The conditions of aspiration pneumonitis and aspiration pneumonia have been well described in both humans and dogs. Aspiration is the inhalation of gastric or oropharyngeal contents into the respiratory tract. Aspiration pneumonitis is the result of direct chemical injury to the airway following inhalation of gastric acid and particulate matter. It can develop into aspiration pneumonia with secondary bacterial colonization and the resultant inflammatory response. The pathophysiology of both aspiration pneumonitis and pneumonia are similar with the initiation of a localized inflammatory cascade resulting in impaired respiratory function.

Aspiration pneumonia develops in three phases with the first two making up aspiration pneumonitis alone. Phase I is the acute airway response which occurs immediately after aspiration, followed by Phase II, the inflammatory phase, and finally Phase III, bacterial colonization of the airways and pulmonary parenchyma. Damage to the airways during Phase I occurs as a direct consequence of exposure to acidic stomach contents. The chemical tissue damage triggers the activation of pro-inflammatory mediators leading to alveolar cell necrosis, bronchiolar constriction, increased mucus production, increased vascular permeability and pulmonary edema. Phase II begins 4 to 6 hours after aspiration and can last for as long as 12-48 hours. This phase is characterized by the infiltration of neutrophils into the alveoli and pulmonary interstitium. During this phase there is ongoing leakage of proteins leading to the development of high protein pulmonary edema, neutrophil sequestration and activation, which further feeds the inflammatory cascade, eventually leading to alveolar collapse and atelectasis.

Phase III constitutes the difference between aspiration pneumonitis and aspiration pneumonia, and involves the bacterial colonization of the airways and pulmonary parenchyma. The bacteria are often commensal organisms such as *Staphylococcus spp.*, as well as *E. coli*, *Klebsiella*, and oropharyngeal *Mycoplasma sp*, in addition to primary respiratory pathogens including *Pasteurella*, *Pseudomonas*, *Bordetella*, and *Streptococcus spp*. Gastric acid aspiration enhances bacterial adherence to the respiratory epithelium and reduces pulmonary clearance of bacteria. Ultimately, the vascular permeability changes lead to pulmonary edema formation, focal atelectasis and collapse of alveoli, resulting in hypoventilation and shunting.

This process of aspiration pneumonitis and pneumonia have been well-described in both humans and dogs. In humans, the preliminary diagnosis is made by either witnessing the aspiration event, detecting gastric contents in the airways, and/or by the development of acute respiratory
distress within hours of vomiting, regurgitation or anesthesia. Known risk factors in humans are conditions that impair protective airway reflexes such as coma, head trauma, sedation/anesthesia, muscle relaxants, seizures, airway trauma or other laryngeal/pharyngeal dysfunction. Also, anything that causes large volumes of intragastric food or fluid can lead to aspiration, such as reasons for delayed gastric emptying (ileus, bowel obstruction, pain, opioids, obesity and pregnancy), overfeeding by enteral tube or a recent meal prior to anesthesia/surgery. Abnormalities of esophageal function such as megaesophagus, reflux esophagitis and myasthenia gravis are also known contributors.

Dogs have been reported to have many of the same predisposing risk factors as humans, with the additional major category of vomiting and regurgitation. Dogs that have recently been sedated or undergone general anesthesia are at risk for aspiration. Premedication with opioids can predispose patients to gastric reflux, regurgitation and possible aspiration. Neurologic conditions that affect esophageal or laryngeal function (laryngeal paralysis) as well as head trauma or seizures also predispose patients to aspiration. A presumptive diagnosis of aspiration pneumonia in dogs is based on the history, physical exam findings and radiographic findings consistent with aspiration pneumonia. Often the history includes a predisposing condition but the actual aspiration event is rarely witnessed. Owners may report regurgitation, coughing, panting or labored breathing, as well as other nonspecific signs including lethargy and inappetence.

Physical examination findings often include fever, tachypnea and/or dyspnea. Thoracic auscultation may reveal increased lung sounds, wheezes, crackles or dull lung sounds. Retrospective studies in dogs have demonstrated that 31-57% of dogs with aspiration had a normal rectal temperature, 58% had a normal respiratory rate and 28-31% had normal lung sounds at the time of diagnosis.

Thoracic radiography is the gold standard for preliminary diagnosis. Three-view radiographs are advised because multiple lung lobes may be involved. According to the results from a retrospective study of 88 dogs, the right middle, right cranial and the caudal subsegment of the left cranial lung lobes were most frequently affected in 48%, 38%, and 38% of cases respectively. This can be explained from an anatomical perspective, as the main lobar bronchi leading to each of these three lobes are the most proximal and dependently-positioned pathways. In most patients, more than one lung lobe was affected with an average of 1.9 lung lobes involved. Predominantly alveolar infiltrates were noted in 74% of dogs and a predominantly interstitial pattern in the other 26%.

Definitive diagnosis is made on positive cultures taken via airway sampling such as tracheal wash or bronchoalveolar lavage. The reported sensitivity of wash cultures has been reported to be between 44 and 90%. Specificity is typically reported to be low, often due to contamination
from the oral cavity. The bacteria identified in positive cultures are often commensals of the oropharyngeal cavity. Species isolated are *E. coli*, *Pasteurella*, *Staphylococcus*, *Streptococcus*, *Klebsiella*, *Enterococcus* and *Mycoplasma*. In most cases, infections are mixed although single-agent infections can occur. Anaerobic bacteria are rare unless pulmonary abscessation or other nidus of infection is present.

Antimicrobials are the gold standard for treatment of aspiration pneumonia, and additional supportive care including oxygen supplementation, fluid therapy and nebulization and cupage is often indicated. Broad spectrum antibiotic therapy including coverage for both gram negative and gram positive bacteria should be initiated while culture tests are pending. Airway cytology with gram stain can be of benefit for early directed antibiotic therapy.

Overall, dogs diagnosed with aspiration pneumonia have a fair to good prognosis for survival with supportive care, with reported survival rates of 77-82%. Interestingly, survival has not been shown to be related to the character or number of predisposing etiologies, or to the severity of radiographic signs. Recurrent aspiration from chronic diseases such as laryngeal paralysis however, may contribute to an owner's decision to euthanize.

Currently there are no published studies that have characterized aspiration pneumonia in cats. It is thought to happen very infrequently, possibly due to better inherent airway defense mechanisms, such as the strong laryngospasm reflex encountered during endotracheal intubation. There have been few publications describing feline patients with aspiration pneumonia. Some of these have been cats with documented laryngeal disease such as laryngeal neoplasia and laryngeal paralysis. Aspiration pneumonia has also been reported in cases with neurologic deficits including feline dysautonomia and cases of permethin toxicity.

Retrospective evaluation of cases of cats with aspiration pneumonia was recently performed at MSU. The incidence of feline aspiration pneumonia cases was low, with 28 cases that met our study inclusion criteria identified over a 12 year period. Data to describe the clinical syndrome was collected, including patient signalment information, predisposing factors, radiographic findings, laboratory data and survival rates.

The most common presenting complaints were dyspnea (28.5%), anorexia (25%), vomiting (21%) and lethargy (18%). Cats were also presented for cough (14%), drooling/oral bleeding (11%), regurgitation (7%), weight loss (7%), diarrhea (3%) and known neurotoxin exposure (3%).

Predisposing factors included vomiting (43%), recent anesthesia (39%), concurrent upper respiratory signs including nasal sinus disease (21%), concurrent enteral nutrition (18%),
esophageal disease (14%), previously diagnosed gastrointestinal disease including inflammatory bowel disease (7%), neurologic disease (7%) and one case of laryngeal trauma (3.5%).

Survivors were defined as cats who were discharged alive from hospital. 89% of cases survived to discharge, with the 11% non-survivors including cases that both died and were euthanatized.

While previously thought to not occur significantly in cats, here we identify that the process of aspiration pneumonia does occur in cats for many of the same reasons as it does in both people and dogs. This is something that we should watch closely for in at-risk patients, especially any cat or dog who develops a fever or respiratory signs following anesthesia or vomiting.

REFERENCES:

5. Amis TC and McKiernan BC. Systematic idenfication of endobronchial anatomy during bronchoscopy in the dog. AJVR 1986; 47(12):2649-2657.