AVIAN VIRAL DISEASES
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There are several primary viral diseases in pet birds, but there are also several viruses in which secondary organisms or factors help to create a multifactorial syndrome.

PSITTACINE CIRCOVIRUS (PSITTACINE BEAK AND FEATHER VIRUS, PBFD)

This single-stranded circular DNA virus has been recently placed in the Circoviridae along with the chick anemia agent virus and the porcine circovirus. Various species of white cockatoos and pink cockatoos (galahs, gang gangs, Major Mitchells) are most commonly infected, but there are reports of PBFD in the black palm cockatoo, lovebirds, budgerigars, Vasa parrots, Eclectus parrots, African Gray parrots, lories and lorikeets, Australian parakeets (*Psittacula, Platycercus, Neophema, Polytelis, Psephotus*), King parrots, *Pionus* parrots, a Jenday conure, a red-olated Amazon, a blue-front Amazon, and most recently a Scarlet macaw. Infection of wild Neotropical parrots (South and Central American parrots) has not been documented, and disease in captive Neotropicals is rare. The disease is associated with French molt in Australian budgerigars.

Psittacine circovirus causes severe immunosuppression and progressive feather dystrophy, and birds commonly die of secondary bacterial, chlamydial, fungal and viral infections. Two forms are observed: the acute neonatal form and the chronic adult form. The infection is found to be strongly age related with 92% of diagnosed birds being less than 3 years old. Juvenile susceptibility appears to be related to infection and destruction of the bursa.

**Acute Form:**
This form is observed in young or fledgling birds during their first feather formation after replacement of the neonatal down. Chicks as young as 3-12 weeks of age have been described with classic lesions. Peracute disease is suspected in neonatal cockatoos showing signs of septicemia, pneumonia, crop stasis, anorexia, enteritis, rapid weight loss and death. PBFD is also suspected as a cause of embryonic mortality. Acute infections in fledglings are characterized by sudden changes in developing feathers: necrosis, fractures, bending, hemorrhage into the feather shaft, or premature shedding. Diarrhea and depression are followed by death in 1-2 weeks. The acute fledgling form is particularly common in sulfur-crested cockatoos and lovebirds. There is also a syndrome in juvenile African Grey parrots that die of acute overwhelming bacterial sepsis prior to the development of any feather abnormalities, due to viral destruction of the bursa before it is able to seed the rest of the body with B cells.

**Chronic Form:**
Chronic PBFD is characterized by the progressive appearance of dystrophic feathers with each successive molt. The first signs of PBFD in cockatoos usually involve the powder down feathers and body contour feathers located over the lumbar area, with progression to secondary flight, tail and crest feathers, and finally primary flight and tail feathers. Other species, such as lovebirds, have substantial changes in the flight and tail feathers, as well as body contour feathers. Birds eventually become bald. Feather dystrophy is described grossly as retention of feather sheaths; hemorrhage within the pulp cavity; fractures of the rachis; short clubbed feathers; deformed, curled feathers; and circumferential constriction ("pinching") of the proximal feather shaft.
Beak lesions are also described but are much less common: elongated, overgrown beaks with transverse or longitudinal fractures; palatine necrosis; and oral ulceration. The outer surface of the beak is often black and shiny in affected white cockatoos because of the loss of the powder down feathers. Recurrent or chronic fungal, bacterial, viral or chlamydial infections are common in these immunosuppressed birds. The disease is generally considered fatal, with death ensuing 6-12 months after clinical onset. Apparent clinical recoveries with conversion to negative PCR status have been described in budgerigars, lorikeets and lovebirds, but the carrier status of these recovered birds is unknown.

PBFD virus infection is very common in lovebirds, with surveys documenting infection rates of 40 to 60% of submissions. Most are fairly asymptomatic infections. Infection with the microsporidian parasite, *Encephalitozoon hellem*, may be so common in lovebirds because of infection with psittacine circovirus.

“French molt” is a term that describes a common form of chronic pterylo-dysplasia in budgerigar flocks with complete or near-complete absence of primary and secondary wing feathers. The owners often referred to these birds as “runners” or “creepers” because their feather abnormalities do not allow them to fly and they run around on the floor of the aviary. Concurrent avian polyomavirus infection (APV) is also common in budgerigars with PBFD virus infection and APV can contribute to the feather abnormalities.

Infection with psittacine circovirus is relatively common in lories and lorikeets (nectar-eating psittacines). Infected when they are quite young, lorikeets develop the typical bursa lesions. One-third die before their first molt, another one-third develop chronic feather dystrophy, and the remaining one-third drop their feathers, but go on to molt and develop normal feathering. The circovirus strain that most commonly infects lories and lorikeets appears to be slightly different from the strain that typically infects other psittacine birds. It is unclear whether the apparent recovery and conversion to PCR negative status is attributable to the virus strain or to the innate immunity of lories and lorikeets.

**Transmission:**
Circovirus virions are found in feather follicle dust, feces and crop washings of infected birds. Inhalation and ingestion appear to be the most common routes of infections. High concentrations of the virus have been recovered from feather dust collected in a room housing infected birds. Experimentally, the minimum incubation period is 21-24 days. In the natural disease, the incubation period ranges from a few weeks in nestlings to 2 years or more in older birds. The time variance in the incubation period may be related to the differences in the concentration of maternally-derived antibodies, the titer of virus exposure and the host responses.

**Diagnosis:**
Historically, before the PCR DNA probe technology was developed, histopathology was best diagnostic method available. Major histologic changes are found in the feather shaft and are collectively termed pterylodysplasia. These changes are characterized as necrosis of epithelial cells in the epidermal basal, intermediate and collar zones of the developing feather; edema, hemorrhage and inflammation (heterophils, macrophages and/or plasmacytes depending on acute or chronic disease) of the feather pulp; and basophilic globular intracytoplasmic viral inclusions in epidermal cells and macrophages within the epithelium and pulp. Less commonly, intranuclear inclusions may be found. Necrosis, inflammation and inclusions are observed in affected rhamphotheca and adjacent dermis. Necrosis and inclusions are also reported in thymus, bursa and bone marrow, especially in young birds. When the bursa of juvenile African Grey parrots, dying acutely of overwhelming bacterial sepsis, is examined microscopically, typical circoviral inclusions are found, and there is profound bone marrow hypoplasia, especially of the granulocytes.
Best results for histologic diagnosis in the live bird depended significantly on selecting several grossly deformed blood feathers for histopathology. Viral inclusions could be difficult to demonstrate in early infections, therefore, repeated biopsy may have been necessary.

A much more sensitive method of diagnosis is the PCR DNA probe, which is capable of detecting infection prior to feather abnormalities. The psittacine circovirus DNA probe is available through the Infectious Diseases Lab at the University of Georgia, as well as other private laboratories, such as Veterinary Molecular Diagnostics, Inc. The probe detects viral nucleic acid within leukocytes in blood samples and in infected tissue swabs from necropsy (liver, spleen, bursa, kidney, heart blood). Feathers and other tissues preserved in 10% formalin may also be tested with an in situ DNA hybridization technique or by immunohistochemical staining, but this tends to be more difficult and more expensive than the PCR DNA probes.

The PCR DNA probe testing is an extremely important part of the pre-purchase and post-purchase examinations of both breeding birds and companion birds. It is also extremely important as part of the testing done during attempts to “test and remove” birds from infected aviary populations and during the quarantine of newly acquired birds, so that this disease does not enter the clean aviary. Many aviaries have been able to eradicate psittacine circovirus infection from the flocks. Unfortunately, the less expensive birds, such as lovebirds and budgerigars, often go untested, and remain as a reservoir for the virus. When lovebirds and budgerigars are mixed in the same air space as larger, more expensive psittacines, such as in a pet shop situation, transmission and infection can occur in the larger psittacines.

**Treatment:**
There is no definitive treatment. Supportive therapy would include good nutrition and husbandry, appropriate antibiotics and antifungals, as needed. Immunostimulants, such as levamisole, and autogenous bacterins have been used, as well, with variable success. Some birds, such as lories, may apparently recover from the infection and become DNA probe negative, although whether a recrudescence could occur is still questionable.

**Prevention:**
Research on a vaccine has been worked on at the University of Georgia, but is still probably years from commercial availability. Preventing exposure of juvenile birds to infected adults has been the only viable method of prevention at this time. Artificial incubation, hand rearing, strict isolation of juveniles and removal of infected adult birds have been involved in most eradication programs. Disinfection of the premises is also necessary because the virus is so resistant in the environment. This disinfection needs to include air ducts and incubators, followed by drag swabs for PCR to confirm that disinfection has removed the virus from the facility. Before new breeding birds, especially cockatoos, are added to an aviary they should have a DNA probe test performed during their aviary quarantine period, realizing that they may be in the long incubation period. The DNA probe seems to be capable of preventing introduction of infected birds into aviaries.

**Pigeon Circovirus:**
Viral inclusions consistent with a columbid circovirus infection in pigeon squabs have been described. The squabs are often dying of bacterial, fungal, and other viral infections, and fail to thrive. Histopathology of the bursa reveals the typical circovirus inclusions. PCR DNA probe testing can also be performed on pigeon blood, but the clinician needs to be sure that the specific columbid circovirus primers are being used, or that a more generic circovirus probe is used.
Canary Circovirus: 
A novel circovirus was sequenced from a flock of canaries experiencing high mortality, especially in 10- to 20-day-old chicks. Abdominal and gall bladder distension and airsacculitis were commonly seen in affected chicks. Typical intracytoplasmic, and sometimes intranuclear, circovirus inclusions were found in tissues on histopathology.
AVIAN POLYOMAVIRUS (APV):

Avian Polyomavirus (APV) is currently classified in the genus Polyomavirus of the family Papovaviridae. It is technically a polyomavirus, but one will see this virus called both polyomavirus and papovavirus in the literature. The polyomaviruses that infect birds appear morphologically and antigenically similar. Polyomaviral disease was first described in 1981 by Davis in budgerigar avaiaries, and was called Budgerigar Fledgling Disease. Since then, polyomavirus infections have been described in numerous species of psittacines (conures, macaws, Amazons, Pionus, cockatoos, Eclectus, caiques, Australian parakeets, and ringneck parakeets) and in several finch species (Lady Gouldian finches, waxbills, crimson finches, zebra finches, canaries, and black seed-crackers). African Grey parrots and cockatiels appear to be fairly resistant to clinical disease, but may still shed the virus for periods of time. Although most deaths due to polyomavirus occur in baby birds, adult Eclectus parrots, lovebirds and caiques appear susceptible to acute death due to polyomavirus as well. The adult Eclectus parrots and lovebirds are usually co-infected with psittacine circovirus.

Incubation Period:
The incubation period in the budgerigar is 10-21 days (average 14 days). The incubation period for the other psittacine species is largely unknown, but appears to vary according to the size of the bird. Smaller species, such as conures, die at younger ages and large species, such as macaws, die at older ages. Conures tend to die at 20-35 days of age, while Scarlet Macaws tend to die at 3-4 months of age. The large psittacine susceptibility range is roughly 20-140 days of age, with most deaths occurring between 20 and 60 days of age. Adult birds are thought to be relatively resistant to outward signs of disease, but may shed the virus for a period of time, usually 10 months or less, in the feces.

Clinical Signs:
The acute form in baby birds is characterized by crop stasis, vomiting, abdominal distension, lack of daily weight gain, depression, dehydration, anorexia, subcutaneous hemorrhages and death in a matter of hours. Mortality rates vary from 30-100% of susceptible young. The chronic form may occur in sub-lethal infections and is characterized by stunting; poor crop emptying and gut transit time; polyuria and renal failure; abnormal feathering; failure to wean; and secondary fungal and bacterial infections. Recovered budgies typically have poor feather formation and often are called "runners", because of their inability to fly. These recovered budgies remain carriers, shed virus intermittently, and are responsible for persistence of the virus in the aviary.

Diagnosis:
Gross necropsy may reveal hydropericardium, ascites, cardiomegaly, hepatomegaly, splenomegaly, swollen kidneys, subcutaneous hemorrhages and edema, and serosal and epicardial hemorrhages. Histopathology reveals acute hepatic and splenic necrosis. Large, clear to amphophilic, karyomegaly, intranuclear viral inclusions are most common in the vascular endothelium of the spleen in acute cases, but may also be observed in kidney, liver, heart, crop epithelium, bone marrow and feather follicle. In chronic cases in budgerigars, inclusions are most common in feather follicle and kidney. In other psittacines, feather inclusions are extremely rare. The virus is extremely difficult to isolate, requiring budgerigar fibroblast cell culture, so histopathology is the most common method of diagnosis.

The best confirmatory test at this time is the PCR DNA probe. This test detects virus shed in feces and oral secretions of live birds by using oral/cloacal swabs. It also is used to confirm the disease in swabs taken from fresh or frozen tissues at necropsy. The DNA probe is available from the Infectious Diseases Lab at University of Georgia and from other commercial laboratories. Polyomavirus antibody testing can also be
performed there. More testing information is available at U of Georgia’s website:  
http://www.vet.uga.edu/idl/. The U of Georgia’s lab also offers in situ DNA hybridization detection on formalin-fixed, paraffin-embedded tissues. One commercial laboratory that offers the PCR DNA probe testing is Veterinary Molecular Diagnostics; see their website at:  http://www.vmdlabs.com/.

To detect birds that are shedding virus, both blood and a combined oral and cloacal swab should be tested by PCR. Birds may no longer be viremic and may be negative on the blood test, but positive on the oral/cloacal test because virus shedding from the GI tract persists for a longer period time.

Infected birds, both clinically ill and asymptomatic, raise a detectable virus-neutralizing (VN) antibody titer within 1 to 2 weeks after exposure. The presence of antibody does not predict whether the bird is shedding virus, as birds with high antibody titers may still be shedding virus from the GI tract for many weeks. Even after a bird has ceased to shed virus from the GI tract, high antibody titers may persist for many years. Therefore, serology is of little help, except in the case of show birds, such as budgerigars and lovebirds, coming from a virus-free aviary. If these show birds are sero-positive after returning from the show, this indicates that they have been recently infected, and are likely also shedding virus. This is why birds that leave the aviary for whatever reason, do not re-enter until they have served a quarantine period. If the returning show birds are sero-negative after a 2-week quarantine period, they can be considered to not have been infected with APV during their sojourn. If returning show birds are sero-positive, they need to be tested periodically during quarantine until they have 1 or 2 negative oral/cloacal PCR tests, before they can be re-introduced to the aviary.

Transmission:
Several routes of transmission are possible: (1) parents feeding babies via regurgitation of sloughed crop epithelial cells; (2) inhalation or ingestion of feather follicle dust; (3) via the urates, as inclusions are found in the kidney of carrier birds; (4) via the feces; (5) via contaminated feeding utensils used in handfeeding babies. Asymptomatic carrier birds are extremely common for several weeks after an outbreak, but the shedding declines over the next 7 to 10 months. Only rare birds continue to shed virus longer than 10 months, but these are probably the sources of virus for subsequent outbreaks. Often the breeder birds are totally asymptomatic, but babies die at various ages depending on the species. Egg transmission in budgies is suspected, as virus has been identified in day-old budgie chicks.

Differential Diagnoses:
Chlamydophilosis, Pacheco's Disease, salmonellosis, fungal infections and Gram-negative bacterial infections.

Treatment:
There is no specific treatment once infection has occurred, only supportive treatment. Few birds with acute clinical signs recover, but numerous birds may be subclinically infected and develop immunity. A killed vaccine is available from Biomune.

Prevention and Control:
Practice good hygiene in feeding hand-fed babies. Do not use the same feeding utensils for different clutches of chicks. Do not mix chicks from different clutches or different sources. Closed breeding flocks are strongly recommended, with vaccination of the breeder birds in the off season. Although not conclusively proven, it is expected that the hen will pass protective maternal antibodies to the chick through the yolk sac. Vaccination of the breeder flock also decreases the amplification of virus numbers as it passes through a subclinical bird and also decreases the amount of virus in the environment.
Extensive disinfection of the nursery and any incubating equipment is necessary, as the virus can persist outside the body for weeks to months. Drag swabs from air ducts, door frames, shelves, equipment and incubators can be tested with the PCR DNA probes to determine the efficacy of the cleaning. Often incubators cannot be completely disinfected, and therefore should be disposed of (and not sold or given to some unsuspecting breeder).

Vaccination in the face of a nursery outbreak can be done, but it is a race to develop protective antibodies before the chicks reach the susceptible age in a contaminated environment, therefore it is not the most economical use of funds. It is better to assume that all the chicks in the nursery at the time of the outbreak are positive for virus on blood and oral/cloacal testing, and to immediately stop introducing new baby birds into the nursery. Once mortality has ceased in 2 to 4 weeks, testing of oral/cloacal swabs can be instituted, so that it can be determined when viral shedding has ceased, and the birds can be safely sold or exit the nursery.

It is recommended that the vaccine be given to nestlings that are 4 weeks of age, with a second vaccination 2 weeks later. It is believed to provide protection 2 weeks after the second vaccination is given. Unfortunately, this protocol often does not fit with the dynamics of an APV outbreak, as most birds cannot be immunized early enough in their lives to provide predictable protection from infection. Plus 4-week-old conures are quite small and vaccinating them can be technically difficult. VN antibody titers were not detected in vaccinated nestlings, suggesting that they may not be responding adequately to vaccination due to maternal antibody or an immature immune system. Even vaccination of adult birds only produced low antibody titers. Vaccination does not appear to change the shedding status of already infected birds, so vaccination does not exclude chicks from PCR testing for evidence of shedding.

Suspension of breeding will also decrease the number of susceptible birds and reduce the amount of virus present in the environment. Artificially incubating eggs may not break the cycle of infection, since APV may be egg-transmitted.

With the advent of the PCR DNA probe test, it may be possible to eradicate the virus from an aviary. In addition, testing newly acquired birds as part of the pre-purchase or post-purchase examination will hopefully prevent new carrier adults from being introduced into a clean aviary. Again the extent of the asymptomatic carrier problem is unknown, and is likely highly variable between aviaries. Routine monitoring tests during times of stress would appear useful in detecting intermittent or prolonged shedders of the virus.

Pet stores are the most common sources of APV outbreaks, since they often obtain their birds from multiple sources and then mix them together in cages and rooms. Pet stores often also sell lovebirds, cockatiels and budgerigars (the three species that are most likely to be virus shedders), in the same air space that they are selling naive, susceptible species, such as macaws, conures, and Amazon parrots. The less expensive lovebirds, cockatiels and budgerigars usually don’t die, but the more expensive larger psittacine chicks contract the virus, amplify it, shed more virus into the environment, spread it throughout the store, and die. Pet stores can prevent this problem by bringing only completely weaned psittacines into the store, since weaned birds are usually old enough, that even if infected with APV, they do not develop clinical disease. Alternatively, if a pet store wants to sell unweaned, susceptible, high-value psittacines, they should not sell lovebirds, cockatiels or budgerigars. Pet stores should purchase chicks from aviaries that maintain APV-free flocks. However, if the stores then bring in lovebirds, cockatiels, or budgerigars, which often are not tested because their economic value does not justify the cost of testing, they have put the higher value, naive birds at serious risk for acute infection and death.
There are conflicts between those practitioners that recommend using vaccination programs to prevent APV disease, and those that recommend maintaining APV-free flocks through testing. Both have their merits and it usually comes down to economics as to which program breeders opt for. Some practitioners feel that vaccination is more appropriate and economical, since chicks are then protected through their susceptible period and beyond, and because it is so difficult to keep polyomavirus out of an aviary or a pet store. However, vaccination may not 100% effective.

Once either a vaccinated or unvaccinated bird is installed in its final home or household, booster vaccines for APV are of questionable value. This is because exposure is unlikely to occur, unless these birds are boarded in facilities that take in birds from multiple sources. In that case, annual boosters may be appropriate, but hiring a pet sitter familiar with tending birds is probably a better choice, not only for exposure to APV, but to Pacheco’s Disease and other agents.

**APV Infection in Non-psittacine Birds:**
APV infection has been documented in several species of passerines, most commonly in canaries and Lady Gouldian finches. The clinical disease may follow a classical psittacine APV infection, but more often it is a mild or sub-clinical infection that leaves the young birds unthrifty, thin, and prone to other conditions. At necropsy, there may be hepato-splenomegaly. Histologic lesions are less necrotizing than in psittacines, and tend to be more lymphoplasmacytic lesions, involving the liver, kidney, heart and spleen. Viral inclusions are infrequent and usually occur in the liver. PCR DNA probe testing for a passerine or generic polyomavirus has been positive on swabs from necropsy tissue, as well as *in situ* DNA hybridization on tissues.

There is a single published report of death in a rhamphastid, a green aracaris, due to an APV virus. Sequencing of this virus found that it was nearly identical to the psittacine APV.
**PSITTACINE PROVENTRICULAR DILATATION DISEASE (PDD)**

**History and Research:**
This syndrome was first reported as Macaw Wasting Syndrome, but is also known as psittacine myenteric ganglioneuritis, infiltrative splanchnic neuropathy, and neuropathic gastric dilatation. First reported in the late 1970's in macaws, this disease is now seen in cockatoos, Amazons, conures, African Grays, *Poicephalus* parrots, *Ectlectus* parrots, cockatiels and budgerigars. The single species most commonly affected is the African Gray parrot. Greater than 50 psittacine species have been observed with PDD. There are also reports of PDD-like disease and lesions in Canada geese, swans, and a canary.

In the 1990's, Dr. Branson Ritchie at the University of Georgia, reproduced the disease experimentally with crude virus extracts, and viral particles could be found on electronmicroscopy of the fresh feces from affected birds. Virus isolation attempts and further characterization was not successful. However, the virus appears to be quite labile and does not live long outside the host. This made it difficult to visualize the viral particles on EM if examined greater than 24 hours after collection. The virus also did not survive freezing well.

Finally, in 2008, two independent research groups detected bornavirus sequences in tissues of naturally infected psittacine birds with histologic lesions of PDD using the Viro-Chek system. It was determined that a novel avian bornavirus was at least one of the causes of PDD in psittacine birds. Previously bornavirus, the causative agent of Borna Disease, was the only known member of the family Bornaviridae. Borna Disease virus was the cause of a neurologic disease occurring in horses and sheep and was confined to the area around the town of Borna, in the state of Saxony in Germany.

Since 2008, 6 different genotypes of ABV have been found in psittacine birds, as well as additional genotypes in a canary, trumpeter swans and wild Canada geese. ABV is an RNA virus. Psittacine genotype 4 seems to be the predominant genotype worldwide, and also appears to be more pathogenic than the other genotypes.

Experimental infection of Patagonian conures with virus grown in duck embryonic fibroblasts, resulted in seroconversion at 33 days post-infection. Detection of fecal shedding of virus could not be detected until 60-62 days post-infection. Clinical disease developed at 66 days post-infection. Typical lesions and bornavirus could be detected in brain post-mortem, suggesting that the disease originated as a viral encephalitis. In another experimental study utilizing cockatiels, findings were similar. The avian bornaviruses could not be grown in mammalian cell lines, suggesting that they may be unable to infect mammals.

**Transmission and Incubation Period:**
The route of transmission has not been definitively determined, but it appears to be feco-oral. There is also emerging research that ABV can be transmitted vertically from the hen to the egg. The incubation period for natural infection is unknown, but it appears to be long and quite variable. This may be because of our previous inability to detect early ABV infection; variability in the severity of lesions; variability in the route of transmission; or variability in the pathogenicity of ABV genotypes. Reports of this disease in contact birds have long suggested an infectious etiology. Occasional "outbreaks" of this disease have occurred in aviaries with the loss of several birds over the period of a few months. Often a few of these birds in the outbreaks would lack the gross lesions, but still have striking histologic lesions consistent with the disease. Macaws as young as 12 weeks of age have died with classic lesions.
ABV is quite labile and does not survive long outside the host and usually requires close contact for transmission. This feature helps to prevent its spread within an aviary. Juvenile birds, raised by infected parents or exposed to other infected juveniles, seem to exhibit a higher incidence of disease than birds that are adult when they are exposed. Prevention of transmission typically involves sanitation, disinfection of cages and premises, improving ventilation, and spreading out the birds (increasing the distance between individual birds or breeding cages). When a mate dies of PDD, the remaining mate should be quarantined for a long period of time and radiographed periodically. Even with this, the surviving mate is still highly suspect, and it is questionable whether the remaining mate should be paired with another bird. Since there is research indicating that ABV can be transmitted through the egg, it may not be possible to raise virus-free chicks by incubating the eggs artificially and rearing the chicks away from the infected birds.

Clinical signs:
This disease appears to involve an early systemic viral infection with a tropism for neural tissue, both the central and autonomic nervous systems. Clinical signs may include weight loss, muscle wasting, passage of intact seeds in the droppings, regurgitation and/or abdominal distension. This disease may also present with primarily neurologic signs, which may be vague and inconsistent, or may involve weakness, ataxia, tremors, proprioceptive deficits, or seizures. The disease is commonly fatal, but some birds, which are mildly affected have been maintained for long periods (weeks to months) on pelleted, pureed or liquid diets and metoclopramide to promote peristalsis of the GI tract. These birds, however, are at risk for aspirating food into the lungs during regurgitation episodes and dying acutely.

Diagnosis:
Ante-mortem diagnosis can be difficult, but is usually made presumptively from the clinical signs, radiographic, and/or laparoscopic findings. Often a contrast upper GI radiographic study is necessary to outline the proventriculus. Clinical pathology findings are surprisingly unremarkable, although an elevated CPK is a fairly common, but non-specific finding. Hematology parameters are usually normal, except when there is an aspiration pneumonia present. Birds with the neurologic form may not be diagnosed until necropsy and histologic examination of the brain and spinal cord are performed.

Ante-mortem biopsy of the wall of the crop has been recommended as a method of definitive diagnosis of PPD, however, the segmental distribution of the lesions often results in normal findings in the crop, but the bird still dies from PPD affecting other areas of the GI tract. Therefore, a negative crop biopsy does not rule out this disease. In my experience, less than 30% of crop biopsies from clinically affected birds contain the lesions of PDD, despite the eventual diagnosis of PDD lesions in other parts of the GI tract or the CNS at necropsy. Proventricular and ventricular biopsy may be performed, but segmental lesions are still a problem and biopsy of a thin-walled, dilated proventriculus carries the inherent risks of rupture and dehiscence. Ventricular biopsy is often less problematic and can be accomplished endoscopically.

Pathology:
Gross pathologic findings consist of emaciation; a dilated, thin-walled proventriculus filled with ingesta; ventricular and proventricular ulcers; and/or a thin-walled ventriculus with smooth muscle atrophy. Occasionally, the duodenal loop may be quite dilated. Intestinal candidiasis is also common, probably due to slowing of the gut transit time. Histologic lesions may include lymphocytic leiomysitis with smooth muscle degeneration and lymphocytic myenteric ganglioneuritis, with segmental lesions in the crop, esophagus, proventriculus, ventriculus and/or duodenum. Lesions are also common in the brain and spinal cord, especially in birds with neurologic signs: multifocal lymphocytic encephalitis with perivascular cuffing and gliosis and lymphocytic poliomyelitis. Birds dying from the neurologic disease often also have microscopic lesions in the GI tract, but may be grossly unremarkable. Lymphoplasmacytic adrenalitis and myocarditis (usually involving the Purkinje fibers and epicardial nerves) are also fairly common lesions.
Eosinophilic intranuclear and intracytoplasmic inclusions have been observed in nerve cells of intestinal ganglia, but these are rare. There may also be microscopic lesions in the spinal ganglia, peripheral nerves, the vestibulo-cochlear ganglia, and the eye (optic nerves, choroid, iris, pecten and ciliary body); severe retinal lesions and blindness are reported rarely, but this may be because ante-mortem detection is difficult and the eyes may not be routinely examined at necropsy.

To increase the chance of diagnosis (segmental disease is common), include brain, spinal cord, adrenal, crop, proventriculus, ventriculus, duodenal loop and heart in the tissues sent for histopathology.

Histologic lesions have been the gold standard for decades. Although post-mortem confirmation of ABV infection can now be done with various detection methods (RT-PCR, IHC, virus isolation, etc.), this testing has been primarily done in research situations, and is not usually done in clinical cases. Further research may change this paradigm.

**Prevention:**
What clinicians and owners want is a protocol for the accurate ante-mortem diagnosis of birds with PDD or infected with ABVs, so that infected birds can be isolated from other naive birds or excluded from breeding aviaries. Research is ongoing and protocols are likely to change as more information is obtained.

Serum antibodies to ABVs have been demonstrated, but serology could not distinguish between birds with PDD and symptomatic ABV carriers. Unfortunately, data has shown that ABV-infected birds are not consistently viremic, so serial blood PCR tests may not be consistently positive. ABV RNA can be detected in blood, choanal/cloacal swabs, feces and crop tissue ante-mortem, usually with a PCR or RT-PCR technique. It has also been shown that ABV shedding may occur intermittently in the saliva and feces, and that surgically collected crop biopsies may test ABV-negative in some patients with PDD. To complicate matters further, some naturally infected birds may shed virus without obvious clinical signs of PDD, but they are infectious to other birds.

A highly sensitive PCR test for detecting ABV shed in the feces or from the oral cavity/choanal slit has been developed by researchers at Texas A&M University, home of the Schubot Exotic Bird Health Center. See their website at [http://vetmed.tamu.edu/schubot/services](http://vetmed.tamu.edu/schubot/services). Single sample collections may be tested, but three samples collected one week apart may also be done in an effort to catch intermittent shedders. The best sample seems to be a choanal swab, or combination choanal/cloacal swab, but cloacal/fecal swabs may also contain virus. RT-PCR testing is also offered by the U of Georgia Infectious Disease Lab, and some commercial labs may also offer testing.

Interpretation of choanal/cloacal/fecal PCR results may raise additional questions until sufficient research has been conducted to solidify the most appropriate protocol. According to the TAMU website, a positive test indicates that a bird is infected with some strain of ABV, however, the test sensitivity may not be the same for all ABV genotypes. In addition, a single positive test does not imply that a bird is currently ill or that a bird will become ill with PDD in the future. Currently, it is not known whether all ABV-infected birds will become ill from the virus infection. ABV shedding has been detected in clinically healthy birds, and ABV shedding has been detected in closed aviaries that have had no clinical signs of PDD or deaths from PDD for years. There may be differences in the immune response between Old World and New World birds. There may be pathogenic and non-pathogenic strains of ABV in birds.

Until we have more research information, especially with longitudinal studies, euthanasia **is not recommended** for ABV-positive birds. Removal from the breeding aviary or segregation from ABV-negative birds in the home does seem to be prudent, however. Solitary pet birds in the home should not be
a problem, as long as they remain solitary.

**Treatment:**
The clinical manifestations of PDD are quite variable as to body systems involved, and are often variable in their severity. Critically ill birds have a very poor prognosis, and euthanasia may be the best option. However, depending on the severity of clinical signs, owners may opt for a period of treatment (2 to 4 weeks, usually), and assessment of response.

Celecoxib, a COX-2 inhibitor, had produced fairly good results in cases documented by crop biopsy and radiology. The rationale for the use of COX-2 inhibitors is to decrease the inflammatory reaction involving the myenteric nerves initiated by the virus infection. The recommended dose is 20 mg/kg BW once daily if given directly orally, or 40 mg/kg BW once daily in the bird’s food; this can be provided in a small amount of food fed before the normal ration is provided. Clinical response is gradual and slow, and birds usually need at least 2 weeks of treatment before there is much detectable response.

Meloxicam has been used for PDD treatment, but its response has been inferior to celecoxib in most situations. Tepoxalin has shown promise at 40 mg/kg BW daily mixed in a small amount of food. Often the ability to get a consistent dose of any medication into a bird makes all the difference.

The most common side effect of celecoxib or tepoxalin therapy is GI ulceration and bleeding. Droppings should be monitored daily for evidence of melena or fresh bleeding, and treatment discontinued immediately if seen, followed by prompt evaluation of the bird by the clinician. There has also been a report of a macaw that developed severe facial pruritus during treatment, that resolved with discontinuance of the celecoxib. Since most NSAIDs are eliminated through the kidneys, pre-treatment evaluation of kidney function is recommended, and treatment used cautiously in cases of existing renal disease. Regular monitoring of chemistry panel changes is also recommended with long-term use to detect evidence of developing renal disease.

The inflammatory lesions can often be reversed to some degree, and it is possible for function to return to near normal, but this treatment is not a cure and does not by itself reduce viral shedding or otherwise make the infected bird less infectious to other birds. There is some concern that unscrupulous owners/breeders may sell a successfully treated bird without explaining the possible consequences to the new owner.

There are many other supportive measures that can help infected birds with clinical PDD. Excellent reviews of the whole PDD story, as well as supportive therapy suggestions, can be found in *Veterinary Clinics of North America: Exotic Animal Practice*, volume 12, number 3, September 2010, pp 471-494 and pp 495-508.
AVIAN HERPESVIRUSES
These double-stranded DNA, enveloped viruses, commonly develop intranuclear inclusion bodies. These avian herpesviruses are alpha-herpesviruses, and all produce persistent infections with periods of recrudescence. Viremia develops rapidly with many of these viruses and often causes a necrotizing process in parenchymatous organs.

Pacheco's Disease Virus (PDV) – Psittacid Herpesvirus (PsHV)
Pacheco’s Disease was first described by Pacheco and Bier in Brazilian psittacines in 1930. The first reported cases in the U.S. were in 1975. This is a highly contagious disease and all psittacines are susceptible to some degree. Infection with PDV may cause acute, severe necrotizing hepatitis and death, or may cause subclinical disease and subsequent asymptomatic carriers. There is also now evidence that internal papillomatous disease (aka cloacal papillomatosis) is caused by infection with PDV in persistently infected birds.

There are four major genotypes of Pacheco’s Disease (PD) viruses. Genotypes 1, 2 and 3 are highly pathogenic to Amazon parrots in North and South America; genotypes 1, 2, 3, and 4 kill Amazon parrots in Europe. Genotype 4 is the most common cause of PD in conures and macaws. Cockatoos, cockatiels, and other Austral-Asia parrots are relatively resistant to clinical disease with PD, but when they do develop clinical disease it can be related to infection with any of the 4 genotypes. African Grey parrots are susceptible to infection with genotypes 2, 3 and 4.

Viral transmission is via ingestion, inhalation or conjunctival contact with droppings, oral secretions and/or vomitus. Birds that develop clinical disease rarely survive, but there appear to be subclinical infections that result in chronic asymptomatic carriers that may shed virus continually or intermittently. The persistently infected birds do not subsequently develop PD, but some go on to develop internal papillomatous disease.

Persistently infected species include macaws, Amazons, some of the Aratinga conure species and Patagonian conures. Cockatiels, cockatoos, lovebirds and possibly additional species may also be persistently infected. The highest risk of persistent infection is in birds that have survived an outbreak of PD in an aviary, wild caught birds that have passed through a quarantine station, and the parent-raised chicks of wild caught birds. Often an outbreak starts after some stress, such as a change in environment (importation, quarantine station, pet shop, breeding, etc), which causes a recrudescence and viral shedding increases in amount and frequency.

Pacheco’s disease was once dreaded and feared by psittacine breeders, but in the last 10 years, there has been a marked decrease in diagnosed cases. Factors that have probably played a role in the marked decrease in clinical PD include cessation of importation, and thus quarantine station exposure, the “closed aviary concept”, and the detection and isolation of asymptomatic carrier birds.

Clinical signs:
Clinical signs may be absent initially, and all that is seen is acute death of well-fleshed birds. Very early in the course of the disease, one may see regurgitation, bright yellow urates, +/- diarrhea, and facial icterus (in macaws with white face patches). The incubation period is as little as 48 hours in experimentally infected cockatiels and budgies, but in field cases the incubation period ranges from 5 to 14 days.

Diagnosis:
A history of several acute deaths of previously healthy birds is highly suspect for PD. Gross necropsy lesions include well-fleshed birds with swollen, pale, yellow livers with petechiations; swollen, friable
spleens; epicardial and serosal hemorrhages; and sometimes swollen kidneys. Some birds may have no grossly detectable lesions. Histopathology reveals widespread, acute, hepatocellular necrosis, with little inflammation, and splenic necrosis. In some cases, there may also be acute necrotizing pancreatitis, and necrotizing enteritis. Characteristic intranuclear, eosinophilic, and sometimes basophilic, intranuclear inclusions in hepatocytes can be seen, and occasionally may be found in the spleen and pancreas. The viral inclusions are highly suspicious for PD, but should be confirmed with the use of PCR DNA probes. Failure to identify viral inclusions does not rule out PD. At necropsy, a swab from the cut surfaces of fresh or frozen liver, spleen and kidney can be submitted for PCR. In situ DNA hybridization is also available for formalin-fixed and/or paraffin-embedded tissues, and some laboratories offer fluorescent antibody or immunohistochemical confirmation. Virus isolation and electron microscopy are seldom necessary, but are available at some laboratories.

PCR DNA probes are available for testing live birds, using a combined swab from the oral cavity and cloaca, and using blood samples. Serologic diagnosis of exposed and/or carrier birds can be done, but its significance is not clearly known.

**Differential Diagnoses** include chlamydophilosis, salmonellosis, avian adenovirus, avian reovirus, Newcastle Disease (paramyxovirus), bacterial septicemias, lead/zinc poisoning, and toxic inhalations.

**Treatment:**
Usually by the time clinical signs are evident, there is little chance that treatment will save infected birds. The aim is to attempt to stem the rapid spread of the virus to adjacent cages and perhaps reduce viral replication and death in birds in the incubation stage. The prophylactic use of acyclovir (Zovirax) has shown some efficacy during outbreaks in decreasing mortality in exposed birds. Mortality often ceases within 24 hours of treatment initiation. Acyclovir is recommended at the rate of 1 mg/ml of drinking water and food at 400 mg/quart of seed simultaneously for birds that are asymptomatic and capable of eating and drinking. Acyclovir can also be administered by gavage at 80 mg/kg BW every 8 hours in ill birds or high risk birds. An oral dose of 330 mg/kg BW every 12 hours has also been recommended. The appropriate length of treatment has not been determined, but most clinicians treat flocks prophylactically for 7 days after the last death, and treat clinically ill birds for 2 weeks. It is not known whether acyclovir treatment cures the viral infection, but it probably does not, so surviving birds should be treated as possible carriers until proven otherwise through testing.

Close attention needs to be paid to methods of virus transmission within the aviary by fomites, fecal dust, feather dander, and personnel. Moving cages farther apart and/or placing barriers between cages can decrease the viral spread. Traffic through the aviary needs to be minimized, and the clinically ill birds should be treated last in the treatment schedule, to prevent transmitting the virus to apparently normal birds. Hygiene should be improved, but it should also be realized that extensive cleaning may aerosolize infectious virus and cause further spread of the disease within the aviary. Certainly, no new birds should be introduced during or shortly after an outbreak.

The outbreak may disappear as quickly as it occurs, or may linger for weeks to months with periodic mortalities. Rarely do all exposed birds die, and this may indicate that surviving birds are immune for some reason, whether because they have been previously exposed to a less virulent strain and are persistently infected; have more innate immunity to the particular genotype involved; or have been previously vaccinated.
**Control and Prevention:**
A killed virus vaccine, Psittamune PDV, manufactured by Biomune, has been available periodically in the United States. Two immunizations, 2-4 weeks apart, with an annual booster are recommended. This is an oil emulsion vaccine; most avian veterinarians give it SQ, not IM. Severe granulomatous myositis has been reported in cockatoos with IM vaccine administration. The capability of this vaccine to protect against all genotypes of PDV is unknown. Protective antibody titers should not be expected until at least 2 weeks after vaccination, so there is questionable benefit to vaccinating in the face of an outbreak. Vaccination of high risk species in mixed collections may be the most beneficial use.

Husbandry and management procedures, such as the closed aviary concept, good quarantine procedures, and obtaining birds from reliable sources can go a long way in preventing PD outbreaks. In the closed aviary concept, if a bird leaves the aviary, it does not return until it has gone through the quarantine period, and testing. PD outbreaks are less common in outdoor aviaries, but other diseases are more common in outdoor aviaries.

The advent of PCR testing of live birds has become extremely helpful in preventing PD outbreaks. Detection of persistently infected birds can be accomplished by PCR testing of blood and combined oral and cloacal swabs. Persistently infected birds are then strictly isolated from the negative birds. A second testing of negative birds should pick up any birds that may have slipped through the first test. All incoming birds need to serve their quarantine period, and be tested at the start of the quarantine period.

**Internal Papillomatosis (IP):**
Some of the birds that are persistently infected with PDV may go on to subsequently develop IP. The most commonly affected birds are macaws, Amazon parrots, hawk-headed parrots and rarely, conures. Lesions are usually confined to the oral, glottis and/or cloacal mucosa, but occasionally lesions may be found on the conjunctivae, crop, proventriculus and ventriculus. The cloacal papillomata are often first recognized by finding blood in the droppings or the cage bottom. The cloacal lesions become ulcerated and may periodically prolapse through the vent. Oral lesions are usually asymptomatic, unless they involve the glottal opening, in which case, they may cause episodic respiratory signs. It has long been known from clinical cases, that mates, and even cagemates of different species, may develop IP lesions, suggesting the transmission of an infectious agent. Transmission from parent to offspring is also seen, if the chick is parent-fed for any period of time.

The papillomatous lesions typically wax and wane in size and severity, and may occasionally spontaneously resolve completely. Ulcerated lesions can become secondarily infected, resulting in pain, hemorrhage, and systemic signs of illness; these infected lesions usually also smell bad. Prolapsing lesions are prone to drying and trauma by the bird or by cagemates. The oral lesions are usually pedunculated. The cloacal lesions may be pedunculated, but at least as often are sessile and broad-based. The cloacal lesions may be focal or quite extensive, sometimes being almost circumferential within the cloacal mucosa.

Treatment may involve attempts to remove all or part of the lesions; partial removal may cause the remaining lesion(s) to regress, much like papillomas caused by papillomaviruses in mammalian species. Various methods have been used, including cold steel sharp dissection, cryotherapy, chemical cauterity (silver nitrate), electrocautery, and laser surgery. Stricture formation may be a sequel both to surgery and the mere presence of the chronic or extensive lesions. Often surgery is done in stages, removing a small portion of the mass at each stage. These lesions are often painful when ulcerated and inflamed, so antibiotics and pain medication are appropriate supportive therapy. There are anecdotal reports of lesion regression with feeding dried red chili peppers as 1% of the diet, suggesting that capsicum may a beneficial effect.
Bile Duct and Pancreatic Duct Tumors:
A small, but significant, percentage of birds with IP go on to develop tumors of bile duct and/or pancreatic duct epithelium. These tumors may start out as benign proliferations, but eventually progress to florid malignancies, that encompass most of the liver and/or pancreas. Chronic infection with PsHV genotype 3 appears to predispose birds to developing bile duct carcinomas. Parents and their offspring have died with these tumors, so transmission probably occurs during parent feeding. The clinical signs of bile duct carcinomas are non-specific. Loss of weight and condition, beak overgrowth and abdominal enlargement may be seen and elevations in serum GGT have been reported in advanced cases. At ultrasonography, an enlarged liver with hyperechoic, round to irregular masses can be seen. Confirmation is by biopsy, although most of the affected birds are diagnosed at necropsy. These masses have not been amenable to excision or other treatment in most cases due to their extensiveness.

Herpetic Foot Lesions:
Macaws with slightly raised, depigmented plaques on the toes have been recognized for many years, especially in wild-caught birds. Papillary, warty lesions on the plantar surfaces have been seen in cockatoos. Both of these lesions have been reported to regress with topical acyclovir, which is fortunate since they often are not amenable to surgical excision. The lesions usually do not seem to cause the affected birds concern. The DNA sequence of a herpesvirus from a plaque on a macaw closely resembled the sequence of the PsHVs that cause PD and IP. Further investigation is needed.

Miscellaneous Herpesvirus Infections in Pet Birds:
Severe respiratory infections in Bourke’s parakeets (Neophema bourkii) have been reported at necropsy. Necrotizing tracheitis, bronchitis and airsacculitis with intranuclear inclusions were seen histologically and virions consistent with a herpesvirus were seen at electronmicroscopy, but the virus could not be cultured or further characterized. It occurs only rarely now.

A severe herpesviral respiratory disease syndrome, termed Amazon tracheitis, was reported in Amazon parrots coming out of quarantine stations. At necropsy, conjunctivitis, blepharitis, sinusitis, rhinitis, pharyngitis, laryngitis and a necro-hemorrhagic tracheitis and bronchopneumonia were the predominant lesions; diphtheritic pseudomembranes and fibrinous exudates were seen, with death due to asphyxiation from inhalation of exudates. Herpesviral inclusion bodies were seen in the sloughing tracheal and bronchial epithelial cells, very closely resembling the disease of chickens, infectious laryngotracheitis (ILT), which is also caused by a herpesvirus. Experimental infections of chickens and Caucasus pheasants with the herpesvirus isolated from the naturally occurring disease in Amazon parrots resulted in the clinical signs and microscopic lesions of typical of ILT. The virus had group specific serologic similarities to ILT and was thought to be a mutant of the chicken ILT virus. The incubation period in experimentally infected Green-cheeked Amazons was 3-4 days, with contact birds dying after 6 days. This infection has only rarely been reported in the last 15-20 years, and seemed to be primarily a disease of importation.

Infection of Lady Gouldian, melba and purple grenadier finches with a cytomegalic herpesvirus was reported. Mortality of 25 to 100% was seen in the affected flocks. Severe conjunctivitis, dyspnea, weight loss and anorexia were the clinical signs. Histologically, large cytomegalic intranuclear inclusion bodies were found in the conjunctival, tracheal and airsac epithelium and herpesvirus virions were seen electronmicroscopically.

Pigeon herpesvirus (PHV):
PHV is a problem in racing pigeons worldwide. Clinical signs include protrusion of the third eyelid, conjunctivitis, rhinitis, depression, anorexia, dyspnea, weight loss and diarrhea. Diphtheritic plaques and ulcers can be seen in the oral cavity, pharynx, esophagus and crop. Viral inclusions can be found in
epithelial cells. This viral infection may be seen in conjunction with a number of other infectious disease processes in pigeon lofts, such as chlamydophilosis, mycoplasmosis, pigeon pox, trichomoniasis, candidiasis, salmonellosis and nematode infestations, such as with *Capillaria* spp in the pharynx, esophagus, crop and proventriculus. Therefore, stress is likely a complicating problem, whether from other disease processes, breeding or racing. Persistent infection occurs, so once this is diagnosed in a loft, it remains a problem that may wax and wane in its severity. It appears to be possible to break the infection cycle by fostering eggs from infected hens under uninfected hens, since the virus is not transmitted vertically. Since PHV may be similar to PDV, it not recommended to raise pigeons and psittacines in the same facility.

**Falcon herpesvirus:**
This herpesviral infection results in acute necrotizing inclusion body hepatitis in falcon-type raptors. It is associated with feeding infected pigeons to raptors. This virus may actually be the pigeon herpesvirus.

**Duck Virus Enteritis (DVE) aka Duck Plague, Pest du Canard:**
DVE is caused by the anatid herpesvirus, which infects ducks, geese and swans (members of the Anatidae family of the Order Anseriformes). Outbreaks occur most often in late spring and early summer. Wild, semi-domesticated and domestic ducks mix on small ponds which are often densely populated, and this increases the chance for transmission. The virus is widespread in the wild anseriform population, usually as asymptomatic carriers with periodic viral shedding. Disease is more common in the young ducks.

The susceptibility to disease is very dependent on the species of waterfowl. Among the wild waterfowl, blue-winged teal, wood ducks and redheads are highly susceptible. Muscovy ducks and gadwall are moderately susceptible. Mallards and Canada geese are less susceptible; pintails are the least susceptible. Mallards are considered a natural reservoir for the virus, since they are often more resistant to its lethal effects. Domestic ducks and geese usually contract the virus from their wild counterparts when they mix on small ponds.

DVE is transmitted by direct contact between infected and susceptible birds or indirectly through contaminated environments (pens, ponds, etc). Virus is shed in the feces and can contaminate land and water. Oral and intranasal routes of infection are the most common. The incubation period is 3 to 7 days, with death occurring 1 to 5 days after overt signs of disease appear. Profound weakness, bloody droppings, yellow urates, anorexia, anemia and dehydration are the most common signs, and may lead to ataxia and inability to move.

The gross lesions can be found in a number of organs. Vascular damage and tissue hemorrhages are common on epicardial and serosal surfaces. Widespread petechial hemorrhages are present in the liver, which is paler and yellower than normal. A hemorrhagic ring is common in the mucosa of the esophageal-proventriculus sphincter. Linear hemorrhages and necrosis in the esophageal mucosa is common. Large, depressed, hemorrhagic areas corresponding to the lymphoid patches in the intestine and cloaca are the source of the bloody droppings. Urates may be yellow or pale green due to widespread necrotizing hepatitis. Splenomegaly and splenic hemorrhage are common. Histopathology reveals hepatocellular necrosis with intranuclear, eosinophilic viral inclusions. Viral inclusions are also commonly found in the renal tubular epithelial cells. Lymphoid aggregates in the intestines are necrotic and hemorrhagic.

Virus isolation and identification by PCR based assays are confirmatory. These tests are offered by The Duck Research Laboratory at Cornell University. The Duck Research Laboratory also offers consultations on duck disease control and prevention, and sells a vaccine for DVE, as well as vaccines for other duck diseases. See [http://www.duckhealth.com/ducklab.html](http://www.duckhealth.com/ducklab.html) for more information.
AVIAN POXVIRUSES

Avian pox is caused by a variety of viruses of the genus Avipoxvirus of the family Poxviridae. This DNA virus is the largest virus known so far in vertebrates (the baculoviruses of insects are larger). Poxviruses produce intracytoplasmic, eosinophilic viral inclusions called Bollinger bodies in epithelial cells and are observed on histopathology. These inclusions are considered pathognomonic for avian pox infection.

Avipoxviruses are relatively resistant to environmental factors such as drying and sunlight, but steam destroys the virus easily. Virus in scabs can remain viable for years. Virus in contaminated soil can survive for 1-2 years. Avipoxviruses also tend to be chemoresistant, but 5% phenols are effective against it.

Avipoxviruses cannot penetrate intact epithelium; they need disturbed skin or mucosal surfaces. Transmission in the wild is probably via mosquitoes and other biting insects. Outbreaks in avaries have been attributed to fighting and the use of feeding utensils among several birds. Recovered birds may remain carriers of the virus for very long periods, probably for life, and may intermittently shed the virus from the skin or intestinal tract. For these reasons, once pox is diagnosed on a premises, it is considered permanently contaminated and susceptible birds may become infected.

Numerous species of birds are susceptible to their own adapted avipoxvirus strains. The typical avipoxvirus is fowlpox, which affects primarily chickens. There are also pigeonpox, falconpox, turkeypox, Amazonapox, lovebirdpox, canarypox, quailpox, starlingpox, sparrowpox, mynah pax, and many more. Most of the host-adapted strains tend to stay confined to their particular hosts, but there may be occasional cross-infection between similar species or genera of birds.

In general, avipoxvirus infections may present in three forms: dry pox, wet pox and septicemic pox. Dry pox is the cutaneous form. The lesions can be dry, crusty, umbilicated lesions, or can be raised, proliferative lesions that may resemble a tumor. Wet pox, the diphtheritic form, typically involves the mucous membranes of the oral cavity, esophagus, crop, nasal cavity, trachea and eye. This form is particularly dangerous because the diphtheritic membranes may slough and occlude the trachea, resulting in death. The septicemic form, primarily seen in canaries, is associated with a necrotizing pneumonia, and splenomegaly and hepatomegaly may also be seen.

Amazonapox:
Amazon parrots, especially the Blue Front Amazon parrots, and Pionus parrots are most susceptible. Ocular pox is the most commonly seen form with conjunctivitis, blepharitis, and keratitis, which may progress to anterior uveitis, endophthalmitis and phthisis bulbi. Recovered birds may have significant scarring of the eyelids and/or cornea. Fibrino-necrotic lesions involving the choanal slit, tongue, pharynx, and larynx may also be seen. Secondary bacterial and fungal infections of the lesions can be expected.

Since the end of importation, active cases of Amazonapox are rare. But clinicians can still see distorted eyelids and corneal scars in wild caught birds from the importation era. There is concern that under stress, recrudescence of lesions and viral shedding may occur. However, this is rarely reported.

Lovebirdpox (Agapornispox):
Predominantly a cutaneous form is seen with lovebirdpox, but the wet form with oral and ocular lesions can occur. The predominant lesion is located in the skin of the axillary area and is very pruritic. Violent self-mutilation occurs. Lesions are often chronic and recurring. Pox in lovebirds is uncommon now, but it should remain in the differential for any chronic self-mutilative cutaneous condition, along with bacterial, fungal, neoplastic and behavioral dermal conditions.
Canarypox:  
The cutaneous form is the most common, but a septicemic or pneumonic form is also observed. Bumblefoot or loss of toes due to secondary bacterial infection may be a sequel to canarypox infection. Canarypox remains a significant problem in avaries of canaries and certain finch species. In the pneumonic or septicemic form, respiratory difficulties are seen because of lesions in the nasal cavity, trachea and lung. High mortality can be seen with acute death, respiratory dyspnea, conjunctivitis, chemosis or sick bird syndrome (depression, anorexia). Canaries that survive the acute stage go on to produce skin lesions.

Poxvirus infections of wild birds are quite common in nature. Everyone has probably seen a finch, sparrow or starling at a bird feeder with discrete swellings on the toes or hocks, or with a swollen eye. Pigeonpox is also fairly common among wild pigeons, but can certainly spill over into the domesticated pigeons kept for racing. The young birds are more susceptible. Some of the pox lesions in pigeons are inverted and darkly pigmented. Even sea birds, such as puffins, have developed cutaneous pox lesions.

Falconpox is also quite common in a variety of falcons and hawks. The lesions are typically present on the unfeathered portions of the body, i.e., the legs (shanks) and feet, eyelids, and around the nose and the commissures of the mouth.

Diagnosis:  
Diagnosis is via history, lack of significant response to antibiotics, and biopsy of lesions. The spread of the disease tends to be slow, and this is one of the diseases, at least in chickens, where vaccination in the face of an outbreak may be appropriate. The infection spreads slowly enough that there is time for vaccination to become effective in flockmates. Histopathologic examination reveals epidermal hyperplasia with ballooning degeneration and large intracytoplasmic eosinophilic inclusions. Virus isolation and electron microscopy may be necessary in cases of septicemic canarypox, as histologic lesions may not be diagnostic.

Differential Diagnoses:  
The differential diagnoses for the wet pox form include vitamin A deficiency, trichomoniasis, candidiasis, aspergillosis, bacterial infections, and in Amazons only, Amazon tracheitis herpesvirus. For the dry pox form, bacterial and mycobacterial granulomas, and various tumors would be rule outs. For the pneumonic/septicemic form in canaries, bacterial infections are the primary differentials.

Treatment:  
Treatment is supportive with vitamin A injections, fluid therapy, force feeding and appropriate antibiotics or anti-fungals for secondary infections. The viral lesions regress on their own, but may take up to 6 weeks for resolution.

Prevention:  
Quarantine new birds. The fowlpox vaccine is a modified live turkeypox virus, which is administered by a stick or scratch technique in the wingweb area. A killed psittacine pox vaccine has been periodically available and so far appears effective in preventing both Amazonapox and Agapornispox. However, because of the rarity of cases in psittacine birds in the past several years, vaccination is rarely practiced now, and vaccine may be difficult to obtain. A separate vaccine is available for canarypox, which is administered by the wingweb stick technique.
AVIAN ADENOVIRUSES (AVIADENOVIRUSES):
Adenoviruses are double-stranded DNA, non-enveloped viruses. Transmission is via the feco-oral route, the respiratory route and possibly via the egg. These viruses have been isolated or observed on electron microscopy in sporadic outbreaks in many different species of psittacines, but infections appear to be rare now.

As a group, aviadenviruses tend to have a fairly species-specific host range and have been isolated from asymptomatic carriers. Most aviadenviral infections produce large basophilic or eosinophilic intranuclear inclusions in liver lesions, intestine, pancreas, and/or kidney.

Histologic lesions of a necrotizing hepatitis with large basophilic intranuclear inclusions were most commonly seen in recently imported birds. There have been multiple reports of adenoviral infections in African Grays, suggesting that the African psittacines, such as African Grays, Poicephalus parrots (Senegal, Cape, Red-bellied), and lovebirds, may be more susceptible to these viruses. Inclusion body pancreatitis has been reported in Nyasa and Peach-faced lovebirds. Inclusions may be found in the renal tubular epithelial cells of lovebirds and budgerigars that die of other diseases, so there may be an asymptomatic carrier state.

Non-suppurative meningo-encephalomyelitis, pancreatitis and adenoviral inclusions in enterocytes were described in Neophema parakeets and Pionus parrots with persistent torticollis and CNS signs, similar to those seen with paramyxovirus infections. A non-suppurative adenoviral encephalitis has been described in a cockatoo and in budgerigars in Germany.

The Infectious Disease Laboratory at the University of Georgia offers a PCR DNA probe for generic avian adenovirus which is most often performed on swabs from fresh or frozen tissues from necropsy. Immunohistochemical and/or in situ DNA hybridization is usually also available for formalin-fixed, paraffin-embedded tissues. Electron-microscopy can be performed on paraffin-embedded tissues, as well, usually at the California Animal Health and Food Safety Laboratory System at Tulare; Dr. H.L. Shivaprasad is an excellent contact person.

Aviadenviral infections have also been well described in quail (quail bronchitis virus). Pigeon adenovirus infections are a significant problem in European racing pigeons, resulting in significant enteritis and occasionally some liver necrosis.

Aviadenviral pancreatitis in guineafowl has been reported, as have occasional infections in raptors, grouse and waterfowl. Marble spleen disease in pheasants and hemorrhagic enteritis in turkeys are well-known aviadenviral infections of poultry.
**AVIAN PARAMYXOVIRUSES (PMV):**

These single stranded RNA viruses possess broad host ranges. There are 9 serogroups, with multiple strains within each serogroup. These viruses are significant in poultry.

**PMV-1 or Newcastle Disease (ND):**

ND is often divided into velogenic, mesogenic and lentogenic strains. Velogenic visceralotropic Newcastle Disease (VVND), is now known as Exotic Newcastle Disease (END), and is a reportable disease. It is transmitted via aerosol and feco-oral routes. Immune carriers exist which shed virus. The incubation period is 2 to 15 days, but up to 25 days depending on host species, dose and the pathogenicity of the strain. When quarantine stations still existed, they tested all imported birds via cloacal swab and virus isolation. All birds that died in quarantine were necropsied and tissues submitted for virus isolation.

**Poultry** (domestic chickens and turkeys) are highly susceptible to END. Morbidity and mortality approach 100%. Incubation period is 2 to 15 days in poultry. Clinical signs in poultry include lethargy, rapid respiration, edema around the eyes, green diarrhea, dyspnea, weakness, neurologic disease, dramatic drop in egg production, prostration and death.

Waterfowl (ducks, geese, cormorants) are readily infected, but are usually resistant to disease. Pigeons are also susceptible, but less so than poultry, and neurologic and respiratory disease predominates. Newcastle virus has also been isolated from ostriches with neurologic signs and torticollis in Israel.

**Psittacines and Passerines:**

The susceptibility among psittacine species is extremely variable and clinical signs are dependent on the strain and the host species. The acute disease signs are quite non-specific and may include depression, anorexia, weight loss and/or diarrhea. Respiratory disease signs may be present or absent. Birds surviving the initial infection may go on to develop neurologic signs, which may include ataxia, head tilt, head bobbing, chorea, opisthotonus and/or paralysis. Cockatoos, macaws, Amazons, Austral-Asian parakeets and conures are very susceptible, with primarily CNS signs. Cockatiels, Eclectus parrots and caiques are moderately susceptible. Weaver finches (Gouldian, Cordon-bleu) are highly susceptible to primarily respiratory symptoms.

**Necropsy Findings:**

Petechiation on serosal surfaces and mucosal surfaces of the larynx, trachea and proventriculus are commonly observed, as is necro-hemorrhagic enteritis with necrosis of lymphoid follicles. Histologically, a non-suppurative encephalitis with perivascular cuffing is seen, with or without interstitial pneumonia, splenic necrosis and lymphoid necrosis.

**Diagnosis:**

Diagnosis is usually made by virus isolation from cloacal swabs, feces, tracheal swabs or necropsy tissues (trachea, lung, intestine and contents, brain). Serology can indicate exposure, but this can be quite variable.

**Differential Diagnoses:**

For intestinal and respiratory signs or lesions, the primary differentials are chlamydophilosis, Pacheco's Disease, salmonellosis and other PMV infections. For CNS signs, differential diagnoses would include salmonellosis (brain abscess), PMV-3, Proventricular Dilatation Disease, West Nile virus, EEE, WEE, *Baylisascaris* larval migration, and other bacteria.
Treatment:
There is no specific treatment. Report suspected outbreaks to the regulatory agencies (USDA-APHIS, or state agriculture agencies).

Control:
Federal quarantine of imported birds was instituted to prevent the introduction of END into the US. Any suspected smuggled exotic birds should be reported to authorities. Any "bargain" Amazon parrots are suspicious. The 1990-91 Newcastle Disease outbreak in California was traced to Yellow Nape Amazons sold at flea markets; the 2003-2004 outbreak was traced to fighting gamefowl. Vaccines for psittacines are not available. In poultry, depopulation is the usual control method.

Pigeon PMV-1:
The pigeon PMV-1 is closely related to Newcastle Disease, but is serologically, biochemically and pathogenically unique. It was first recognized in domestic pigeons in the Middle East and Europe in the late 1970's. By 1981, the viral disease had spread all over Europe, affecting particularly racing and show pigeons. Non-descript clinical signs include polydipsia, polyuria, anorexia, diarrhea and vomiting, followed by wing paralysis, tremors and torticollis. Dyspnea is not reported. Control is through vaccination.

PMV-2:
This serotype appears to be endemic in African weaver finches, though it is also reported in the African Gray parrot. It is uncommon in psittacines in general. Signs include weight loss and pneumonia.

PMV-3:
This serotype is primarily a problem in pigeons, but has been isolated from lovebirds, Neophema spp. and other Australian parakeets (rosellas, etc), cockatiels, budgerigars, macaws, Amazons, and some passerine species. Disease is quite variable among the strains, but is primarily CNS-associated. It is not common in psittacines, except in Neophema. In Neophema, head tilt, torticollis and various neurologic abnormalities are seen. Non-suppurative encephalitis, pancreatitis, labyrinthitis and viral inclusions in the auditory epithelium of the middle ear are characteristic. In survivors, emaciation and voluminous, poorly digested droppings may develop due to chronic lymphocytic pancreatitis and pancreatic insufficiency. In passerines, conjunctivitis is the initial clinical sign, followed by dysphagia, diarrhea and dyspnea.
WEST NILE VIRUS (WNV)
WNV, a flavivirus, is a member of a complex of antigenically related, mosquito-transmitted viruses that includes St. Louis encephalitis virus, Japanese encephalitis, Murray Valley encephalitis virus, Kunjin virus and Israeli turkey meningoencephalitis virus.

WNV was first diagnosed in the United States in New York City in 1999, where American and fish crows were dying from acute progressive neurologic disease – ataxia, tremors, circling, wing droop and seizures. The disease was first diagnosed in the US by a veterinary pathologist at the Bronx Zoo, even before the first human case was diagnosed. The virus marched across the continental US and southern Canada provinces, and reached Oregon in 2005.

Free-ranging birds serve as the principal amplifying host. Infected migratory birds serve as both reservoir and transport hosts. Several mosquito species serve as vectors. Horses and humans are considered incidental dead-end hosts (barring blood transfusions and organ donations).

Outbreaks correspond with the mosquito season. In Oregon, that tends to be from June to early October (first killing frost). The virus also appears to overwinter in hibernating mosquitoes and transovarial transfer to progeny occurs.

Bird species vary markedly in their susceptibility to developing clinical signs of disease from WNV. Some birds develop high titers of virus and sustained viremia, while others clear the virus rapidly. Corvids (crows, ravens, jays, magpies) are very susceptible, develop rapid, high viral loads, progressing rapidly to death. House sparrows develop sustained viremia, but are asymptomatic, so they are probably important in the maintenance of virus and its dissemination. Waterfowl, pigeons and doves can be clinically affected and mortality is seen. Flamingos and pheasants are quite susceptible. Raptors (hawks, eagles, falcons, owls) are also very susceptible to severe neurologic disease. Certain owl populations have been decimated. Chickens seroconvert, but remain clinically normal, so they are used as sentinels.

Fortunately, psittacine birds seem to be fairly resistant to this disease, although there are reports of the virus killing parrots, usually those housed outdoors. Among the various psittacine species, so far there has been very low morbidity and mortality associated with WNV. Older psittacines seem more apt to be affected than younger birds. Serology is available in some states for ante-mortem diagnosis; there may be some cross-reactivity with St. Louis encephalitis virus, a related flavivirus.

WNV has been reported in zebra finches, cockatiels, cockatoos, macaws, loriikeets and emus. Most cases are diagnosed at necropsy. Care during necropsy is very important since there have been a couple of reports of transmission of WNV to humans from puncture wounds associated with the necropsy of infected animals. Personal protection that has been recommended during necropsy, include a face shield, mask, and three glove technique (latex layer, heavy Playtex-type glove layer, and Kevlar glove or filleting glove layer).

Necropsy findings may include brain hemorrhage, splenomegaly, and pale foci in the heart. Histologic lesions may include meningo-encephalitis, acute pancreatic necrosis, nephritis, myocarditis, and mesenteric vasculitis; other lesions are less common, but possible. Since the gross and histologic lesions can be caused by other viruses (PDD, Newcastle disease, EEE, WEE, PMV-3), definitive diagnosis of WNV requires demonstration of the virus. This is usually done by using a PCR technique to demonstrate viral antigen in fresh or frozen necropsy tissues (usually brain and kidney). Immunohistochemical tests may also be available for use on formalin-fixed or paraffin-embedded tissues. Virus isolation may also be available, but this procedure takes much longer, is more technically difficult than PCR, and requires live
virus particles for isolation. Virus isolation does not seem to work well in psittacines, probably because they maintain low titers of virus and rapidly seroconvert. Serology is most often used in surveillance and in testing sentinel chickens for seroconversion.

Currently, in Oregon, suspected cases should be reported to the office of the state veterinarian as a part of local epidemiologic importance to bird, horse, and human populations, and to county health departments or mosquito control districts. The number of cases in birds, horses, and humans, and the number of positive mosquito pools peaked in 2006. Virus activity was low in 2008 through 2011, but increased in 2012. Updated information can be found at several websites (state health departments, state veterinarian websites, CDC, etc).


Treatment is supportive care. Mildly affected birds can recover. Severely affected birds, such as owls or falcons, seem to linger, but many eventually die or are euthanized because of failure to respond, or because of persistent neurologic deficits. Zoos and collections of raptors (falconry aeries, educational exhibits) have been vaccinating their birds with the killed virus Fort Dodge equine vaccine.

Not much work has been done on the efficacy of the vaccine and most of the information is anecdotal at this stage. A study at Louisiana State University reported on the effect of immunizing a group of cockatiels with the equine vaccine; the birds tolerated the vaccine, but did not produce demonstrable antibody titers. The cockatiels were not challenged with live virus, so it is not known whether they were protected or not. Despite these findings, zoos that had experienced deaths due to WNV, found that mortality and morbidity was markedly decreased in vaccinated birds. The question remains whether the vaccine actually worked, or whether the birds mounted their own immune response to WNV during natural infection.

Facilities are also screening their buildings, windows, and outdoor flights. Getting rid of any sources of standing water (mosquito breeding sites) is highly recommended. This may include bird baths, tires, storm drains, clogged gutters, pet water dishes, etc. Changing the water every day or so in outdoor pet water dishes and bird baths should prevent mosquito larva from continuing toward hatching.
AVIAN VIRAL SEROSIS
Gaskin at the University of Florida described a syndrome in juvenile macaws in a single aviary in the early 1990's. The consistent clinical sign was marked abdominal distension. At necropsy, severe ascites, edematous lungs, pericardial effusion and proventricular dilatation were observed. Histologic lesions consisted of non-suppurative serositis, proventricular and ventricular leiomyositis, myocarditis and interstitial pneumonia. A suspected togavirus was isolated and inoculation of young Leghorn chicks reproduced the disease. The virus isolated was serotyped at the Center for Disease Control as Eastern Equine Encephalitis Virus (EEE). This condition is reported rarely now.

Since that discovery, EEE was isolated from emus with fatal hemorrhagic enteritis and hepatitis in Louisiana in the mid 1990's. EEE has also been isolated from groups of Lady Gouldian finches with neurologic signs that were housed outdoors in Florida. These findings show that psittacines and non-native passerines are susceptible to EEE, and possibly other togaviruses, and that protection from mosquitos is important in endemic areas.

The most common avian species developing encephalitis with EEE virus are pheasants and partridges. The virus is spread by blood-sucking insects (mosquitos, mites, lice, blackflies, culicoides) and also by cannibalism. Western equine encephalitis (WEE) has been isolated from ratites with neurological diseases in the southern United States.

AVIAN REOVIRUSES
The reoviruses are double-stranded RNA viruses. Three genera exist in birds: orthoreovirus, orbivirus and rotavirus. Infectious bursal disease of chickens was previously classified as a reovirus, but is now classified as a birnavirus. Many different reoviruses exist and they tend to cross species lines in outbreaks.

Most of the reoviruses in psittacines were associated with wild caught, recently imported African Grey parrots, with horizontal spread to cockatoos in quarantine stations. This virus is rarely isolated now, with the cessation of importation in the US, but since Greys continue to be imported to Europe, it continues to be a problem there.

Horizontal, feco-orlal transmission is presumed; the incubation period is 4-6 days in experimentally infected African Grays. Reoviruses are occasionally isolated in concurrent bacterial and/or fungal infections; the reovirus may be immunosuppressive or may be associated with pre-existing immunosuppression from psittacine circovirus infection. Clinical signs are non-specific and include depression, weakness, weight loss, yellow urates and death.

At gross necropsy, hepatomegaly and splenomegaly are observed. Histopathology reveals multifocal, acute hepatic necrosis and lymphoid depletion in the spleen. No inclusions are observed. Diagnosis is via virus isolation in chick embryos. Differential diagnoses would include Pacheco's disease, adenovirus infection, chlamydophilosis, and bacterial septicemias. African Grays and other Old World parrots appear more susceptible to infection, while New World parrots seem more resistant. A wide variety of psittacines have been infected with reoviruses. Asymptomatic carriers are suspected. Reoviruses have been isolated from both symptomatic and asymptomatic birds in quarantine stations.