**CANINE INFECTIOUS DISEASE UPDATE**

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*Bordetella bronchiseptica* INFECTION and PREVENTION  
Closely related to *Bordetella pertussis*, the cause of “whooping cough” in humans, *Bordetella bronchiseptica* is a gram negative, aerobic coccobacillus particularly well adapted to colonize the ciliated respiratory epithelium of dogs and cats. Today, this organism is regarded as the principle etiologic agent of canine infectious tracheobronchitis (ITB). In the clinical setting, however, *B. bronchiseptica* infection should not be regarded as synonymous with ITB. Dogs infected with canine parainfluenza virus (CPiV) or canine adenovirus-2 (CAV-2) are expected to experience more severe respiratory disease when co-infected with *B. bronchiseptica* than with any these agents alone. Canine bordetellosis, i.e. *B. bronchiseptica* infection in the absence of either CPiV or CAV-2, is known to occur and can be associated with acute, fatal pneumonia in young dogs. *B. bronchiseptica* is transmitted through aerosolization of respiratory secretions. Bacteria can also be transmitted directly by contaminated dishware, human hands, and other fomites. Because *B. bronchiseptica* possesses several intrinsic mechanisms for evading host defenses, it is recognized for its role as a significant complicating factor in dogs with multiple-agent respiratory infections. The fact that outbreaks of canine ITB are common, despite widespread use of topical and parenteral vaccines in dogs for over 20 years, highlights the fact that current vaccines are not highly effective in preventing infection. On the other hand, our understanding of the role that *B. bronchiseptica* has in feline respiratory disease is only beginning to undergo scientific scrutiny.

**PATHOGENESIS OF INFECTION**  
Most of what is known about the pathogenesis of *B. bronchiseptica* is based on information derived from studies in dogs. In addition to dogs, infections have been documented in cats, pigs, various laboratory species, and humans. *B. bronchiseptica* rarely infects tissues outside the respiratory tract, a fact that supports the ease of transmissibility among dogs, particularly when housed in close quarters. Contributing to the ability of *B. bronchiseptica* to colonize respiratory epithelium is the fact that the bacterium possesses both fimbrial and non-fimbrial adhesins, that facilitate the attachment to host cells. Two non-fimbrial adhesins, filamentous hemagglutinin (FHA) and pertactin (Prn), are essential for the attachment of *B. bronchiseptica* to respiratory epithelial cells. Understanding the role of such proteins in the pathogenesis of *B. bronchiseptica* infection has been fundamental in investigations that may lead to the first acellular whooping cough vaccine. It is not unreasonable that such research may ultimately lead to improved vaccines for dogs and cats. Fimbriae, hair-like appendages extending from the cell membrane of *B. bronchiseptica*, recognize specific receptors within the respiratory tract. This allows *B. bronchiseptica* to colonize specific tissues where it then releases various exotoxins (such as adenylate cyclase-hemolysin and dermonecrotic toxin) and endotoxins that impair function of the respiratory epithelium (ciliastasis) and compromise the ability of the infected host to eliminate the infection. These potent toxins not only disrupt the protective action of the mucociliary apparatus, but also are believed to compromise phagocytosis and...
suppress both cellular and humoral immune responses. Additionally, *B. bronchiseptica* is regarded as an extracellular pathogen that has the unique ability to invade host cells. Once contained within the intracellular environment, bacteria are able to avoid immunologic defense mechanisms and establish a persistent infection (months) or carrier state.

**CLINICAL PRESENTATION**

Clinical signs of canine infectious tracheobronchitis (ITB) include paroxysmal coughing episodes, frequently associated with retching and expectoration, in an otherwise healthy, active dog. Swelling of the vocal folds, associated with laryngitis, can result in a loud, high-pitched cough often described as a “goose honk” or “seal honk.” Expectoration of mucus following an episode of retching or hacking behavior may be misinterpreted by the owner as vomiting. Anorexia, fever, and lethargy may be observed among infected dogs during an outbreak. The onset of clinical signs typically ranges from 3 to 10 days following exposure. In most clinical cases, the onset of clinical signs can be associated with recent exposure to other dogs or general anesthesia and endotracheal intubation. The ability to elicit a cough on manipulation of the trachea is an inconsistent clinical finding that should not be used exclusively to rule canine ITB in or out.

A second, more severe respiratory syndrome has been observed in dogs residing within kennel environments during an outbreak of ITB. Although cough may be present, the predominant clinical sign is associated with mucoid to mucopurulent nasal and ocular discharge. Pneumonia is likely to be a complicating factor and, in some cases, may become life threatening, particularly in puppies. In these cases, *B. bronchiseptica* has been isolated from the pharynx and trachea as a pure culture. Affected dogs are characteristically febrile, lethargic, anorexic, and may show some degree of respiratory distress or even dyspnea. Such cases are difficult to distinguish from those with bacterial pneumonia as well as non-bacterial causes of pneumonia. We have observed outbreaks to occur at any time of the year and may affect more than 50 percent of the dogs in a densely populated environment. Puppies are more severely affected and are at significant risk of dying if not treated.

The recent introduction of a topical (intranasal) vaccine against feline *B. bronchiseptica* has prompted concerns over the actual prevalence of feline respiratory infections caused by or associated with *B. bronchiseptica* and the indications for vaccinating. Unfortunately, there are few published reports that describe the clinical features and pathogenesis of *B. bronchiseptica* infection in cats. Among cats with confirmed infections, cough is the predominant presenting complaint. Unlike the dog, the character of the cough is neither unique-sounding nor inordinately loud. Age and housing may be important risk factors for infection. Most reports of severe respiratory infections associated with *B. bronchiseptica* involve multiple kittens (< 6 months of age) housed together. While the occurrence of *B. bronchiseptica* infection is likely to be greater among cats maintained in multiple cat households with a history of “respiratory disease”, the overall prevalence of infection within the cat population is not known. Serological surveys have shown rate of seropositivity range from 30% to as high as 85% in multiple cat households. Over 130 isolates of *B. bronchiseptica* examined by pulse field gel electrophoresis have been recovered from cats. Cats housed together were found to carry similar or identical strains and subtypes. The fact there was no reported difference in the electrophoresis patterns in isolates from
carrier cats and those with clinical infections implies that active infections are likely to be opportunistic. Furthermore, the likelihood of a particular isolate to produce disease may be related to host or environmental factors such as concurrent respiratory virus infection, crowding, and stress.

It is important to note, however, that the presence of *B. bronchiseptica* antibody in an individual cat is not indicative of active infection. Furthermore, *B. bronchiseptica* may be one of many resident bacteria in the oral cavity of healthy cats. Until additional information can be made available, specific recommendations for vaccination of cats against *B. bronchiseptica* will be difficult to make.

**DIAGNOSIS**
A clinical diagnosis of infectious tracheobronchitis is based on historical or physical examination findings that meet clinical criteria described above. In addition, a history of exposure to other dogs (whether or not they have signs of coughing) is helpful in establishing the diagnosis. A favorable and rapid response to empiric antibacterial and antitussive treatment supports the diagnosis of uncomplicated ITB. Routine thoracic radiography, hematology, and biochemistry profiles are neither diagnostic nor prognostic in uncomplicated cases. An inflammatory leukogram with significant leukocytosis or left shift may develop in dogs with a complicated infection associated with pneumonia. It should be noted that because of the large number of indigenous microflora in the canine respiratory tract, bacterial isolates from the nasal and oral cavities will not distinguish a primary infection from a secondary or opportunistic infection. In dogs with uncomplicated ITB, thoracic radiographs are typically unremarkable. Dogs with respiratory complications associated with ITB may have radiographic signs of pulmonary hyperinflation, interstitial pneumonia, and segmental atelectasis.

**TREATMENT RECOMMENDATIONS**
Treatment of *B. bronchiseptica* is centered around oral administration of an appropriately sensitive antimicrobial. However, it may be in the patient’s best interest to administer cough suppressants in the form of anti-inflammatory and/or antitussive drugs, particularly on a short-term basis.

**Antimicrobials**
Most cases of uncomplicated ITB can be regarded as self-limiting and do not necessarily require antimicrobial therapy. However, conventional practice standards include empiric, short-term administration of an antimicrobial to prevent opportunistic infections. Whether or not dogs with clinical signs of ITB are at significant risk of developing bacterial pneumonia has not been definitively established. On the other hand, evidence of a mucoid to mucopurulent nasal and/or ocular discharge justifies administration of an antimicrobial. Doxycycline, administered orally at 5.0 to 10.0 mg/kg, once daily, for a minimum of 2 weeks is the first choice of antibiotic due to its efficacy against *B. bronchiseptica*. However, the ability of *B. bronchiseptica* to persist in the respiratory tract of infected dogs for as long as 3 months justifies a treatment duration of up to 30 days, particularly when attempting to manage simultaneous infections in multiple dogs living in the same environment.

**Glucocorticoids**
Short-term administration of glucocorticosteroids, administered concurrently with an antimicrobial, is safe and effective in attenuating severe cough in dogs having an uncomplicated infection. Prednisolone can be administered at anti-inflammatory doses, 0.25-0.5 mg/kg, orally, once or twice daily, for up to 5 days as needed to control cough. Since some of the antimicrobials recommended in the treatment of canine ITB are bacteriostatic, concurrent use of glucocorticoids should not be extended beyond 5 days. It is recommended that antimicrobial therapy be continued for at least 5 to 7 days beyond the day that the corticosteroid is discontinued.

**Antitussives**
Antitussives alone and in combination with bronchodilators, have been recommended in the treatment of canine ITB. Either hydrocodone or butorphanol are recommended antitussives. In cases of ITB that are complicated by bacterial pneumonia, administration of narcotic antitussives is not recommended.

**Bronchodilators**
The benefits of bronchodilator therapy in dogs and cats with *B. bronchiseptica* infection remain unclear. At issue is whether or not the airway response to bacteria and viruses increases airway hyperactivity and baseline resistance to airflow. Two categories of bronchodilators are used in veterinary medicine: the methylxanthine derivatives and beta2-agonists. The beta2-agonists, terbutaline and albuterol, have been shown to be of benefit when administered to dogs with chronic bronchitis and are preferred when managing severe *B. bronchiseptica* infections. These drugs have the advantage of reducing cough as well as reducing pulmonary infiltrates associated with uncomplicated bronchitis. Excitability or tremors may be encountered during the first few days of treatment. The role of bronchodilators in treating cats with *B. bronchiseptica* infection has not been described and is currently not recommended.

**Aerosol Therapy**
In contrast to humidification therapy, aerosol therapy, also called nebulization, refers to the production of a liquid particulate suspension within a carrier gas, usually oxygen. Dogs and cats with ITB that derive the most benefit from aerosol therapy are those with excessive accumulations of bronchial and tracheal secretions and those with bacterial bronchial or pulmonary infections. Small, disposable, hand-held jet nebulizers are inexpensive and readily available through hospital supply retailers. Experience has shown that patients do benefit from aerosol therapy when from 6 to 10 ml of sterile saline is nebulized over 15 to 20 minutes, 1 to 4 times daily. Oxygen must be delivered at flow rates of 3 to 5 liters per minute to effectively nebulize saline. Aerosol therapy must be administered in the hospital and is generally administered over 1 to 4 days as needed to control respiratory signs. There is no value in nebulizing mucolytic agents. They can be irritating and induce bronchospasm. Nebulization of glucocorticoid solutions, such as methylprednisolone sodium succinate, have not been critically studied in veterinary medicine. Dogs unresponsive to oral or parenteral administration of antibiotics may respond to nebulized antibiotics.

**VACCINATION**
Several commercially licensed canine vaccines for protection against *B. bronchiseptica*, CAV-2, and CPIV are available. At this time, there is only one vaccine licensed for protection against feline *B. bronchiseptica* infection. Canine vaccines are available for topical (intranasal) as well as parenteral administration while the feline *B.
*B. bronchiseptica* vaccine is approved for topical (intranasal) administration only. The efficacy of vaccination administered by either the topical or the intranasal route is well documented. Regardless of the route of administration, vaccinated dogs experience substantially less coughing when compared to control dogs following challenge. Vaccination is not expected to completely eliminate the risk of infection and development of subclinical to mild infection following exposure. At issue, however, is whether or not sequential vaccination (i.e., administering a topical *B. bronchiseptica* vaccine and a parenteral vaccine during the same appointment) provides superior protection compared to either vaccine given alone. From the studies available at this time, it is suggested that sequential vaccination may, in fact, provide a superior protective response in seronegative puppies than either the intranasal or parenteral product alone. On the other hand, administration of an intranasal vaccine may not effectively booster young adult, seropositive dogs whereas subcutaneously administered vaccine do. There appears to be no benefit to administering both parenteral and topical vaccine to adult, seropositive dogs. Duration of immunity studies on parenterally vs. topically administered vaccines have not been compared. With regard to the onset of immunity, it is recommended that dogs be vaccinated at least 5 days prior to a known (or potential) exposure to ITB, e.g. being housed in a boarding kennel. Despite the assumption that topically administered vaccine provides the most rapid onset of immunity, it is not known if intranasally administered vaccine will immunize a susceptible dog in less than 5 days.

Recommendations outlined by the American Association of Feline Practitioners indicate that *B. bronchiseptica* vaccination in not required in all cats. Use of the vaccine is generally limited to cluster households and shelters where *B. bronchiseptica* is known to be associated with lower respiratory infection in cats. As occurs in dogs, transient post-vaccinal sneezing or cough is expected in some cats within 24-hours post-vaccination.

**PUBLIC HEALTH CONSIDERATIONS**

Human infection with *B. bronchiseptica* is most likely to occur in children and immunocompromised adults. Although infections are uncommon, at greatest risk are individuals whose immunosuppression is related to alcoholic malnutrition, hematologic malignancy, long-term glucocorticoid therapy, concurrent HIV infection, splenectomy, and pregnancy.

**FURTHER READING**

1. Ellis JA, Krakowka GS, Dayton AD, and Konoby C: Comparative efficacy of an injectable vaccine and an intranasal vaccine in stimulating *Bordetella bronchiseptica*-reactive antibody responses in seropositive dogs.
LEPTOSPIROSIS: THE 2-WAY? THE 4-WAY? ...OR THE “NO WAY”?

Leptospirosis is a gram negative, worldwide zoonotic infection. All mammals are susceptible. Transmission rates are very high with only 10 organisms needed to cause infection and disease. Almost all species of mammal are susceptible – there are over 250 serovars. All pathogens belong to the genus Leptospira. There is tremendous geographic variation in prevalence of serovars: The United States has 6-7, Latin America has 15-20, while Scandinavia has a number of unique serovars not typically seen in the US. In order to persist, leptospires need a maintenance host, a role that humans do not serve. The maintenance host (typically wildlife) does not become clinically ill despite the fact that organisms are sustained in the kidneys and shed months to years or for the life of the animal. Natural hosts do not make significant antibody titers when infected. Immediately following infection, there is a significant rise in Antibody followed by a rapid decline. In addition, leptospires can be maintained in the liver of wildlife hosts. Different species show differences in pathogenesis (raccoon more susceptible than dogs).

On the other hand, incidental hosts, ie, humans, are epidemiologically irrelevant in outbreaks because they are usually short term hosts with acute severe disease; all ages are susceptible. The organism is cleared as the host recovers and urinary shedding is short term. Incidental hosts, following infection, produce high titers and are relatively easy to detect if illness ensues.

The route of infection of leptospirosis is either direct or indirect through contact with urine (especially through mucous membranes). The actual infectious dose is not known, although the organisms are highly infectious and can spread through tissues rapidly. They can cross the placenta and infect fetuses. Any damaged skin allows entry.

Pathogenesis: within 15-20 minutes after being deposited in the eye – organisms can be found in blood. A low-level bacteremia develops after which it spreads to liver, kidney, spleen, etc. The antibodies that do develop are effective at clearing the organism from the tissues. In the incidental hosts the antibodies are so effective they can completely clear the organisms making diagnostic confirmation problematic. In addition, Leptospires can be transmitted across the placenta. In maintenance hosts – the antibodies clear the organisms from spleen, etc. but leave organisms in select sites like the kidney, which leads to long-term shedding.

Clinical Infections in Dogs: First described in 1899, in dogs, the serovars *L. icterohemorrhagiae* and *L. canicola* represented the predominant infecting serovars. Such infections were common in the US throughout the 70’s and 80’s. However, most authors agree that the frequent use of vaccines against these common serovars has effectively reduced the disease prevalence. In the mid to late 1980s, leptospirosis in dogs reemerged with the principle serovars being reported as: *L. grippotyphosa*, *L. pomona*, and *L. bratislava*.

Clinical presentations of infected dogs typically, although not always, involves young, large breed dogs presented for acute onset lethargy and significant fever (103° to 104° F). Muscle pain, vomiting, and dehydration are common. Acute onset icterus in a young, outdoor dog always places leptospirosis at the top of the differential diagnosis.
list. Other signs include bleeding diathesis, tachypenia, cardiac arrhythmia, and shock (vascular collapse). A sub-acute form of clinical leptospirosis is reported in dogs presented for renal failure, with no known predisposing cause. Affected dogs will also have high fever, myalgia and even hyperesthesia.

Hematologic findings typically include leukocytosis and thrombocytopenia. Several significant biochemical abnormalities may be detected in the same patient: azotemia (or uremia), elevated ALT and alkaline phosphatase, hyperamylasemia (with concurrent increase in lipase), and hyperbilirubinemia. Abdominal ultrasound exam may be consistent with intussusception, acute renal failure (large kidneys), and pancreatitis.

1. **Microagglutination Test (MAT)** – the ‘gold standard’ test used in the US and throughout the world, Human and Veterinary. Specific, but difficult to standardize, insensitive for some serovars, and vaccinated animals cause False + results. Dogs with Lyme don’t react to the test. Samples sent to 3 different labs will have 3 different titers. Maintenance hosts which are chronically shedding will be seronegative. Vaccination titers usually last only 60-120 days post vaccination and titers are less than 1:800. But, titers don’t correspond to immunity. **Diagnosis**: In chronically vaccinated animals, MAT titers can reach 1:1600. **Interpretation** – the highest titer is assumed to be the infecting serovar. Titers can go up after successful treatment so getting additional samples may confuse the picture. Antibiotics will kill organisms stimulating an immune system leading to high convalescent titers. Don’t re-test treated patients for at least 6 months following treatment.

2. **Urine Dark Field** – RARELY helpful...too many false + results ; not sensitive.

3. **Histopathology** – insensitive diagnostic test because biopsies are seldom representative of infected tissue.

4. **Culture** is the only technique in which can identify the serovar, difficult, expensive, takes 10 to 16 weeks depending on the serovar and is very hard, considering that the shedding period is only about 3 weeks after acute infection. Have to have urine before the animal has been treated.

5. **PCR** – sensitive (can find less than 100 organisms) – But, is not serovar specific. They have not been used in enough samples or compared to gold standards. Because it works in human blood does not mean it will work in dog urine.

6. **Acute & Convalescent Titers**: baseline titer is repeated in 1-2 weeks. Single antibody titer is difficult to establish without knowledge of vaccination Hx and clinical signs. A 4-fold increase in serial titers is diagnostic.

**Treatment** – No “initial” treatment with amoxicillin...just use doxycycline @ 5 to 10 mg/kg, PO, bid, for 2 weeks. Management of underlying kidney and/or renal disease is a must. 10-30% die. Post recovery: *L. canicola* – dogs shed from months to years. *L. icterohemorrhagiae* – dogs shed several months, other serovars – dogs shed 3 weeks to a month.

**Vaccination**: Is there a significant increase in the occurrence of canine leptospirosis to justify routine administration of vaccine to all dogs presented to the practice?
The answer is “NO”. Based on recommendations of the AAHA Vaccine Task Force, to be published in the 2006 Canine Vaccination Guidelines, leptospirosis is categorized as a NON-CORE (or “optional”) vaccine. It is regarded by many as the most reactive vaccine used in dogs and, as such, should only be administered to dogs with a reasonable risk of exposure or dogs that reside in geographic locations where confirmed cases of leptospirosis have been recognized.

Immunity is serovar specific (there’s NO cross protection); need at least 2 initial doses and boost once per year. Lepto vaccines are the most reactive vaccines. There’s a need for less reactive vaccines. Do we need to vaccinate? Yes...when and where the risk is established. Some have stipulated that there is a re-emergence of leptospirosis...still, cases are uncommon. There is no evidence that the 4-way vaccines are less reactive than the 2-way vaccines.

**Does zoonotic potential of Leptospirosis justify vaccination?** NO...not necessarily...vaccination protects the dog...vaccination does NOT prevent infection nor does it prevent shedding!
CANINE INFLUENZA VIRUS INFECTION

At Issue: Earlier this year, numerous television and newspapers reports addressed, in only ways that the media can do this, recent research findings that confirmed the fact the equine influenza virus ‘jumped’ species and had infected dogs. [Primary Investigators: Dr. P. Cynda Crawford, University of Florida, College of Veterinary Medicine, Gainesville, FL; Dr. Ed Dubovi, Cornell University, College of Veterinary Medicine, Ithaca, NY.]

What’s more, these reports cited concerns of widespread respiratory disease affecting, and even killing, dogs. And then...is this the forerunner of the impending avian influenza outbreak that (maybe) threatens humans throughout the world?? Will pet dogs threaten human health???

NOT QUITE!

The Facts:

The Clinical Disease: In January 2004 (correct...2004), an outbreak of respiratory disease occurred in 22 racing greyhounds at a Florida racetrack. Two clinical syndromes were reported:

1. a mild cough, with fever, lasting 10-14 days with subsequent recovery (14 dogs), and...

2. peracute death associated with extensive lower respiratory tract hemorrhage (8 dogs...36%) involving the lungs, mediastinum, and pleural space. Histology of the lungs revealed suppurative bronchopneumonia as well as bronchiolitis, and tracheitis.

In a summary statement to the CDC, Dr. Crawford points out that canine influenza is NOT a highly fatal disease, indicating that 80% of infected dogs develop nasal discharge, cough, mild fever and recover spontaneously. Several others will show NO CLINICAL SIGNS whatsoever. What makes this disease particularly problematic, clinically speaking, is the fact that it is contagious from dog-to-dog. Susceptibility rates, obviously, are very high.

Complicated infections are UNCOMMON and, unlike the human influenza A virus, are associated with bacterial infection (B. bronchiseptica would be a likely candidate!) of the lower respiratory tract.

Mortality in dogs is estimated to be from 6% to 8%.

Virus Identification: After considerable research, it was (recently) confirmed that interspecies transmission of an entire equine influenza A (H3N8) virus (documented as a cause of equine respiratory disease for over 40 years) to the dog [ie, the virus sequence corresponds with the H3 immunoglutenin and the N8 neurominidase subtype]. What’s more, investigators at U of FL examined archival lung tissue from greyhounds that died from hemorrhagic bronchopneumonia in March 2003. Sequence analyses of virus isolated from lungs indicated that viruses had infected greyhounds prior to 2004. Further studies comparing the equine and canine influenza viruses have shown that only 4 amino acid changes differentiate the two viruses.
Seroprevalence in Racing Greyhounds: From January to May 2005, blood from 96 dogs at 7 Florida racetracks was collected (acute and convalescent samples). 100% were seropositive. 100% of dogs (n = 25) in West Virginia racetracks were seropositive. Ten dogs in Wisconsin were also seropositive.

Occurrence of Canine Influenza in Pet Dogs: Blood samples collected from 70 dogs with respiratory disease in shelters in Florida and a variety of veterinary practices in Florida and New York City showed 97% were positive for antibody to the influenza virus. This study demonstrated that canine influenza virus infection was not unique to the greyhound breed. Today, investigators have ONLY confirmed canine influenza in dogs in Florida, New York (City) and possibly Massachusetts.

The Conclusion: Identification of infected dogs in widespread geographical locations from 2003 to 2005, support the conclusion that a single virus transmission event from horses to dogs occurred. What’s more, horizontal spread of the adapted virus from dog to dog was documented.

Transmission: Experimental studies in dogs suggest that virus will persist in the nasal cavity and oropharynx of challenged dogs and suggests that shedding is possible. Dog-to dog transmission could occur via large aerosolized droplets from the upper respiratory tract, fomites, or direct mucosal contact.

Clinical Diagnosis of Canine Influenza Virus: Serum Antibody titers (acute and convalescent) is the primary tool used to establish a diagnosis in dogs. However, a commercial test is not currently available. U of Florida and possibly Cornell are the only facilities that are working on virus identification. Therefore, prior permission to send serum or tissue (lung preferably) from dogs that died of acute, hemorrhagic respiratory infection would be warranted.

Treatment: Supportive care with a broad spectrum antimicrobial is indicated to manage the risk of secondary bacterial bronchopneumonia (suggest: doxycycline, amoxicillin-clavulanic acid, azithromycin, or a fluoroquinolone). The accumulation of fluid in the pleural space, although regarded a grave prognostic sign, should be removed via thoracentesis. Care should be taken to properly dispose of materials (endotracheal tubes, catheters, needles, syringes, oxygen tubing, etc.).

Vaccine: No vaccine is currently available...but count on one becoming available in the near future. Important...there is NO cross protection between the parainfluenza virus vaccine or adenovirus-2 vaccine in protecting against canine influenza.

NOTE: a recombinant equine influenza vaccine (parenteral) has been licensed in Europe (Merial) and is expected to be approved for use in horses in the US in the near future. HOWEVER, there is no evidence that this vaccine is immunogenic in dogs.

Zoonotic potential: There is NONE. Although the authors of this study added: “evidence of canine influenza infection in pet dogs, a primary companion animal for humans, raises the possibility that dogs may provide a new source for transmission of novel influenza A viruses to humans. “ ....there is absolutely no evidence even suggesting that dog-to-human transmission has occurred....nor does it suggest that zoonotic transmission will occur.
The canine influenza virus is so close to the equine influenza virus, 4 amino acids, (and the equine influenza virus has NEVER infected humans in 40 years), investigators have indicated that the canine influenza virus may even be LESS likely than the equine virus to infect humans.

Recent concerns over the avian influenza A virus that has also jumped species, infected humans, with the subsequent documentation of (limited) human-to-human transmission has fueled concerns over the ability of the canine influenza A virus to infect humans. The viruses are, based on the classification of influenza viruses, somewhat distant in their species specificity. Unlike the canine influenza A (H3N8) virus, the avian influenza virus (H5N1) causes acute and hemorrhagic respiratory disease, but pleural effusions and secondary bacterial pneumonia are not characteristic.