

Veterinary Detectives: Solving the Mystery of Respiratory Diseases in Dogs and Cats

Written by Jill Shuman

Diseases of the upper and lower airway can constitute a challenge for veterinarians. While clinical signs such as coughing and respiratory distress are typically caused by primary problems of the respiratory tract, they may also occur secondary to disorders of other organ systems or even to an allergic response.

Carol Reinero, DVM, DACVIM (SAIM), PhD, and Isabelle Masseur, DVM, DACVR, PhD, both of the University of Missouri College of Veterinary Medicine, Columbia, Missouri, USA, presented a case study of a dog with an unusual set of respiratory symptoms. They then walked through the diagnostic steps required to identify and manage the animal's respiratory disease.

Dr. Reinero began by reminding the attendees that a thorough physical examination is always the first step in determining why an animal might be in respiratory distress. The physical examination begins with a distance examination—which includes an auditory examination performed without a stethoscope. Here the clinician can determine whether the animal has a noisy or quiet breathing pattern and the precise character of the noise (stertor, stridor, or wheeze). A visual examination should follow to determine whether the animal is in inspiratory distress, expiratory distress, or both or exhibits another feature, such as paradoxical breathing.

A hands-on examination should follow, with the clinician listening to the heart for any arrhythmias, murmurs, or muffled heart or lung sounds. Pulses should be felt simultaneously, followed by auscultation of the lungs for any abnormal (adventitious) sounds that consist of wheezes, crackles, and increased bronchovesicular noises. To differentiate true upper airway sounds from referred upper airway noises, Dr. Reinero suggested placing the stethoscope over the trachea and comparing the volume and character of the sounds as the stethoscope is toggled to the lung. If character and pitch of the 2 sounds is the same but the volume is greater at the trachea than the lungs, the noise is considered to be true upper airway noise and referred to the lungs.

In clinical practice, it is important to identify the animal's breathing pattern because different patterns are associated with different sets of possible conditions. With merely the distance and hands-on examinations, the 4 distinct patterns can each be categorized and directly and immediately recognized by a specific set of accompanying symptoms (Table 1). Of note, there are 4 other categories of respiratory distress that require additional diagnostic testing: pulmonary parenchymal disorders, pleural cavity diseases, pulmonary thromboembolism, and look-a-like disorders (diseases outside the respiratory tract not causing hypoxemia but presenting as respiratory distress—eg, severe anemia, hyperthermia, pain, acidosis, administration of opioids).

Dr. Reinero and Dr. Masseur then presented the case study of Lucy, a dog admitted to the clinic with likely lower airway obstruction associated with a loud wheeze, as well as a grade III/IV systolic apical murmur. Various differentials for the expiratory respiratory distress with wheeze included intrathoracic tracheal collapse, hilar lymphadenopathy, an intra- or extraluminal mass

Table 1. Identification of Breathing Patterns by Accompanying Symptoms

Inspiratory respiratory distress, stridor	Upper airway obstruction
Expiratory respiratory distress, wheeze	Lower airway obstruction
Evidence of trauma, broken segment of ribs, focal paradoxical respirations	Flail chest
Quiet inspiratory distress, severe abdominal enlargement	Pregnancy, ascites, pyometra

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impinging on the large airways, or diffuse small airway narrowing (ie, diffuse bronchomalacia). Differentials for the heart murmur included structural degenerative valve disease, cardiomyopathy, fever, or anemia.

The first series of laboratory tests included pulse oximetry, thoracic radiographs, an echocardiogram, complete blood chemistry (CBC), a chemistry panel, and a urinalysis. Pulse oximetry and CBC revealed hypoxemia and pronounced eosinophilia, respectively.

Dr. Masseau then reviewed results from the thoracic radiographs. These suggested severe, diffuse, bilateral bronchial thickening and some patchiness in the left caudal lung field, which are typically associated with chronic bronchitis. Echocardiography revealed that Lucy had moderate to severe mitral valve regurgitation and mild tricuspid valve regurgitation; the right and left atria were normal in size. She also had mild pulmonary hypertension that was deemed not clinically relevant.

Dr. Reinero and Dr. Masseau then discussed the discrepancy between Lucy's clinical symptoms and the laboratory tests. According to Dr. Reinero, hypoxemia and labored respiration (wheezing) are not typically associated with chronic bronchitis, nor did Lucy have the cough typical of chronic bronchitis. To further investigate, computed tomography (CT) scan with contrast, tracheobronchoscopy, and bronchioalveolar (BAL) lavage were performed. These latter 2 tests can provide information about what dynamic changes are occurring with airway caliber, with the animal intubated and breathing. A sample of the bronchial fluid was also obtained for cytology and culture.

Lucy's CT scan showed thickened bronchi diffusely but without true interstitial lung disease or involvement outside the airway. The trachea was diffusely reddened with prominent vasculature and grade 1 tracheal collapse with 25% narrowing of the tracheal diameter. The bronchi showed diffuse erythema, severe dynamic collapse, and increased mucus; BAL cytology revealed eosinophilic inflammation. Dr. Reinero and Dr. Masseau attributed Lucy's wheezing to dynamic collapse of the distal bronchial airways; as the dog exhaled, her airways completely closed. There were also large amounts of mucus throughout the lung and thickened airway walls, both contributing to the narrowed airway caliber.

Based on results from the CT scan and the tracheobronchoscopy, the diagnosis was acute eosinophilic bronchitis warranting additional testing for the cause of the eosinophilia. While there are myriad causes of eosinophilia, it often represents an allergic reaction or a parasitic infection [Kuehn NE, ed. Allergic pneumonitis in small animals: etiology. In: Aiello SE et al, eds. *Merck Veterinary Manual*. 10th ed (online). 2013]. To confirm

the underlying cause, the doctors checked Lucy for heartworm infection and various other fecal parasites; all test results were negative. Because dogs can develop eosinophilic bronchitis secondary to allergies to inhaled substances, Lucy underwent testing for serum allergen-specific immunoglobulin E testing. She subsequently tested highly reactive to 2 species of house dust mites.

Lucy was treated with oxygen support while she was hypoxemic, in addition to prednisone at a dose of 2 mg/kg/d, which resolved the hypoxemia and caused dramatic improvement in clinical signs. Other ancillary treatment included saline nebulization, followed by coupage to loosen the lung mucus and an empiric course of fenbendazole in case she harbored an unidentified parasite.

Other environmental strategies were recommended, including minimizing exposure to cigarette smoke, aerosols, dusts, and other environmental irritants; maintaining an ideal body condition score; and avoiding triggers (eg, excitement, exercise) that might exacerbate her condition. One week later, radiographs indicated that Lucy's lungs had dramatically improved, with reduction of the peribronchial thickening. Clinically, she no longer had evidence of labored breathing. When the results of the serum allergen-specific immunoglobulin E test returned, the owners were instructed how best to reduce the dust mite exposure (Table 2).

The long-term medical plan was to perform a slow taper of the prednisone over 6 to 8 months. If Lucy remains asymptomatic off steroids, a BAL could be repeated to determine if the acute disease had been cured. If this second BAL still showed evidence of inflammation, the bronchitis would now be considered chronic and incurable but treatable with lifelong low-dose steroids.

Table 2. Strategies to Reduce Dust Mites in the Home

Maintain humidity < 50%
Reduce the number of house plants, which increase humidity
Regularly change heating and air-conditioning filters
Replace heavy drapes with washable window coverings
Steam clean mattresses, pet bedding, and upholstered furniture
Switch to mattress covers and pet bedding made of microporous fabric (dust mite covers)
Eliminate decorative stuffed pillows and toys
Use pillows and bedding without feathers
Wash bedding weekly in hot water
Remove carpets where possible
Vacuum all carpets and upholstery at least weekly