الحقيبة التعليمية

قسم الصحة الحيوانية

المرحلة الثانية

المادة / أمراض دواجن

أسم المدرس / د.وليد محمد حسن

وزارة التعليم العالي والبحث العلمي
هيئة التعليم التقني
التخصصات : الزراعية
القسم:الصحة الحيوانية
الفرع:

عرشي

المقررات الدراسية

اسم المادة	باللغة العربية	امراض دواجن	السنة الدراسية	عدد الساعات الاسبوعية			
	باللغة الانكليزية	Poultry Diseasea	الثانية	نظري	عملي	المجموع	عدد الوحدات
لغة التدريس للمادة		الانكليزية		2	3	5	5

اهداف المادة العامة والخاصة: يتعرف الطالب على جميع الامراض التى تصيب الطيور الداجنة وكيفية تشخيصها وعلاجها والحد من انتشارها

المفردات النظرية	
الاسبوع	*تفاصيل المفردات*
الاول	تشريح الدواجن ـ عملية فتح الجثة ـ الاجهزة والادوات المستعملة ،نظرة عامة عن اجهزة الجسم المختلفة .
الثاني	الجهاز الهضمى ،الجهاز التنفسي
الثالث	الجهاز البولي والتناسلي ، جهاز الدوران
الرابع	الجهاز المناعي ،الجهاز العصبي
الخامس	تعريف المرض ،اسبابة ،تصنيف امراض الدواجن حسب مسبباتها المرضية ،كيفية تشخيص امراض الدواجن
السادس	الامراض الفايروسية (النيوكاسل ،التهاب الشعب الهوائية المعدي ،جدري الطيور ،الكمبورا
السابع	التهاب الدماغ والحبل الشوكي ،التهاب الكبد المعدي ،انفلونزا الطيور
الثامن	الامراض البكتيرية : (البلورم ،التايفونيد ،اليارانايقونيد ،الاصابة بالشريشيا القولونية)
التاسع	السل ، زهري الطيور ،التهاب الامعاء النخمري
العاشر	التهاب الاكياس الهوائية ،الزكام المعدي ،كوليرا الدواجن
الحادي عشر	الامراض السرطانية : (مرض مارك ،البكواسز ،الاورام السرطانية)
الثاني عشر	الامراض الطفيلية : (كوكسيديا ،هيستوموناس ،ترايكوموناس)
الثالث عشر	امراض نقص التغذية : (البروتين ،الكاربوهيدرات ،الدهون ،الفيتامينات ،الاملاح)
الرابع عشر	الامراض الفطرية (داء الرشاشيات ،فطريات القناة الهظمية ، امراض الجلد)
الخامس عشر	امراض اخطاء التربية (التسمم ،خراج القدم ،التهاب عضلة الصدر)

المقررات العملية	
الاسبوع	تفاصيل المقررات
الاول	تشريح الدواجن ،التعرف على اجهزة الجسم
الثاني	التعرف على التغيرات المرضية والافات التي تحدثها الامراض المختلفة ، تشخيص الامراض بالطرق التشريحية .
الثالث	اسباب هلاك الدواجن ،العوامل التي تساعد على حدوث المرض والحد منها .
الرابع	التفريق بين الامراض البكتيرية ، الفايروسية ،الطفيلية ، وامراض نقص التغذية
الخامس	الاعراض والوقاية والعلاج للامراض الفايروسية ،النيوكاسل ،التهاب الشعب المعدي ،جدري الطيور
السادس	الكمبورا ،التهاب الدماغ والحبل الشوكي
السابع	التهاب الكبد المعدي ،انفلونزا الطيور
الثامن	الاعراض والوقاية والعلاج للامراض الفايروسية (البلورم ،التايفونيد البارا تايفونيد)
التاسع	الاشيريشيا القولونية ،السل ،زهري الطيور ،التهاب الامعاء النخثري.
العاشر	التهاب الاكياس الهوائية ،الزكام المعدي ، الكولير ا
الحادي عشر	اعراض الامراض السرطانية
الثاني عشر	انواع الطفيليات الداخلية والخارجية والتعرف عليها وطرق السيطرة عليها
الثالث عشر	البرنامج الوقائي لحقول الدواجن ، طرق اعطاء الادوية
الرابع عشر	التوصيات المختبرية لغرض تشخيص امراض الدواجن (الدم البراز) التعقيم والتطهير في حقول الدواجن
الخامس عشر	زيارة علمية لاحد حقول الدواجن

1. Disease

Any change of animal from a state of good health can be called a disease.

This may be caused by specific pathogenic agents, nutritional deficiencies, or an environment to which the animal can n't readily adapt. .

The diseases are caused by :-

(1) Bacteria.

(2) Viruses.

(3) Fungi.

(4) Protozoans.

(5) Environmental poisons.

(6) Behaviour a bnormalities.

(7) External parasites.

Animal have many defense Mechanisms against attack by disease – producing agents. Much of strategy in disease control uses these natural

defense of the animal by methods such as astimulating antibody production against specific disease agents through immunization, or decreasing the intensity of exposure to disease - causing agents through sanitation, quarntine, and good ventilation so that the odds against the animal are improved.

Disease may be spreads from one animal to another by two basic routes.

1- **Horizantal spread of disease** occurs between one infected animal and another this may

Invoive :-

(a) contact with infected animals.

(b) contact with contaminated letter.

(c) Air born dust.

(d) Particles containing disease organisms.

(e) Feed or water contaminated infected animal.

Poultry

NEWCASTLE DISEASE

(Avian pneumoencephalitis)

Review

Etiology and Epidemiology: Newcastle disease is caused by an RNA virus, paramyxovirus-1 (PMV-1), that can be categorized into three groups: the **velogenic** strains, which are highly pathogenic and easily transmitted; the **mesogenic** strains, which show intermediate pathogenicity; and the **lentogenic** strains, which show low pathogenicity in chickens. Isolates that cause the respiratory-nervous syndrome, even those that are highly pathogenic, usually produce few or no distinctive gross lesions; however, isolates that cause the viscerotropic syndrome often do. Velogenic and mesogenic virus isolates kill injected 10-day-old chicken embryos in 2-4 days, lentogenic isolates usually in 4-6 days or not at all.

Virus is shed during incubation, during the clinical stage, and for a varying but limited period during convalescence. It is present in exhaled air, respiratory discharges, feces, eggs layed during clinical disease, and all parts of the carcass during acute infection and at death. Chickens are readily infected by aerosols and by ingesting contaminated water or food. While the chicken is the primary source of virus, other domestic birds and certain wild birds are susceptible and may be sources. Parrots, mynahs, and such caged birds as pittas that moved in commercial channels were the principal source of infection during the 1970-72 pandemic (USA) of the velogenic viscerotropic form of Newcastle disease. An outbreak of PMV-1 occurred in pigeons in the USA and the UK during 1984. This pigeon paramyxovirus is lentogenic for chickens and occurs worldwide in some pigeon populations.

Clinical Findings: The signs depend greatly on whether the virus is neurotropic or viscerotropic. The neurotropic viruses result in respiratory and nervous signs. The viscerotropic viruses, which predominate worldwide, cause respiratory signs with peracute disease, watery-greenish diarrhea, and swelling of the tissues of the head and neck. Nervous signs are also frequently seen in exotic bird species (parrots, etc) infected with viscerotropic viruses and in poultry species that survive beyond the acute phase of the disease.

Onset is rapid, and signs appear simultaneously throughout the flock 2-12 days (average 5) after exposure. Young chickens are more susceptible and show signs sooner than older ones. Respiratory signs include gasping, coughing, sneezing, and rales. Nervous signs include drooping wings, dragging legs, twisting of the head and neck, circling, depression, inappetence, and complete paralysis; they may accompany

but usually follow the respiratory signs. Clonic spasms are seen in moribund birds. Laying flocks may have partial or complete cessation of production and not recover. Eggs from infected flocks may be abnormal in color, shape, or surface, and have watery albumen.

Mortality from either type of virus depends on virulence of the virus, species infected, environmental conditions, and the immune status of the flock. In general, mortality is higher in young flocks, but 100% mortality may occur in adult flocks as well.

Lesions: Lesions are highly variable, reflecting the variation in tropism and pathogenicity of the virus. Petechiae may be seen on the serous membranes; hemorrhages of the proventricular mucosa and intestinal serosa are accompanied by hemorrhagic and necrotic areas on the mucosal surface, especially at lymphoid foci such as cecal tonsils. Congestion and mucoid exudates may be seen in the respiratory tract, with opacity and thickening of the air sacs.

Diagnosis: Tentative diagnosis of a rapidly spreading, respiratory-nervous disease may be confirmed by isolation of the hemagglutinating virus identified by inhibition with Newcastle disease antiserum. A rise in hemagglutination-inhibition antibodies in paired serum samples also confirms the disease. The acute form should be differentiated from highly pathogenic avian influenza. QuickSearch Although it is difficult to differentiate between Newcastle strains other than by the rapidity with which they kill injected embryonating chicken eggs and adult chickens, grouping strains according to their geographic and temporal appearance using monoclonal antibodies has had some success. Oligonucleotide fingerprinting of the viral RNA has been used to differentiate between strains similar in all other respects.

Prevention and Treatment: Proper administration of a high-titered vaccine is essential for induction of a good immune response. Live virus vaccines are widely used. Lentogenic strains, chiefly B1 and LaSota in the New World, are administered in drinking water or in spray. Sometimes, administration is via the nares or eye. Healthy chicks are vaccinated as early as day 1-4 of life. However, delaying vaccination until the second or third week avoids interference with an active immune response by maternal antibody. Mycoplasma and some other bacteria, if present, may act synergistically with some vaccines to aggravate the vaccine reaction after spray administration. Failure to follow instructions (eg, use of sprays in open or windy houses, or use of chemically treated water to dilute the virus) may result in incomplete or no protection after vaccination.

When other infections are present in the flock, and where required by law, killed vaccines should be used. Killed vaccines with oil adjuvants give the longest protection. Whether killed vaccines, lentogenic mass vaccines, or IM-injected mesogenic strains are used, repeated vaccination is required to protect chickens throughout life. The frequency of revaccination largely depends on the risk of exposure and virulence of the field virus.

Disease control officials in the USA and some other countries use import restrictions and eradication methods to prevent establishment of the highly virulent, viscerotropic form of the disease; other countries depend on vaccination.

Zoonotic Risk: Newcastle disease virus can produce a transitory conjunctivitis in man, but the condition has been limited primarily to laboratory workers and vaccination teams exposed to large quantities of virus and, before vaccination was widely practiced, to crews eviscerating poultry in processing plants. The disease has not been reported in people who rear poultry or consume poultry products.

Poultry

INFECTIOUS LARYNGOTRACHEITIS

Review

Infectious laryngotracheitis is an acute, highly contagious, herpesvirus infection of chickens and pheasants characterized by severe dyspnea, coughing, and rales. It can also be a subacute disease with lacrimation, tracheitis, conjunctivitis, and mild rales. It has been reported from most areas of the USA in which poultry are intensively reared, as well as from many other countries.

Clinical Findings: In the acute form, gasping, coughing, rattling, and extension of the neck during inspiration are seen 5-12 days after natural exposure. Reduced productivity is a varying factor in laying flocks. Affected birds lose their appetite and become inactive. The mouth and beak may be bloodstained from the tracheal exudate. Mortality varies, but may reach 50% in adults, and is usually due to occlusion of the trachea by hemorrhage or exudate. Signs usually subside after ~2 wk, although some birds may cough for 1 mo. Strains of low virulence produce little or no mortality with slight respiratory signs and lesions and a slight decrease in egg production.

After recovery, some birds remain carriers for extended periods and become a source of infection for susceptible birds. The latent virus can be reactivated under stressful conditions. Infection also may be spread mechanically. Several epidemics have been traced to the transport of birds in contaminated crates.

Diagnosis: The acute disease is characterized by the clinical signs and finding blood, mucus, and yellow caseous exudate or a hollow caseous cast in the trachea. Picture Microscopically, a desquamative, necrotizing tracheitis is characteristic. In the subacute form, punctiform hemorrhagic areas in the trachea and larynx, and conjunctivitis with lacrimation permit a presumptive diagnosis. In uncomplicated cases, the air sacs usually are not involved. The diagnosis may be confirmed by demonstrating intranuclear inclusion bodies in the tracheal epithelium early in the course of the disease; by isolating and identifying the specific virus in chick embryos, tissue culture, or chickens; or by inoculating the infraorbital sinus or vent of known immune and susceptible birds. Developing chicken embryos (9-12 days old) are preferred for virus isolation. Chorioallantoic membrane of developing chicken embryos is inoculated with the specimen. Microscopical examination of the chorioallantoic membrane lesion shows intranuclear inclusions. Infectious laryngotracheitis must be differentiated from the diphtheritic form of fowlpox, QuickSearch especially with tracheal lesions. Fowlpox virus produces intracytoplasmic inclusions.

Field isolates and vaccine strains of infectious laryngotracheitis virus can be compared

by restriction endonuclease analysis of viral genomes. This method is useful for comparing closely related DNA genomes.

Nucleic acid probes prepared from cloned genomic fragments of infectious laryngotracheitis virus can also be used for diagnosis. This procedure is especially useful for differentiation of infectious laryngotracheitis from the diphtheritic form of fowlpox with tracheal lesions.

Polymerase chain reaction can be used to amplify laryngotracheitis virus genomic DNA sequences of various sizes using specific primers. This procedure is useful when an extremely small amount of viral DNA is present in the sample.

Prevention and Treatment: Some relief from signs is obtained by keeping the birds quiet, lowering the dust level, and using mild expectorants, being careful that they do not contaminate feed or water. Vaccination should be practiced in endemic areas and on farms where a specific diagnosis is made. Immediate vaccination of adults in the face of an outbreak shortens the course of the disease. Vaccination is best done with modified strains of low virulence applied to the conjunctiva (eye drop). Mass methods of vaccination such as spray or drinking water administration are less consistent in their results. Broiler flocks in some areas where the disease is endemic must be vaccinated when young, but this is unlikely to be effective if done at <4 wk of age. Some vaccine producers recommend revaccination when birds are to be held to maturity.

Poultry

INFECTIOUS BURSAL DISEASE

(Gumboro disease)

Review

Etiology and Transmission: Infectious bursal disease is caused by a birnavirus (IBDV) that is most readily isolated from the bursa of Fabricius but may be isolated from any organ. It is shed in the feces and transferred from house to house by fomites. It is very stable and difficult to eradicate from premises.

IBDV may be isolated in 8- to 11-day-old, antibody-free chicken embryos with inocula from birds in the early stages of disease. The chorioallantoic membrane is more sensitive to inoculation than is the allantoic sac. IBDV also may be isolated in cell cultures derived from the cloacal bursa and established cell lines, and some strains may be isolated in chicken-embryo fibroblasts. Cell-culture-adapted strains of IBDV produce a cytopathic effect, and such assays may be used for quantitative serologic tests. Two serotypes of IBDV have been identified, and antigenic variation between strains within a serotype is considerable. Serotype 2 infects chickens and turkeys but does not cause significant clinical disease or immunosuppression.

جVariantس strains of IBDV, which have major antigenic differences from the جstandardس strains, cause immunosuppression but not clinical disease in older chickens.

Clinical Findings: Infectious bursal disease is highly contagious; results of infection depend on age and breed of chicken and virulence of the virus. Infections may be subclinical or clinical. Infections before 3 wk of age are normally subclinical. Chickens are most susceptible to clinical disease at 3-6 wk, but severe infections have occurred in Leghorn chickens up to 18 wk old.

Early **subclinical infections** are the most important form of the disease because of economic losses. They cause severe, long-lasting immunosuppression due to destruction of immature lymphocytes in the bursa of Fabricius, thymus, and spleen. The humoral (B cell) immune response is most severely affected; the cell-mediated (T cell) immune response is affected to a lesser extent. Chickens immunosuppressed by early IBDV infections do not respond well to vaccination and are predisposed to infections with normally nonpathogenic viruses and bacteria. Common diseases are usually exacerbated by IBDV infections. Subclinical infections by the newer جvariantس strains occur in immature birds, and severe long-term immunosuppression and bursal atrophy result from early infections.

In **clinical infections**, onset of the disease is sudden after an incubation of 3-4 days. Chickens exhibit severe prostration, incoordination, watery diarrhea, soiled vent feathers, vent picking, and inflammation of the cloaca. Losses range to >20%. Recovery occurs in <1 wk, and broiler weight gain is delayed by 3-5 days. The presence of maternal antibody will modify the clinical course of the disease. Virulence of field strains of the virus varies considerably.

Lesions: At necropsy, the cloacal bursa is swollen, edematous, yellowish, and occasionally hemorrhagic, especially in birds that have died of the disease. Picture Congestion and hemorrhage of the pectoral, thigh, and leg muscles is common. Chickens recovered from IBDV infections have small, atrophied cloacal bursas due to the destruction and lack of regeneration of the bursal follicles.

Control: There is no treatment. Depopulation and rigorous disinfection of contaminated farms have achieved limited success. Live vaccines of chick-embryo or cell-culture origin and of varying virulence can be administered by eye drop, drinking water, or SC routes at 1-21 days of age. The immune response can be altered by maternal antibody, and the more virulent vaccine strains can override higher levels of antibody.

High levels of maternal antibody during early brooding of chicks in broiler flocks (and in some commercial layer operations) would minimize early infection or subsequent immunosuppression, or both. Breeder flocks should be vaccinated one or more times during the growing period, first with a live vaccine and again just before egg production with an oil-adjuvanted, inactivated vaccine. Inactivated vaccines of chick-embryo, bursa, or cell-culture origin are available. The latter vaccines induce higher and more uniform levels of antibody, which persist longer, than do live vaccines. The immune status of breeder flocks should be monitored periodically with a quantitative serologic test such as virus neutralization or ELISA. If antibody levels fall, hens should be revaccinated to maintain adequate immunity in the progeny.

1c

Poultry

FOWLPOX

Fowlpox in Chickens and Turkeys

Fowlpox is a slow-spreading viral infection of chickens and turkeys characterized by proliferative lesions in the skin (cutaneous form) that progress to thick scabs and by lesions in the upper GI and respiratory tracts (diphtheritic form). It is seen worldwide.

Etiology and Epidemiology: The large DNA virus (an avipoxvirus, family Poxviridae) is highly resistant and may survive for several years in dried scabs. Field and vaccine strains have only minor differences in their genomic profiles, although the strains can be differentiated to some extent by restriction endonuclease analysis and immunoblotting. The virus is present in large numbers in the lesions and is usually transmitted by contact to penmates through abrasions of the skin. Mosquitoes and other biting insects may serve as mechanical vectors. Transmission within flocks is rapid when mosquitoes are plentiful. Some affected birds may become carriers, and the disease may be reactivated by stress (eg, moulting) or by immunosuppression due to other infections.

Clinical Findings: Only a few birds develop lesions at one time. Lesions are prominent in some birds and may significantly decrease flock performance. Picture Picture Picture The cutaneous form is characterized by nodular lesions on various parts of the unfeathered skin of the chicken and on the head and upper neck of the turkey. Generalized lesions of the feathered skin may also occur. In some cases, lesions are limited chiefly to the feet and legs. The lesion is initially a raised, blanched, nodular area that enlarges, becomes yellowish, and progresses to a thick, dark scab. Multiple lesions usually develop and often coalesce. Lesions in various stages of development may be found on the same bird. Localization around the nostrils may cause nasal discharge. Cutaneous lesions on the eyelids may cause complete closure of one or both eyes.

In the diphtheritic form, lesions occur on the mucous membranes of the mouth, esophagus, pharynx, larynx, and trachea (wetpox or fowl diphtheria). Occasionally, lesions occur almost exclusively in one or more of these sites. Caseous patches firmly adherent to the mucosa of the larynx and mouth or proliferative masses may develop. Mouth lesions interfere with feeding. Tracheal lesions cause difficulty in respiration and may simulate infectious laryngotracheitis QuickSearch in chickens. Laryngeal and tracheal lesions in chickens must be differentiated from those of laryngotracheitis.

Often, the course of the disease in a flock is protracted. Extensive infection in a layer flock results in decreased egg production. Cutaneous infections alone ordinarily cause

low or moderate mortality, and these flocks generally return to normal production after recovery. Mortality is usually high in the generalized or diphtheritic form.

Diagnosis: Cutaneous infections usually produce characteristic gross and microscopic lesions. When only small lesions are present, it is often difficult to distinguish them from abrasions caused by fighting. Microscopical examination of affected tissues stained with H&E reveals eosinophilic cytoplasmic inclusion bodies. Cytoplasmic inclusions are also detectable by fluoresent antibody and immunoperoxidase methods. Elementary bodies can be detected in smears from the lesions stained by the Gimenez method. Viral particles with typical poxvirus morphology can be demonstrated by negative-staining electron microscopy as well as in ultrathin sections of the lesions. The virus can be isolated by inoculating chorioallantoic membrane of developing chicken embryos, susceptible birds, or cell cultures of avian origin. Chicken embryos (9-12 days old) are the preferred and most convenient host for virus isolation.

Field isolates and vaccine strains of fowlpox virus can be compared by restriction endonuclease analysis of viral genomes. This method is useful for comparing closely related DNA genomes.

Nucleic acid probes derived from cloned genomic fragments of fowlpox virus can also be used for diagnosis. This procedure is especially useful for differentiation of the diphtheritic form of fowlpox (involving trachea) from infectious laryngotracheitis.

Polymerase chain reaction can be used to amplify genomic DNA sequences of various sizes using specific primers. This procedure is useful when an extremely small amount of viral DNA is present in the sample.

Prevention and Treatment: Where pox is prevalent, chickens and turkeys should be vaccinated with live-embryo or cell-culture-propagated virus. The most widely used vaccines are attenuated fowlpox virus and pigeonpox virus isolates of high immunogenicity and low pathogenicity. A turkeypox vaccine has been developed to control pox in turkey flocks in which fowlpox vaccine has been ineffective. This virus appears to be immunologically unrelated to fowlpox virus. In high-risk areas, vaccination with an attenuated vaccine of cell-culture origin in the first few weeks of life and revaccination at 12-16 wk is often sufficient. Health of birds, extent of exposure, and type of operation determine the timings of vaccinations. Because the infection spreads slowly, vaccination is often useful in limiting spread in affected flocks if administered when <20% of the birds have lesions. Because passive immunity may interfere with multiplication of vaccine virus, progeny from recently vaccinated or recently infected flocks should be vaccinated only after passive immunity has declined. Vaccinated birds should be examined 1 wk later for swelling and scab formation ﺟtakeﺱ at the site of vaccination. Absence of ﺟtakeﺱ indicates lack of potency of vaccine, passive immunity, or improper vaccination. Revaccination with another serial lot of vaccine is indicated.

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Naturally infected or vaccinated birds develop humoral as well as cell-mediated immune responses. Humoral immune responses can be measured by ELISA or virus neutralization tests.

Poultry

INCLUSION BODY HEPATITIS

Review

Inclusion body hepatitis (IBH) is an acute disease of young chickens associated with anemia and hemorrhagic disorders. A similar disease has been reported in quail with quail bronchitis QuickSearch caused by an avian adenovirus. Once considered common, IBH is now rarely diagnosed, but it has been seen in many areas of the world.

Etiology, Transmission, and Pathogenesis: Adenoviruses have been considered to be the cause. A birnavirus (the cause of infectious bursal disease [IBD]) QuickSearch and a circovirus (the chicken anemia virus [CAV]) QuickSearch have been associated with adenovirus infections; both of these cause immunosuppression and contribute greatly to the severity of disease caused by adenoviruses.

Adenoviruses are ubiquitous and are transmitted horizontally and vertically. Infections are common and widespread in chickens. Chicks and young chickens are affected most commonly. Infection by adenovirus usually results in minimal hepatic disease; however, if birds infected with the IBD virus or CAV are infected with adenoviruses, clinical disease becomes evident.

Clinical Findings, Lesions, and Diagnosis: Sudden mortality usually is seen in chickens <6 wk old. Mortality is seldom >7%. Signs associated with diseases caused by other pathogens (eg, bacteria, fungi, or viruses) commonly occur if birds are immunosuppressed. In these cases, mortality rates may be >30%. Hemorrhage may occur in any organ. The bursa of Fabricius usually is small. Bone marrow, livers, and other organs usually are pale. Congestion and multifocal areas of light or dark discoloration are seen in livers. Microscopically, solitary prominent basophilic inclusion bodies are seen in hepatocyte nuclei. Picture

A tentative diagnosis is based on typical microscopical findings and confirmed by isolating adenoviruses from portions of liver. Immunosuppression resulting from infection with the IBD virus or CAV usually is the predisposing event that culminates in clinical IBH.

Treatment and Prevention: Treatment consists of nursing care. Antibiotics may help prevent secondary bacterial infections. Sulfonamides are contraindicated if evidence of hematologic disease or immunosuppression is seen.

Broiler-breeder flocks should have high levels of IBD antibody before they begin to lay fertile eggs. Vaccines against IBH or CAV are not commercially available in the USA.

In Australia, a peracute form of IBH has occurred in broiler chickens in the absence of infections with either the IBD virus or CAV. A live IBH vaccine is effective in preventing IBH in progeny when administered PO or in drinking water to breeder chickens several weeks before the onset of lay. In Pakistan, IBH is an essential part of the hydropericardium syndrome of broiler chickens in which an excessive amount of clear, watery or jelly-like fluid accumulates in the pericardial sac of chickens 3-6 wk old. A formalized vaccine prepared from the homogenated livers of experimentally infected chickens is 80-90% effective in preventing the hydropericardium syndrome when administered SC to chicks 10-12 days old.

Poultry

INFLUENZA

(Fowl plague)

Review

Etiology and Epidemiology: The causal orthomyxoviruses are type A influenza viruses. Both virulent and avirulent viruses with any of 14 known surface hemagglutinins infect avian species. The viruses grow readily in embryonating chicken eggs and agglutinate RBC. Specific hemagglutination inhibition is the basis for the serologic test for influenza antibodies. The viruses are distributed worldwide and recovered frequently from clinically normal sea birds, migrating waterfowl, imported pet birds, ratites, and species in live-bird markets.

Clinical Findings and Lesions: The incubation period is highly variable and ranges from a few days to 1 wk. Signs range from only a slight decrease in egg production or fertility to a fulminating infection with CNS involvement, but respiratory signs are most common. Other common signs in severely affected birds are greenish diarrhea; cyanosis and edema of the head, comb, and wattle; discoloration of the shanks and feet due to ecchymoses; and blood-tinged oral and nasal discharges. Sinusitis is not uncommon in ducks, quail, and turkeys.

The location and severity of gross lesions are also highly variable and may consist of hemorrhages, transudation, and necrosis in the respiratory, GI, integumentary, and urogenital systems. Picture Picture

Diagnosis: Isolation of the virus in embryonating chicken eggs results in allantoic fluid that agglutinates RBC. The hemagglutination is not inhibited by Newcastle disease antiserum. A crude antigen prepared by grinding the chorioallantoic membrane of infected chicken embryos gives positive results with a gel-precipitation test using known positive influenza A antiserum. If severe, the disease resembles acute fowl cholera QuickSearch and velogenic, viscerotropic Newcastle disease. QuickSearch In milder forms, it may be confused with other respiratory diseases.

Prevention and Treatment: The use of nonviable oil-emulsion vaccine is complicated by the 14 antigenically distinct hemagglutinin subtypes that may be responsible for the disease. To be effective, the vaccine must be produced using the autogenous virus or a virus of the same hemagglutinin type. The other major viral surface antigen, neuraminidase, is not as important in influenza immunity as the hemagglutinin (but is useful for identification). Treating affected flocks with broad-spectrum antibiotics to control secondary bacterial invaders and increasing house temperatures may help

reduce mortality. Amantadine hydrochloride, approved for treatment of influenza A in man, reduces the severity of influenza in some avian species, but amantadine-resistant virus frequently emerges. Suspected outbreaks should be reported to regulatory authorities.

Poultry

AVIAN PARAMYXOVIRUS INFECTIONS

Avian Paramyxovirus Type 3

Uncomplicated infections of laying turkeys with avian paramyxovirus type 3 (PMV-3) may cause mild respiratory disease and a severe decrease in egg production.

Etiology and Epidemiology: There are nine recognized serotypes of avian paramyxoviruses (PMV-1 to PMV-9). Newcastle disease virus (PMV-1) QuickSearch is the most important pathogen for poultry, but viruses of PMV-2 and PMV-3 may cause disease in infected poultry. PMV-3 viruses have been associated with respiratory disease and egg production problems in turkeys, but there have been no recorded natural infections in chickens, although experimental infections have demonstrated their susceptibility.

Turkeys and caged exotic birds appear to be the predominant sources of PMV-3 isolates. In caged birds, psittacine species seem to be the primary host, although PMV-3 viruses will spread to passerines. Isolates of PMV-3 from turkeys and exotic caged birds have been differentiated using monoclonal antibodies, and it seems unlikely that there is any etiologic link between infections of the two groups of birds. There is no report of isolation of PMV-3 virus from feral birds. The method of transmission between turkeys is unclear, and spread within a flock is usually slow. PMV-3 infections of turkeys have been reported in North America and Europe.

Clinical Findings: In uncomplicated infections of turkeys, the first sign is often a drop in egg production. However, early mild respiratory signs have been reported, which suggests the respiratory tract may be the initial site of infection. The drop in egg production varies considerably with the age of the birds and secondary infections. In complicated infections of birds a few weeks into lay, the effective loss may be ~1-2 eggs/bird/wk for 5-6 wk, after which production returns to expected levels. Production problems are associated with a high level of white-shelled eggs, and the hatchability and fertility of eggs are also reduced. Infection at, or just before, the point of lay may result in more serious losses, with the flock failing to reach target production throughout the laying period. Far more serious respiratory disease and egg production problems have been recorded when dual infection with Newcastle disease virus (including live vaccines), influenza viruses, chlamydiae, or mycoplasmas or other bacteria has occurred. There have been no reported studies on the lesions associated with PMV-3 infections of turkeys.

Diagnosis: Most diagnoses have been by clinical signs confirmed by serology. Antibodies to PMV-3 may be detected by hemagglutination inhibition tests. PMV-1

(Newcastle disease virus) and PMV-3 viruses cross-react in hemagglutination inhibition tests (and in other serologic tests such as ELISA), which causes problems in vaccinated birds. Birds vaccinated against Newcastle disease show a rise in hemagglutination inhibition titers to both viruses if subsequently infected with PMV-3 virus. PMV-3 viruses can be isolated from tracheal or fecal swabs or tissue samples from infected birds by inoculation of 8- to 10-day-old embryonating chicken eggs via the allantoic cavity. The PMV-3 virus can be confirmed by hemagglutination inhibition tests with specific antiserum; proper controls are important because of the cross-reaction with Newcastle disease viruses. PMV-3 viruses are not readily isolated from infected turkeys, which suggests the excretion period may be limited.

Prevention and Control: PMV-3 viruses appear to spread slowly. Good hygiene, including careful disinfection and time between restocking, has not always prevented infection in subsequent flocks. Inactivated, oil-emulsion vaccines are available in the USA and in the UK and other European countries for use in turkey breeding flocks. These are injected twice, 4 wk apart, before the birds begin to lay (usually when 20-24 wk old).

Poultry

SALMONELLOSES

Pullorum Disease

Infections with *Salmonella pullorum* usually cause high mortality in young chickens and turkeys and occasionally in adult chickens. Once common, pullorum disease has been eradicated from most commercial stock. It usually occurs in other avian species only if they are in close contact with infected chickens or turkeys. Infection in mammals is rare.

Transmission is chiefly directly through the egg but also occurs by direct or indirect contact. Infection transmitted via egg or hatchery usually results in death during the first few days of life and up to 2-3 wk of age. Affected birds huddle near a source of heat, do not eat, appear sleepy, and show whitish fecal pasting around the vent. Survivors frequently become asymptomatic carriers with localized infection of the ovary. Some of the eggs laid by such hens hatch and produce infected progeny.

Lesions in young birds usually include unabsorbed yolk sacs; focal necrosis of the liver and spleen; and grayish nodules in the lungs, heart, and gizzard. Firm, cheesy material in the ceca and raised plaques in the mucosa of the lower intestine are sometimes seen. Occasionally, synovitis is prominent. Adult carriers usually have pericarditis, peritonitis, or distorted ovarian follicles with coagulated contents; however, sometimes no gross lesions are seen. In mature chickens, acute infections produce lesions that are indistinguishable from those of fowl typhoid. QuickSearch

Lesions may be highly suggestive, but diagnosis should be confirmed by isolation and identification of *S pullorum*, which is readily isolated by direct plating on most nonselective, aerobic, solid media. Infections in mature birds can be identified by serologic tests, followed by necropsy and culturing for confirmation.

No antibacterials are currently approved by the FDA for treatment of infected flocks. Control is based on routine testing of breeding stock to assure freedom from infection. Chickens are tested by a tube-agglutination or whole-blood method. The latter method is not dependable for testing turkeys, and either a tube-agglutination or serum-plate test is used. Variant or polyvalent antigens are sometimes necessary.

CC

Poultry

SALMONELLOSES

Paratyphoid Infections

Paratyphoid infections can be caused by any one of the many nonhost-adapted salmonellae. Several species can infect a bird or flock concurrently.

Etiology, Clinical Findings, and Lesions: *Salmonella typhimurium* is the most common cause, followed by *S enteritidis* and *S heidelberg*, but the prevalence of other species varies widely by geographic location and strain of bird. In the USA, most infections are produced by 10-20 species; some species or strains are more pathogenic than others. All birds may be susceptible, and infections are common in all species of domestic birds. Usually, the incidence is higher in young flocks. The public health significance of the infections warrants serious attention to control.

Infections are often subclinical. Mortality is usually limited to the first few weeks of age and is higher in ducks and turkeys than in chickens. Shipping, delayed feeding, chilling, or overheating increases mortality. Depression, poor growth, weakness, diarrhea, and dehydration may be seen. The clinical signs are not distinctive. Phage type 4 *S enteritidis* is widespread in parts of Europe and may cause mortality of up to 20% in the first 3 wk of life. This serotype and some of its strains may cause a substantial incidence of infection of the reproductive tract in hens, with true vertical transmission and important public health implications.

Often, there are no lesions, but there may be an enlarged liver with or without areas of focal necrosis, unabsorbed yolk sac with coagulation, and cecal cores. Infections occasionally localize in the eye or synovial tissues.

Diagnosis: Isolation and identification of the causal agent is essential. Direct culture from liver and yolk sac onto almost any standard type of aerobic medium is adequate for isolation. Either a selenite or tetrathionate enrichment broth transferred in 24-48 hr to brilliant green agar or other media may be used to isolate the organism from intestinal or environmental samples.

Treatment and Control: Several antibacterial agents help prevent mortality but cannot eliminate flock infection. Turkeys, in particular, are generally injected with one or more antibiotics (gentamicin and streptomycin) after hatching.

Dependable control methods have not been developed. Strict sanitation in all hatching processes helps prevent transmission between successive lots of birds in a house. Early fumigation of hatching eggs is recommended to prevent penetration of the shell

surface by salmonellae. Washing should be undertaken only under strictly controlled conditions. Infection resulting from true egg transmission is rare, and no method has been devised to destroy the pathogens in the egg.

The heat of pelleting is reasonably effective in destroying salmonellae in feed ingredients. Maintenance of poultry in confinement and exclusion of all pets, wild birds, and rodents help prevent introduction of infection. The water source should be free of contamination. Early colonizing of the gut with selected normal microflora results in significant resistance to subsequent exposure.

Several methods of determining the *Salmonella* status of breeding flocks have been devised. Periodic culturing of environmental samples from litter, dust, water, hatchery debris, and cull chicks can be reasonably accurate for detecting infection. Serology is not highly dependable but has been of value in detecting *S typhimurium* infection in turkey flocks.

Poultry

TUBERCULOSIS

Review

Tuberculosis is a slowly spreading, chronic, granulomatous bacterial infection, characterized by gradual weight loss. All birds appear to be susceptible, although to variable degrees; pheasants seem to be highly susceptible, while the disease is uncommon in turkeys. Tuberculosis is more prevalent in captive than in wild birds. Tuberculosis is unlikely to occur in commercial poultry due to the short life span and husbandry practices used. (*See also* TUBERCULOSIS IN MAMMALS.) QuickSearch

Etiology and Epidemiology: *Mycobacterium avium* serovars 1 and 2 are the usual cause, although *M tuberculosis* has been isolated from parrots and canaries. *Mycobacterium avium* is very resistant; it can survive in soil for up to 4 yr, in 3% hydrochloric acid for ≥2 hr, and in 4% sodium hydroxide for ≥30 min. Tuberculosis occurs worldwide, most commonly in small, barnyard flocks and in zoo aviaries; it is rarely found in young flocks. Wild birds, such as cranes, sparrows, starlings, and raptors, have been found infected. Tuberculosis has been found in emus and also occurs in other ratites.

Infected birds with advanced lesions excrete the organism in their feces. Cadavers and offal may infect predators and cannibalistic flockmates. Rabbits, pigs, and mink are readily infected. Cattle exposed to contaminated feces may respond to mammalian tuberculin and to johnin. *Mycobacterium avium* may cause disease in man; serovar 1, often isolated from tuberculous chickens, has been isolated from people with acquired immunodeficiency syndrome.

Clinical Findings and Diagnosis: Signs usually do not develop until late in the infection when birds become thin and sluggish, and lameness may be seen. In chickens, granulomatous nodules of varying size are usually found in the liver, spleen, bone marrow, and intestine. Some exotic species may have lesions in the liver and spleen without intestinal involvement, but bone marrow and small mesenteric nodules may be found. Lesions are not calcified.

Live birds may be tested with avian tuberculins, although these are of little value in birds that do not have wattles. Large numbers of acid-fast bacteria in smears from lesions provide a tentative diagnosis.

Control: Chemotherapy is ineffective. In commercial poultry flocks, relatively rapid turnover of populations, together with improved general sanitation, has largely eliminated this once common infection. Infected poultry should be destroyed, and

housing facilities thoroughly cleaned and disinfected using cresylic compounds. Dirt-floored houses should have several inches of the floor removed and replaced with dirt from a place where poultry have not been maintained. All openings should be screened against wild birds. Avian tuberculosis in zoos is difficult to eradicate. New additions to the aviary should be quarantined for 2-3 mo. The movement of ratites through sales and the long life of these animals have made tuberculosis a major concern for ratite producers. Isolation of ratites purchased at sales is essential to prevent the introduction of tuberculosis into established flocks.

Poultry

*COLI*BACILLOSIS

(*Coli*septicemia, *Escherichia coli* infection)

Review

Colibacillosis occurs as an acute fatal septicemia or subacute pericarditis and airsacculitis. It is a common systemic disease of economic importance in poultry and is seen worldwide.

Etiology and Pathogenesis: *Escherichia coli* is a gram-negative, rod-shaped bacterium normally found in the intestines of poultry and most other animals; although most are nonpathogenic, a limited number produce extraintestinal infections. Pathogenic strains are most commonly of the 02, 078, and 01 serotypes, but a large number of others also produce disease. Virulence factors include the ability to resist phagocytosis, utilization of highly efficient iron acquisition systems, resistance to killing by serum, production of colicins, and adherence to respiratory epithelium.

Large numbers of *E coli* are maintained in the poultry house environment through fecal contamination. Initial exposure to pathogenic *E coli* may occur in the hatchery from infected or contaminated eggs, but systemic infection usually requires predisposing environmental or infectious causes. Mycoplasmosis, infectious bronchitis, Newcastle disease, hemorrhagic enteritis, and turkey bordetellosis are often complicated by colibacillosis. Poor air quality and other environmental stresses may also predispose to *E coli* infections.

Systemic infection occurs when large numbers of pathogenic *E coli* gain access to the bloodstream from the respiratory tract or intestine. Bacteremia progresses to septicemia and death, or the infection extends to serosal surfaces, pericardium, joints, and other organs.

Clinical Findings and Lesions: Signs are nonspecific and vary with age, organs involved, and concurrent diseases. Young birds dying of acute septicemia have few lesions except for enlarged, hyperemic liver and spleen with increased fluid in body cavities. Birds that survive septicemia develop subacute fibrinopurulent airsacculitis, pericarditis, perihepatitis, and lymphocytic depletion of the bursa and thymus. (Unusually pathogenic salmonellae produce similar lesions in chicks.) Although airsacculitis is a classic lesion of colibacillosis, whether it results from primary respiratory exposure or from extension of serositis is unclear. Sporadic lesions include pneumonia, arthritis, osteomyelitis, and salpingitis.

Diagnosis: Isolation of a pure culture of *E coli* from heart blood, liver, or typical visceral lesions in a fresh carcass indicates primary or secondary colibacillosis. Consideration should be given to predisposing infections and environmental factors. Pathogenicity of isolates is established when parenteral inoculation of young chicks or poults results in fatal septicemia or typical lesion within 3 days.

Control: Treatment strategies include attempts to control predisposing infections or environmental factors and early use of antibacterials indicated by susceptibility tests. Commercial bacterins, administered to breeder hens or chicks, have provided some protection against homologous *E coli* serotypes.

Poultry

ULCERATIVE ENTERITIS

(Quail disease)

Review

Ulcerative enteritis is an acute or chronic enteritis seen primarily in bobwhite quail (*Colinus virginianus*) but often seen in chickens 5-7 wk old and also reported in young turkeys, pheasants, grouse, and other gallinaceous birds. It occurs worldwide.

Etiology, Epidemiology, and Pathogenesis: *Clostridium colinum* is a fastidious, spore-forming, anaerobic rod, ~1 × 3-4 μm, that is difficult to culture. The spores are oval and subterminal.

The organism is shed in the feces of infected birds. Quail and chickens that develop chronic disease remain carriers. To induce experimental infection in quail, ≥10↑6 organisms are given PO; chickens require ≥10↑9 organisms. Outbreaks of ulcerative enteritis in chickens may follow outbreaks of coccidiosis, infectious bursal disease, and inclusion body hepatitis. After oral infection, the organism produces enteritis and ulcers in the lower third of the intestinal tract. Some organisms may pass to the liver via the portal circulation and produce diffuse or focal liver necrosis. Although large numbers of bacteria resembling *C colinum* can be seen in the ulcerative lesions of the gut and necrotizing lesions of the liver, histologic features of these lesions suggest that, in addition to bacterial invasion, a toxin also may be involved in the pathogenesis of this disease. However, no toxin has yet been identified.

Clinical Findings: In susceptible quail, the disease is acute, and mortality may be 100% in a few days. In chickens, signs are usually less dramatic, and mortality is ≤10% during the clinical course of the disease (≥2-3 wk). Some affected quail or chickens may die without obvious signs of disease or weight loss. Infected birds discharge characteristic droppings that are streaked with urates surrounded by a watery ring. Chronically affected birds are listless and anorectic; they appear humped-up, with the neck retracted and eyes partially closed.

Lesions: The primary lesions are found in the lower third of the small intestine, ceca, and liver. Lesions in the intestine and ceca vary from punctate hemorrhages to ulceration. The well-defined ulcers vary in size and may be 5 mm in diameter. The larger ulcers may have yellow, diphtheritic membranes with a depressed center and raised edges. Perforating ulcers are frequent and cause local or diffuse peritonitis. Liver lesions appear as isolated, yellow, necrotic foci or irregularly shaped, yellow, necrotic areas in the parenchyma. The only other organ that may show lesions is the

spleen, which may be enlarged and either hemorrhagic or necrotic.

Diagnosis: Although histomoniasis QuickSearch and inclusion body hepatitis QuickSearch may superficially resemble ulcerative enteritis, coccidiosis QuickSearch causes the greatest problem in differential diagnosis. Often, both infections occur simultaneously. In ulcerative enteritis, the spore-forming rods that resemble *C colinum* can be demonstrated in gram-stained blood, liver, and spleen of septicemic birds. *Clostridium colinum* can be isolated from infected livers and spleens cultured under strict anaerobic conditions on pre-reduced, anaerobic blood agar plates.

Treatment and Control: Bacitracin and streptomycin are the most effective drugs. Bacitracin is used in the feed at 0.005-0.01%, streptomycin at 0.006%. Either drug can be given in the drinking water prophylactically or therapeutically. The tetracyclines and furazolidone at 0.02% in the feed are also effective.

Medicated flocks often have little resistance against reinfection. Therefore, contaminated litter should be removed, and treatment may need to be continued or repeated periodically. Because bobwhite quail are highly susceptible, they should be raised on wire or slat floors as a preventive measure.

Poultry

AVIAN SPIROCHETOSIS

(Avian borreliosis)

Review

Avian spirochetosis is an acute, febrile, septicemic, bacterial disease that affects a wide variety of birds.

Etiology, Epidemiology, and Transmission: The causal organism, *Borrelia anserina*, is an actively motile spirochete, ~0.2-0.3 μm x 8-20 μm and consists of 5-8 loosely arranged coils. No reliable data are available concerning in vitro cultivation. It can be propagated in embryonating duck or chick embryos or in young ducks or chicks.

Spirochetosis is found worldwide, but generally in temperate or tropical regions, wherever the biologic vectors are found. The notable worldwide vector is *Argas (Persicargas) persicus*, the "cosmopolitan" fowl tick, but other *Argas* spp transmit the disease in different geographic areas. In the western USA, a highly efficient vector is *A sanchezi*.

Diverse immunologic and serologic types of *B anserina* have been demonstrated in many areas. Recovery from one type confers solid immunity against the homologous types for ≥1 yr, but none against heterologous strains. Relapses, such as occur with some human *Borrelia* infections, are unknown in *B anserina* infection of birds; any reinfection can be attributed to a heterologous type.

Generally, an infected *Argas* tick can transmit the disease at every feeding and maintains the infection throughout larval, nymphal, and adult stages. The ticks also transmit the infection transovarially, ie, the F↓1 larvae are infective. Ticks remain infected with *Borrelia* despite feeding on chicks hyperimmune to *B anserina* or on chicks with high blood levels of chemotherapeutic agents effective against *Borrelia* (such as the penicillins). Other vectors (lice, mosquitoes, some species of ticks, inanimate objects) can transmit the spirochete mechanically to a susceptible host whenever the piercing apparatus becomes contaminated with blood that contains *Borrelia*. Ingestion of bile-stained fecal droppings containing the spirochete, as well as acts of cannibalism during spirochetemia can result in infection. After the bite of an infected tick, the incubation period is ~4-7 days.

Clinical Findings: Signs are highly variable, depending on the virulence of the spirochete, and thus are not pathognomonic. They include listlessness, depression, somnolence, moderate to marked shivering, and increased thirst. Young birds are

affected more severely than older ones. During the initial stages of the disease, there is usually a greenish yellow diarrhea with increased urates. The course of the disease is 1-2 wk. Mild strains are not unusual. However, in many tick-infested geographic areas, morbidity can approach 100%, and mortality >90% has been recorded.

Lesions: An enlarged spleen with petechial or ecchymotic hemorrhages is the most notable gross lesion. This mottled or marbled appearance is not unlike spleens in marble spleen disease of pheasants. QuickSearch However, a contrasting situation may be seen in Mongolian pheasants, in which the spleen is reported to be small and pale. Occasionally, the liver may be swollen and contain focal areas of necrosis. Kidneys may be enlarged and pale. A green, catarrhal enteritis is common.

Diagnosis: Diagnosis rests on demonstration of *Borrelia* in the blood, either as actively motile *Borrelia* during darkfield microscopy, or as stained spirochetes in Giemsa-stained blood smears. In young birds, the *Borrelia* may reach vast numbers per oil-immersion field and persist for several days. Older birds usually have low numbers of *Borrelia* that are detected only with difficulty, or not at all, and that persist for only 1-2 days. Anemia is common and results in increased numbers of immature RBC.

Agar-gel diffusion and various serologic tests have been described but are of questionable value due to diverse serotypes that exist in some localities. Specific agglutinins clump the spirochetes in successively larger clumps during the terminal stages of the disease. Agglutination-lysis then begins to disintegrate these clumps, and spirochetal degradation products are liberated, which may result in pyrexia. Death occurs most often 1-3 days after *Borrelia* disappear from the bloodstream.

Treatment and Control: Several chemotherapeutic agents are effective. The most widely used are penicillin derivatives, but the streptomycins and tetracyclines are also effective. The antibiotics can be completely efficacious if begun when the number of spirochetes per oil-immersion field is low or moderate; however, if large numbers of spirochetes are present in the bloodstream when chemotherapy is begun, the sudden liberation of large quantities of spirochetal degradation products can result in more deaths than no treatment.

Control must first be directed against the biologic vector. *Argas* ticks are notable for their long life span, ability to survive for extended periods without a blood meal, efficiency in transmitting the spirochete, and an ability to remain securely hidden in cracks and crevices often beyond the effective reach of pesticides. Accordingly, control is difficult. A combination of tick eradication and immunization offers the most effective means of control.

Immunization can be highly successful and, next to eradication of the biologic vector, is the preferred method of control. Bacterins prepared from infective blood have been used with success. The most widely used bacterins are egg-propagated products

composed of yolk material containing the spirochetes, but whole-egg propagated bacterins have been used successfully; usually one or two IM injections suffice. Formalin (0.2%) is usually used to inactivate the spirochetes. The appropriate serotype(s) of the spirochete in any given locality must be used. Little if any cross-protection is afforded to different serotypes.

Poultry

ASPERGILLOSIS

(Brooder pneumonia, Mycotic pneumonia, Pneumomycosis)

Review

Aspergillosis is a disease, usually of the respiratory system, of chickens, turkeys, and less frequently ducklings, pigeons, canaries, geese, and many other wild and pet birds. In chickens and turkeys, the disease may be endemic on some farms; in wild birds, it appears to be sporadic, frequently affecting only an individual bird. It is usually seen in birds 7-40 days old. (*See also* ASPERGILLOSIS IN MAMMALS and CAGED BIRDS.) QuickSearch

Etiology and Epidemiology: *Aspergillus fumigatus* is a cause of the disease. However, several other *Aspergillus* spp, as well as other genera, eg, *Penicillium*, may be incriminated.

Chicks and poults may become infected during hatching as a result of inhaling large numbers of spores in heavily contaminated hatching machines or from contaminated litter. In older birds, infection is caused primarily by inhalation of spore-laden dust from contaminated litter or feed or dusty range areas.

Clinical Findings and Lesions: Dyspnea, hyperpnea, somnolence and other signs of nervous system involvement, inappetence, emaciation, and increased thirst may be seen. The encephalitic form is most common in turkeys. In chicks or poults up to 6 wk old, the lungs are most frequently involved. Pulmonary lesions are characterized by cream-colored plaques a few millimeters to several centimeters in diameter; occasionally, mycelial masses may be seen within the air passages on gross examination. Picture The plaques also may be found in the syrinx, air sacs, liver, intestines, and occasionally the brain. An ocular form, in which large plaques may be expressed from the medial canthus, has been seen in chickens and turkeys.

Diagnosis: The fungus can be demonstrated by culture or by microscopical examination of fresh preparations. One of the plaques is teased apart and placed on a suitable medium, usually resulting in a pure culture of the organism. Histopathologic examination using a special fungus stain reveals granulomas containing mycelia. Pathogenicity of the isolate is confirmed by injecting it into the air sacs of susceptible 3-wk-old chicks.

Differential diagnoses include infectious bronchitis, Newcastle disease, laryngotracheitis, and *Dactylaria* infection.

Control: Strict adherence to sanitation procedures in the hatchery minimizes early outbreaks. Grossly contaminated eggs should not be set for incubation because they may explode and disseminate spores throughout the hatching machine. Contaminated hatchers should be fumigated with formaldehyde or thiabendazole (120-360 g/m↑3). Avoiding moldy litter or ranges serves to prevent outbreaks in older birds. Pens should be sprayed with nystatin, and all equipment cleaned and disinfected.

Treatment of affected birds is considered useless.