ACUTE PULMONARY EDEMA:
5 STEPS TO SUCCESS

Rebecca L. Stepien DVM, MS, DACVIM
School of Veterinary Medicine
University of Wisconsin, Madison, WI

Acute pulmonary edema is a terrifying situation for both patient and clinician. The progression from shortness of breath to dyspnea to outright “air hunger” can occur rapidly and lead to the patient’s prompt demise. Rapid recognition and therapy is necessary to save the patient with acute pulmonary edema.

FIVE STEPS TO SUCCESS

1. **Recognize that you must keep the patient alive until the medications can have an effect.** This usually requires some type of oxygen supplementation, ranging from “blow-by” oxygen to acute anesthesia and ventilation in patients with marked respiratory fatigue. In addition, the patient must be kept in an unstressed environment with as much “hands-off” therapy as possible. The typical time to onset of hemodynamic effect of IV furosemide is 5-15 minutes. Lack of urine production within 30 minutes is an indication for additional dosing, but if a litter box is not provided for a cat or a dog is not removed from the cage, increases in urine production may not be noticed until an opportunity for voiding is available. The level of stress increases significantly in the “frustrated” urinator. *Palpate the bladder!*

2. **Recognize that not all apparent pulmonary edema is due to primary heart disease.** Acute administration of colloids or large doses of corticosteroids can precipitate pulmonary edema in animals with subclinical cardiovascular abnormalities, as can acute stress, humid weather or extreme exercise. This “iatrogenic” pulmonary edema usually responds to furosemide therapy. Alternatively, lack of response to “typical” therapy for pulmonary edema may indicate non-cardiac causes of pulmonary edema (e.g. acute respiratory distress syndrome) or non-edematous conditions that look like edema clinically and radiographically (e.g. pulmonary hemorrhage, diffuse neoplasia).

3. **Be aggressive with furosemide therapy.** Initial dose should be administered intravenously (2-6 mg/kg per dose). Because furosemide has direct venodilating effects if given IV, immediate decreases in left atrial pressure can be obtained even before diuresis can begin. Secondly, the sluggish circulation available to the GI tract during acute heart failure may limit absorption and distribution of orally administered medications. If an intravenous route is not available, administer furosemide intramuscularly in main muscle groups. Furosemide given by continuous rate infusion (CRI) can be given in lower doses continuously (0.66 mg/kg IV loading dose followed by 0.66 mg/kg/hour), providing constant medication with less rebound activation of the renin-angiotensin-aldosterone system. Furosemide given as a CRI provides more diuresis and less potassium loss than repeated IV boluses of furosemide, but may be less effective once the animal begins to become dehydrated.

4. **Use multiple medications for acute pulmonary edema.** Adding a potent vasodilator or positive inotrope can augment the effect of IV furosemide. For animals with systolic failure (e.g. dilated cardiomyopathy), nitroprusside (1-10 µg/kg/min, start at 1 µg/kg/min and increase to effect) acts as an arterial dilator and acutely reduces cardiac afterload, allowing increased forward flow to aid in reducing left atrial pressures. In animals with diastolic dysfunction (e.g. cats with hypertrophic cardiomyopathy) where too-rapid decreases in left atrial pressure can critically reduce cardiac output, a dobutamine CRI (cats: 0.5-2 µg/kg/min, dogs: 5-20 µg/kg/min) can augment diastolic relaxation and aid ventricular filling, thereby reducing left atrial pressure.

5. **Watch for side effects.** Acute administration of high doses of furosemide can significantly decrease serum potassium and magnesium concentrations within hours and supplementation may be required. Acute dehydration is a necessary result of therapy of acute pulmonary edema, but should be closely monitored as the edema clears and rectified as soon as possible. Sodium nitroprusside can cause severe hypotension at low doses and blood pressure should be monitored via intra-arterial catheterization, with a target MEAN arterial pressure of 65-75 mmHg.

REFERENCE