance, look for oocyst with microscope.
- Micro - very small (15 x 14 microns) subspherical oocyst.
- *E. praecox* - slightly pathologic causing diarrhea.
- *E. hagani* - needs research.
DIAGNOSIS

- The presence of lesions and some stage of the life cycle of coccidia (usually oocyst).
- Need to look at live and dead birds.
- Select birds typical of the flock, not culls. Culls may be off feed and not ingesting coccidiostat so are very likely to have cocci even if it is not a flock problem.
- Must use light microscope to confirm.
- Can speciate by location of lesions and type and size of oocyst.
The serosal surface is examined for white plaques and hemorrhagic petechiae. The area of the gut affected is considered.

The gut is opened and the mucosa of the affected area is scraped off with a spatula and placed on a microscopic slide.

A cover-slip is applied to the scraping and mashed to produce a thin smear.

The slide is then examined with a light microscope. Low power (100x) is used for scanning and high dry power (430x) for detail examination and measurements.
Many times coccidiosis will be caused by a mixture of species. Usually if birds are dying, there is a predominance of one deep invading species.

Coccidiasis-presence of stage of life cycle (usually oocyst) without pathology to the gut or effect on production.

Disease has a tendency to be over-diagnosed by servicemen.
COMMENT

- Other enteritis problems may appear similar to coccidiosis. However, a microscopic examination should clarify the diagnosis. Don’t treat for coccidiosis unless you know for sure.
Follow directions closely.
Treatment - usually in water.
Overtreatment can cause drug toxicity (Sulfas).
Take into consideration the season of the year. Sulfa drugs can be toxic if birds change their drinking habits based on temperature.
Some drugs can be used for both treatment and control.
Treatment - really prevention.
## TREATMENT CHICKENS

<table>
<thead>
<tr>
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<th>Water</th>
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<tbody>
<tr>
<td>Sulfaquinoxaline</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Amprol®*</td>
<td>+</td>
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<tr>
<td>Sulfadimethoxine</td>
<td>+</td>
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* Approved for laying hens
PREVENTION

1. Genetics - not developed.
2. Nutritionally - certain vitamins help but not reliable.
3. Quarantine & depopulation - not practical - can make things worse.
4. Sanitation - not practical - may produce more susceptible birds.
   Very resistant to chemicals.
5. Reduced exposure - shift from floor to cage rearing but bird must remain in cages or on wire for life.
6. Immunization - requires exposure (natural or planned) to live coccidia. This is used in broiler breeders or leghorns. Vaccine available.
7. Chemotherapy - coccidiostats - presently most practical and most used.
## Trade Names

<table>
<thead>
<tr>
<th>Ionophores</th>
<th>Withdrawal Period (days)</th>
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<tr>
<td>Coban (Summer)</td>
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</tr>
<tr>
<td>Avatec</td>
<td>5</td>
</tr>
<tr>
<td>Bio-Cox (Summer)</td>
<td>0</td>
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<tr>
<td>Cygro</td>
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<tr>
<td>Monteban (Narasin)</td>
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<tr>
<td>Maxiban (Narasin + Nicarb)</td>
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## Trade Names

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<td>Amprol, Amprol +, Amprol Hi-E (A Thiamine Analog)</td>
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<tr>
<td>Coyden at 0.125% (25% premix)</td>
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<tr>
<td>Zoamix</td>
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<tr>
<td>Rofenaid</td>
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<tr>
<td>Stenoral</td>
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SHUTTLE AND ROTATION PROGRAMS

- These programs have been used to stop strain resistance to specific drugs.
- Rotation involves complete change of drugs for several months or years hoping resistance will disappear.
- Shuttle programs involve the change of Coccidiostats within a grow-out period of a single flock.
TYPICAL SHUTTLE PROGRAM

SUMMER

- Coban - 3-4 weeks
- Avatec - 5th week until withdrawal

WINTER

- Nicarb - 3 weeks
- Coban - 5th week
2-3 cycles of infection to develop sufficient immunity (depending on species of coccidia).

- Coccivac - must give in first 7-10-days and proper management of coccidia shed - need 20% moisture in litter.
- 4-6 species used in the vaccine depending on geography.
- Coccivac - mainly used in breeders, layer pullets, and roasters.
- Coccivac - now given as an eyedrop in the hatchery or sprayed on the feed at 3 days
IMMUNITY (CONT.)

- Important to develop immunity in breeders and floor layers.
- Amprol - step down program.
- Research being done on new immunizing agents.
TURKEY COCCIDIOSIS

- **Etiology** - same genus; *Eimeria*
- **Incubation period** - same as chicken
- **Course of disease** - usually about (4-5 days)
- **Mortality** - low 5% except *E. adenoides*
- **Method of spread** - same as chicken
- **Symptoms** - Not as severe, more like "Shallow" invaders, weight loss
  
  No bloody diarrhea like in chickens.
IMPORTANT SPECIES AFFECTING TURKEYS

- *Eimeria meleagrimitis*
  - "adenoides" pathogenic
  - "gallopavonis"
- 4 other species - non-pathogenic
- *Eimeria dispersa* – affects quail also
  - This one is not host specific.
POSTMORTEM LESIONS

- *E. meleagrimitis* - upper two-thirds white or green cheesy mucous casts
- Blood rare
- Micro-oocyst in small intestine
POSTMORTEM LESIONS

- *E. adenoides* - lower third small intestine ceca and large intestine
- Dilation, edema and caseous exudate "cottage cheese"
- Mortality may reach 100% in young
- This doesn’t occur often and is *not* seen in Georgia.
POSTMORTEM LESIONS

- *E. gallopavonis* - lower third & ceca.
- Same as *E. meleagrimitis*, but in lower intestines and ceca, in between the cecal pouches.
TURKEY TREATMENT

- Sulfaquinoxaline (SQ) - toxic
- Amprol
- Sulfadimethoxine (Agribon®) in water
CONTROL

- Try to develop some immunity in the pouls. Feed coccidiostats during brooding.
  - Sulfaquinoxaline (SQ)
  - Amprol
  - Zoalene
  - Coban - need to start birds on this or can get toxicity characterized by a “knockdown” where birds are recumbent with feet stretched out behind
  - Stenoral
  - Avatec
ENTEROHEPATITIS (BLACKHEAD)

- Most important in turkeys, common in peafowl.
- Occurs in turkeys brooded in old chicken houses.
- Usually in debilitated chickens.
- This occurs in leghorn pullets raised on litter.
CAUSATIVE AGENT

- **Histomonas meleagris** - a one-celled organism/parasite

- Requires cecal worms for natural infection. Turkeys will die of Blackhead before the cecal worms can reproduce. But if cecal worms are present, so is histomonas.

- Viable - 24 hrs - outside host
  151 wks - in cecal worm eggs
INCUBATION PERIOD

3 to 4 days

COURSE OF DISEASE

Subacute or chronic
Subacute: 10 days to 2 weeks
Chronic: Several weeks
No immunity
METHOD OF SPREAD

- Ingestion of cecal worm eggs.
- Infected chickens may be source of turkey infection.
- Earthworms - infected with cecal worms eggs.
- This is a route of infections for pen raised quail.
MORTALITY

- Poults - may reach 80 to 90% when affected before 4 weeks of age
- Older poults - 20-25% or less
- Chicks and chickens - mortality varies
CLINICAL SIGNS

- Typical sick birds
- Sulfur colored droppings - yellow, foamy
- Cyanotic head (turkeys)
- Old birds lose weight
POSTMORTEM LESIONS

TURKEYS AND CHICKENS

Enterohepatitis:
1. Ceca - cheesy core
2. Liver - round sunken lesions
3. Chickens, pheasants and bobwhite quail
   - May not show liver lesions
SUGGESTIVE DIAGNOSIS

- Typical lesions in ceca and liver.

POSITIVE DIAGNOSIS

- Cannot isolate or propagate - will see causative agent on histo in liver tissue
TREATMENT

- Separate sick birds.
- No reliable treatments or preventatives are presently available due to 1987 FDA ruling banning approved drugs.

- **Backyard flocks:**
  - Metronidazole (flagyl)
  - 250 mg/gal-5 days. Palatability?
- A liquid form is used in pet birds.
PREVENTION

1. Rotate range

2. Separate turkeys and chickens

3. Control cecal worms

4. Continuous medication
   Hepzide
LEUCOCYTOZOON DISEASE

- Occurs in Southeast, Mid-west, California, Canada and Western Europe. Usually in Spring or Fall.
- Was reported in Georgia many years ago.
- Causes problems mainly in turkeys under 12 weeks of age.
- A malarial or red blood cell parasite.
- Debilitates birds leading to secondary infection.
ETIOLOGY

- *Leucocytozoon smithii* in turkeys.
- Other species infect ducks, geese, chickens and wild birds.
- Requires the black fly *Simulium solossinae* as a biological vector.
- Sexual stage requires 5 days in a black fly.
- Flies require slow moving water.
INCUBATION PERIOD

- 14-21 days after turkeys are infected by black flies.
COURSE OF DISEASE

- A single "outbreak" usually in flock for 2-3 weeks.
- In individual bird symptoms present for 2-3 days.
- Can become chronic - retards breeding in breeder turkeys.
MORTALITY

- Low to none usually.
- Depends on level of infection which depend on black fly numbers.
- In high black fly density 75% mortality rate has been reported.
POSTMORTEM LESIONS

1. Blood appears thin with prolonged clotting time.
2. Swollen liver with hemorrhages.
3. Swollen, possibly blackened spleen.
4. Congested lungs, black appearing in severe cases.
5. Emaciation with flabby, brownish muscles in chronic cases.
DIAGNOSIS

SUGGESTIVE DIAGNOSIS -
Black fly infestation with bites on turkey heads followed by symptoms which last only 3 days.

POSITIVE DIAGNOSIS -
Symptoms and lesions + demonstration of gametocytes on stained blood smears (Wright's or Giemsa's)
TREATMENT

- Clopidol (coyden) will eliminate the mature gametocytes from blood; however, this will not help treat or prevent the symptoms or lesions.
PREVENTION

- Very difficult.
- Screening out flies, or try to control them with sprays.
- Raise turkeys away from slow-moving stream.
INTERNAL PARASITES (MOST IMPORTANT HELMINTHS)

1. Large roundworms - *Ascaridia sp.* - Most important in chickens and turkeys
2. Cecal worms - *Heterakis gallinarum*
3. Hair worms - *Capillaria obsignata* - quail
4. Crop worms - *Capillaria contorta* and *C. annulata* - quail
5. Tape worms - *Raillietina cesticillus* + 6 others
6. Gapeworms - *Syngamus trachea* - in pheasants
LARGE ROUNDWORMS

Ascaridia galli (chickens)
A. dissimilis (turkey)
A. columbae (pigeons, doves)

Mature worms - A. galli size
Males - 1 1/2-2 1/2 in. long.
Females - 2-4 in. long.
Size affected by crowding.
LIFE CYCLE
(ASCARIDIA GALLI)

DIRECT

*Embryonated egg ↓ Mature adults
   Infective
   Larvae (9 days) ↓
   Penetrate S.I. at 17 day. Young adult in lumen
   *Embryonation takes 10-12 days.

28-30 days
Post infection

Eggs infective 161 weeks on ground.
COURSE OF DISEASE

Worms remain until "self cure" or treatment.

MORTALITY

Usually negligible
Mainly caused by gut blockage.
METHOD OF SPREAD

- Ingestion of sporulated eggs from contaminated environment.
CLINICAL SIGNS

Clinical signs usually seen only in backyard flocks.

1. Depression, loss of weight, diarrhea

2. Retarded growth

3. Lowered egg production in heavy infection (can occur in caged layers exposed to contaminated flies)
POSTMORTEM LESIONS

1. Presence of worms in small intestine
2. Inflamed intestines
3. Emaciation (culling)
 COMMENTS

- Greatest damage - birds under 3 mo. of age - due to larval migration in the small intestinal wall.

- Light breeds more susceptible

- **Malcolm Reid** research
  10-14 worms = 5-7% loss egg production
  1 lb wt loss in broilers per 325 worms
DIAGNOSIS

- Presence of worms on postmortem

- Diagnosis - without sacrifice if worms suspected, can place several birds on wire bottom cage and treat with worming drug. Observe feces.

- Flotation of fecal matter.
TREATMENT

- Piperazine (various salts) nontoxic. Works by paralyzing worms long enough to be evacuated from the intestinal tract.
- Piperazine dihydrochloride
  Most common - powder form for feed or water use.
- Piperazine sulfate (wazine<sup>tm</sup>). Liquid for water use.
- Base dose on Piperazine content.
- Follow directions on label. This doesn’t work as well as it used to. Resistance has developed to the drug.
TREATMENT (CONT.)

Piperazine

- in water most practical.
- Water starve; want high level quickly.

- Dosage:
  
  Chicken & turkey - feed 0.2-0.4%
  
  water 0.1-0.2%

  Single dose chicken 50-100 mg/bird
  
  Turkey < 12 wks 100 mg/bird
  
  > 12 wks 100-400 mg/bird

Severe infestation - repeat in 21 days.
TREATMENT (CONT.)

- **Coumaphos - Meldane 2™ (Hess & clarke)** - feed treatment. May suppress feed intake in heavy breeders.
- Approval - chickens only > 8 wks.
- Pullets - worm 2-3 times with piperazine. Then treat with meldane.  
  - Before laying house.  
  - 40 ppm - 4 lbs/ton - 10-14 days
- Hens - 30 ppm - 3 lbs/ton - 14 days
- Retreat pullets on contaminated floor in 3 wks.
- Effective on roundworms, cecal worms, & Capillaria.
- Levamisole - 8 mg/lb of broiler for Roundworms.  
  - Effective but not approved for use without a prescription.
TREATMENT (CONT.)

- Hygromycin B - chickens only.
  Stops worm egg production
- Usually in pullets, may be used in broilers.
  Not allowed in commercial layers; ok in breeders.
- 8-12 gm/ton continuously
  (12 gm/ton - capillaria)
  (8 gm/ton - rounds & cecal)
- 3 day withdrawal period.
PREVENTION AND CONTROL

- Confinement rearing and caging has reduced problems with most internal parasites.

- Deep litter (4-6" wood shavings) has reduced exposure to worm eggs.

- Worst problems in winter - poor litter conditions are ideal for worm egg maturation.
PREVENTION

1. Proper clean up between flocks.

2. Hygromix continuously. This drug is not longer being produced.

3. Meldane at 3 week intervals.

Examine all flocks routinely at 4-5 weeks, 10 weeks and 15 weeks.
Routinely worm before moving to the laying house.
CECAL WORMS

- *Heterakis gallinarum*

- Primary importance - transmitting Blackhead in turkeys and chickens

- Mature worms - 3/8 to 3/4 inch long
LIFE CYCLE

- **DIRECT** - similar to ascarids. About 65 days to complete. (Not seen in broilers)

- Eggs infective up to 230 weeks on the ground.

- May be transmitted by earthworms.

- *H. gallinarum* eggs pick up histomonas (Protozoa) in gut of infected birds. (Usually chickens)
COURSE OF DISEASE

Worms remain until "self cure" (IgE) or treatment

MORTALITY

Usually negligible
METHOD OF SPREAD
Ingestion of sporulated eggs from contaminated environment

CLINICAL SIGNS
Usually inapparent
POSTMORTEM LESIONS

1. Presence of worms in ceca.

2. Slight thickening of cecal mucosa in heavy infections.

3. Mild cases - no pathology.
DIAGNOSIS

- Presence of worms in ceca.

- Cut off the tip of the ceca and heat with a match.
Phenothiazine in feed - 0.5gm/1 chicken or 1 gm/1 turkey.

Hygromix - 8-12 gm/ton - 8 weeks

Meldane - 30 ppm - 10 days to 2 weeks

Levamisole - effective but not approved.

16 mg/lb of broiler for Heterakis - put in water after water starving birds

Piperazine does not work on cecal worms
PREVENTION

- Same as ascarids.

- Very important to use deep litter (4-6") over floors when changing from chicken production (pullets or layers) to turkeys.

- Blackhead transmission.
CAPILLARIA WORMS

1. Crop & esophagus invaders - *C. contorta* and *C. annulata* burrows in the mucosa

2. Important intestinal form - *C. obsignata* intertwine in intestinal villus

** Capillaria are not a problem in broilers.
CAPILLARIA OBSIGNATA

1. 1/2 - 3/4 in. Long - hair-like

2. Life cycle - direct

3. Eggs infective up to 102 weeks

4. Found in small intestine
MORTALITY

- Negligible
METHOD OF SPREAD

EMBRYONATED EGGS

- May be a problem in heavy breeders in the winter due to increased moisture in the house. Slats in houses decrease the incidence.
CLINICAL SIGNS

1. Infected birds are unthrifty, have diarrhea and loss of weight.
2. Birds are pale.
3. Egg production may decline 15-20% or never peak properly.
4. Produces enzymes to prevent their own digestion which may interfere with Vit. A absorption and may cause white yolks.
POSTMORTEM LESIONS

- Moderate to severe inflammation of duodenum and upper small intestine.

- In severe infections, the intestinal wall may be quite thickened as well as inflamed, resulting in a heavy catarrhal enteritis.
DIAGNOSIS

- Intestinal scrapings washed through 100 mesh screen will reveal the worms. In severe infections, simple scraping with microscopic examination will reveal infection.
TREATMENT

- Meldane - 2-4 lbs. 10% premix/ton - two weeks at two month intervals.

- Hygromycin - hygromix - 12 gm/ton for 8 weeks. May be necessary to maintain flock on 8 gm/ton continuously. This product is not longer being manufactured.

- Levamisol - effective but not approved. 16 mg/lb of broiler for capillaria - in water after water starving birds. It can be used under a prescription.

- Vitamin A - increase 6000 additional units for 2 weeks.
PREVENTION

- Good sanitation.
- Worm layer pullets with meldane just before you put them in the laying house.
- Hygromycin - as in treatment.
- Meldane - as in treatment.
COMMENT

- Problem usually in young adults.
- "Self cure" develops as infection continues.
C. ANNULATA and C. CONTORTA

- *C. annulata* - requires earthworms in life cycle. Occasionally seen in turkeys and game birds.

- *C. contorta* - more important. Most frequent species found in crop infections. Has direct life cycle.

- Adults are from 1 1/2 to 2" long.
COMMENT

- Seen quite frequently in pen raised (on ground) game birds, particularly quail.

- Occasionally seen in turkeys on the range.
MORTALITY

- May be 100% in untreated quail. Seldom high in turkeys, but depresses growth.

- Usually die of starvation.
CLINICAL SIGNS

- **In turkeys** - "penguin" stance due to pendulous crop.

- **Quail** - "typical sick" appearance, birds quite hungry but refuse to eat, just stand and look at feed.

- Birds make "swallowing" motions.
POSTMORTEM LESIONS

1. Thickened and inflamed crop and esophagus walls.

2. Heavy exudate covering lining of the crop and esophagus and sloughing of the membranes.

DIAGNOSIS

- Typical lesions in the esophagus, mouth and crop with burrowing worms present.
- Requires parasitologist for species identification.
TREATMENT

- Meldane - 2-4 lbs. 10% premix/ton - 2 weeks - about 50% effective. May be hard to find.

- Levamisol - effective but not approved.
  1 gm/gal of water for 24 hour consumption.
PREVENTION

- Use new ground.
- Raise on wire.
- Probably continuous use of meldane.
- Method of treating old pens with methyl bromide under plastic tenting is being used with success.
- Need a licensed applicator.
GAPEWORMS (SYNGAMUS TRACHEA)

- Not important in commercial poultry operations.
- Occasionally seen in backyard flocks and pet birds.
- Seen commonly in pheasants grown on straw-type litter in Pennsylvania.
- Transmitted by earthworms mainly.
- Usually in immature birds.
ETIOLOGY

- Male and female worms are generally found attached to each other continuously (y shaped). Worms are attached to the trachea and are red and vary in size depending on the crowding effect.

- Birds gape (mouth breathe) depending on number of worms and amount of blockage.

- A single pair of worms can choke a bird.
DIAGNOSIS

- Worms are found on postmortem in the trachea and bronchi.
- No drugs are approved for commercial use against Syngamids.
TREATMENT AND PREVENTION

- Thiabendazole 0.05-0.1% in feed for 2 wks.
  - Not approved by FDA.

- Levamisol 16 mg/lb of bird for 3 days in water.

- Prevention - stop access of wild birds and stop earthworm exposure.
CESTODES

TAPEWORMS

- Seven species affect chickens
- All require intermediate hosts
- *Raillietina cesticillus* most commonly found - darkling beetle - Intermediate host
Present husbandry excludes the most important pathogenic species.

*Davainea proglottina* requires slug as intermediate host.

Other species probably non-pathogenic except in large numbers.

*Hymenolepis sp.* are so small they may be completely overlooked and found in Coccidia scraping.
DIAGNOSIS

Presence of tapeworms

TREATMENT

No legal treatment available.
PREVENTION

- Control intermediate hosts such as flies, ants and beetles.
- Sevin dust or spray is commonly used for this.
The control of ectoparasites and flies cost industry over $200 million annually.

Most of this money is used on fly control in caged layer manure that constitutes a menace to public health and public perception.

However, ectoparasites on poultry cause production losses and disease spread that must be controlled for poultry to reach its full production potential.
COMMON ECTOPARASITES

MITES AND LICE
- The most common ectoparasites seen in commercial poultry operation.

BEDBUGS
- Occasionally infest chickens and may cause them to become anemic and avoid using nests.

CHIGGERS
- In turkeys kept on the range - skin lesions may cause downgrading of products at slaughter.
MITES

- There are two species of mites that cause economic losses in U.S. Poultry.

- Northern fowl mite - *Ornithonyssus sylvarium* is the most important.

- Chicken mite - *Dermanyssus gallinae*, also known as red mites or roost mites may occur, particularly in backyard flocks.
NORTHERN FOWL MITES
(FEATHER MITES)

- Causes economic loss due to reduced egg production (up to 15%), increased feed consumption, reduced weight gains, and reduced seminal fluid production in roosters.

- Entire life cycle on host that can be completed in 5 days. Adults survive off the host for 3-4 wks. under suitable conditions.

- Most problems are in cooler months.
DIAGNOSIS

- Presence of mites on feathers around the vent area in hens and over the entire body of males.
- Check several birds, some may not be infested.
- Use bright light and magnification.
- Light stimulates mite movement.
- Mites appear grey when looking for a meal and black after feeding.
SIGNS AND LESIONS

1. Most often seen in young layers 20-30 wks. of age.
2. Rough and scaly skin.
3. Darkening of feathers around vent due to the accumulation of dried mite excreta and mite eggs.
4. Heavy infestation may cause anemia since mites eat blood & body secretions.
5. Scabs on "feeding areas" may be seen around vent or on dressed carcasses.
CONTROL

1. Make sure house is clean and free of mites before stocking.

2. Make sure new birds are mite free.

3. Prevent transport of mites on contaminated clothing, egg flats, and equipment.

4. Remember mites can live several weeks off the host. Equipment movement from infested houses to clean houses causes new infestations.
5. Use new egg flats and steam clean all equipment moved into a clean house.

6. Exclude wild birds and rodents from poultry houses.
CONTROL (CONT.)

- If infestation occurs, established chemical control methods must be carried out with spraying equipment that will provide high enough pressure for the spray to penetrate the feathers, especially in the vent area. Wetting agents can help penetration. Use the spray under the cages and spray up at the birds. May also dip each bird in solution.

- Ideal spray pressure is 100 to 125 lbs./sq. inch. (psi) with 1 gallon of spray per 100 birds.
CONTROL (CONT.)

1. Follow the directions and instructions found on the spray container closely.
2. Emulsifiable concentrates mix better and are easier to use than wettable powders.
3. Insecticide dust may be placed in the nest of breeders.
4. In caged layers spray upward beneath the cage (vent area of the bird).
5. Spray breeders at night when they are on the slatted area.
When selecting an insecticide refer to UGA Cooperative Extension Service Circular 805, "Insecticide use chart for egg and poultry producers".

The back side of this circular has an excellent guide on mixing and applying insecticide sprays.
THE CHICKEN MITE
(RED OR ROOST MITE)

- This mite is a blood feeder that infests birds during the mites feeding period which is mostly at night.
- When not feeding, the mite hides in the cracks and crevices of poultry houses and objects within, especially roost slats and nest boxes.
- This mite can transmit diseases caused by viruses and bacteria.
SIGNS AND LESIONS

- Same as for northern fowl mite but mites are found on the birds, usually at night only.
DIAGNOSIS

- Mites are gray or red depending on the timing of the last blood meal.

- Use strong light and magnification to see mites around vent. Best to look at night.

- Infestations with red mites will cause premises to have an offensive odor due to decomposed blood products in mite excreta.
CONTROL

- Same as for Northern fowl mites.

- Since this mite can survive off of the bird for several weeks in protected areas additional attention is needed in the cleaning and chemical spraying of the infested house and its contents.

- A high pressure (175-200 psi) spray is required to penetrate cracks, crevices, and hiding places. Do not use more than 125 psi directly on birds.
The chicken body louse, *Menacanthus stramineus*, is common in both chickens and turkeys.

- It is about 1/8" long.

- Several other species of lice can occur but rarely in numbers large enough for economic losses.
CHICKEN BODY LOUSE

- Body lice may build up large populations in poultry, especially on caged layers. Lice spend their entire life cycle on the host. One pair can replicate to 120,000 lice in 3 months. They live for several months.

- Lice infesting poultry are chewing lice not blood suckers. They feed on dander and feather parts.

- Egg production losses up to 46% and body weight losses can occur in hens.
DIAGNOSIS

- **SUGGESTIVE DIAGNOSIS** - ragged appearance of feathers with fluff missing from feather barbs (eaten off).

- **POSITIVE DIAGNOSIS** - finding lice (about 1/8" long) on feathers and skin usually around the vent. Lice are photophobic and move very fast.

- Nits (white egg capsules) - present around the base of vent feathers.
CONTROL

- Same as Northern fowl mites.

- Due to slow hatching of lice eggs, a second spray 10-14 days after the first may be needed since chemicals are not ovicidal.
**BEDBUGS**
**(CIMEX LECTULARIS)**

- Usually seen in breeder houses and interferes with production by causing anemia and causing birds to resist using infested nests. Bedbugs are about 1/4" long and suck blood.

- Hard to diagnose - examine house. Look in cracks and crevices.

- Behave similar to chicken mites (red mites) but are much larger (about 1/4" long). They are nocturnal feeders and may be hard to diagnose because they get on bird for a blood meal that takes only 5-10 mins. Usually diagnosed by finding them in cracks and crevices of the house or equipment. Suspected when insect bites appear on anemic chickens.
CONTROL (BEDBUGS)

- Same as for chickens mites (red mites).
- Concentrate on cracks and crevices of house and equipment.
- May have to spray a second time due to the low toxicity of available approved chemicals.
- Use a product with good residual effect.
CHIGGERS

- A problem in turkeys ranged on low brush-type pasture usually including pine trees.
- Soil type is important. The parasite requires clay type soil that cracks in dry weather.
- The free living form of this parasite has never been found in nature.
- Cause down grading because of reddened lesion where the chigger embeds in the skin.
DIAGNOSIS

- Should check market birds about 5 weeks before market time.

- Look for red areas under wings and in areas where feathers are sparse.

- Takes 3 1/2 - 4 weeks for lesion to heal.
CONTROL

- Treat infested or suspect infested ranges as described in UGA Cooperative Extension Service Circular 805.

- Dursban* (chlorpyrifos) or Malathion** are treatments of choice.

- *Use on ranges only.

- **Can be used on birds.
DARKLING BEETLES

- These litter beetles will migrate from chicken houses to peoples’ houses after the chickens are moved out.

- They pupate in the soil. Placement of boric acid on the soil will destroy the larvae and control populations.

- These beetles are able to concentrate Botulism toxin, carry tapeworm eggs, and transmit bacterial and viral diseases.