Coughing dog with a murmur...what to do next?

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A dog presents with a cough and on auscultation a murmur is detected. This can be a clinical challenge: is the murmur due to heart disease, lung disease or both? Add to this puzzle is the dog with the above problems presenting in respiratory distress. By following some basic guidelines the mystery of the coughing dog can be sorted out. Age, breed and respiratory patterns can help sort out cardiac from pulmonary disease.

Clinical considerations

Dogs presenting in respiratory distress can be a challenge and be very stressful for all involved. Unfortunately they will not tell you why they are in distress. Stabilization is often necessary before a diagnosis can be determined; the patient is too fragile to be put through diagnostic tests. It is important to recognize respiratory patterns to help sort out differentials. Rapid shallow breathing patterns (i.e. low tidal volume) is usually found with pleural space disease. Dyssynchronous pattern or paradoxical breathing pattern is characterized by opposing motions of the chest and abdominal wall during inspiration and expiration. During inspiration, the caudal ribcage collapses inward while the abdomen expands outward. This can occur with a variety of respiratory disorders but in the dog typically implies pleural space disease.

Pulmonary parenchymal disease can have normal to increased respiratory rate but increased respiratory effort is noted (often described as dyspnea or labored breathing). These respiratory patterns are often accompanied by specific postures. Dogs in respiratory distress often adopt an orthopneic posture--elbows abducted and neck extended. Reluctance to lie down is often seen. Also note dogs in respiratory distress are not interested in the surrounding environment or the owners; all effort is concentrated on breathing.

History is very important to sorting out the cause of the cough and labored breathing. Congestive heart failure in the dog is preceded by several days of progressive coughing and exercise intolerance. If a small breed dog, the presence of a murmur before presentation is helpful. The cough is described as a wet cough and often the pet may be seen swallowing shortly after coughing. Coughing due to non-cardiogenic causes is often chronic and described as a dry or hacking cough. Surprisingly, many pneumonia cases do not have coughing as a primary complaint. A history of vomiting or regurgitation accompanies most aspiration pneumonia cases.

Most of our patients with murmurs are middle to older age. A young patient presenting with a murmur should prompt the consideration of a congenital or developmental condition. Finding a murmur in a juvenile pet should enable the practitioner to narrow down the possibilities based on the intensity and location of the murmur, breed and age of the pet. Remember that congenital does not necessarily imply genetic or inherited.

Once a murmur has been detected, diagnostic work up, regardless of age, should include radiography and an ECG, which can be done in most clinics. Ideally an echocardiogram completes the picture. Older dogs presenting with acquired murmurs tend to fall into two basic categories: small breed dogs and large breed dogs. For the most part, small breed dogs with a

murmur have endocardiosis leading to mitral +/- tricuspid insufficiency. Large breed dogs with a murmur tend to suffer from dilated cardiomyopathy (DCM) although they, too, can have chronic mitral valve disease (CMVD). Cocker spaniels tend to be the middle breed: they can have either CMVD or DCM. Here is a general rule for small dogs: a small dog with a cough who you suspect is suffering from congestive heart failure SHOULD HAVE A MURMUR. No murmur, no heart failure. Frustratingly, some dogs with DCM do not have a murmur, even when presenting in heart failure (Doberman Pinscher is a classic example of this).

Initial physical exam may be brief if the patient is in severe respiratory distress. In addition to the observation of respiratory patterns, mucous membrane color, capillary refill time, thoracic auscultation, femoral pulse quality and rectal temperature can all be evaluated quickly. Then the patient needs oxygen therapy +/- anxiolytics.

Initial empirical therapy

Oxygen can be provided by a variety of means. Flow-by, face mask, nasal prongs, oxygen collar and oxygen cage are all options. Oxygen concentrators can be used instead of depleting the hospital's supply of tank oxygen. Human infant incubators with flow-by oxygen into them, fish tanks or plastic bins can also be used for short term oxygen supplementation. Issues with these for long term use include carbon dioxide build up and hyperthermia.

Anxiolytics come in many forms. It is important to use those that have minimal cardiovascular effects. Butorphanol (0.2-0.4mg/kg) is an excellent mild sedative for respiratory distress cases. Diazepam or midazolam is also another anxiolytic medication that may help the pet to settle down and allow for more diagnostic testing. Acepromazine is not an appropriate anxiolytic in an emergency setting as it is not reversible, can last 6-8 hours and may cause issues if cardiopulmonary arrest occurs.

Radiology of the Heart

The assessment of heart enlargement is both subjective and objective. True to form most radiologists depend on the their experience for a subjective interpretation. More objective criteria include intercostal spaces and % width of the chest. These depend on a normal full inspiratory effort, which is often inhibited by underlying pulmonary, pleural or chest wall diseases, pain and obesity. The vertebral heart scale is a useful objective system of assessing dog and cat hearts. For cats I measure the maximum width on the VD, which should not exceed 4 vertebra. For dogs the combined length and width on the lateral view should not exceed 11 vertebrae. These criteria are most useful for overall cardiomegaly. For specific chamber or side of enlargement the heart face analogy is very useful. Some misused criteria include "reverse D" shape, which is normal (gentle almond?) and sternal contact, which again is a normal finding.

In dogs, unlike cats, the pulmonary veins often become enlarged with moderate to severe pulmonary congestion. This is evidenced by disparity between veins and arteries or when the veins exceed the width of the proximal 4th rib measured at the level where they cross this rib on the lateral projection or the 9th rib on the VD/DV view.

Radiology of the lungs

The most important premise to the conclusion that the enlarged heart is causing the lung opacity associated with cardiogenic pulmonary edema, is the interpretation of an alveolar lung pattern in the perihilar region. Often more opaque on the right than left side, this is often easily seen on the DV or VD projection. This pattern begins, often subclinically, as an interstitial

pattern. The capillaries that weep with excessive hydrostatic pressure associated with pulmonary venous congestion live in the interstitial space. With increased time or severity the fluid weeps out of the interstitial space and into the adjacent alveoli. The fluid-filled alveoli and terminal bronchi illicit a cough response.

How do we interpret an alveolar pattern? The most encompassing sign is border effacement. Lobar sign, air bronchograms and loss of distinctness of borders of the adjacent heart, pulmonary vessels, and diaphragm are all examples of this effect. The regional distribution of perihilar edema seems highly specific for heart failure.

Alveolar disease in other regions is much less commonly associated with heart failure in dogs, although quite common in cats. Ventral, middle and cranial distributions are seen with bronchopneumonia. Patchy widespread alveolar disease can be due to bleeding nodules or inflamed granulomatous diseases. Blood can accumulate either dependently (like bronchopneumonia) or locally in the case of contusions. Noncardiogenic causes of edema, including cerebral edema, may be caused by head trauma, seizuring or neck injury and have a specific very dorsal and caudal distribution. Other causes, including vasculitis, DIC, ARDS and toxicities are more widespread and variable.

Conclusions

Left-sided cardiomegaly causes cough by two mechanisms; 1) static compression of the left mainstem bronchus caused by left atrial dilation and/or 2) pulmonary edema associated with a perihilar alveolar pattern caused by excessive pulmonary congestion. These are both associated with left-sided heart enlargement. Noting this enlargement and the concurrent effects are important aspects of associating a heart murmur with a patient's cough. If the association seems weak, the pattern of heart enlargement or lung opacity does not fit these causes, then strongly consider a second, unrelated disease and treat appropriately.