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Noncardiogenic Pulmonary Edema

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Abstract

Pulmonary edema may develop secondary to several cardiogenic and noncardiogenic conditions. Cardiogenic pulmonary edema (CPE) is associated with heart disease, an elevation in left atrial pressure, and an increase in pulmonary venous and capillary pressures. In contrast, noncardiogenic pulmonary edema (NCPE) can occur without pathologic cardiac disease and an elevation in left atrial pressure. NCPE has been associated with an increase in capillary membrane permeability with or without an increase in hydrostatic pressure. Signalment, history, and thoracic radiography may help distinguish NCPE from CPE. Some types of NCPE are self-limiting, and treatment may be largely supportive; others may require pharmacologic intervention and advanced respiratory support.

*Pulmonary edema is defined as a pathologic accumulation of fluid in the extravascular space of the lung.¹ It is typically categorized as cardiogenic or noncardiogenic. The term *noncardiogenic* is used for all nonidiopathic cases of pulmonary edema that are not the direct result of cardiac disease and subsequent elevations in left atrial pressure; these patients have a pulmonary capillary wedge pressure (PCWP) <18 mm Hg^{2,3} (**BOX 1**).*

Box 1. Causes of Noncardiogenic Pulmonary Edema

Postobstructive

- Choke
- Strangulation
- Laryngeal disease

Neurogenic

- Traumatic brain injury
- Seizures
- Electrocutation

Acute lung injury/acute respiratory distress

- Systemic inflammatory response syndrome
- Sepsis
- Pancreatitis
- Neoplasia
- Pneumonia
- Uremia
- Parvovirus

Near drowning/submersion injury**Smoke inhalation****Adverse drug effects**

- Anesthetic drugs (e.g., ketamine)
- Thiazides
- Cisplatin (in cats)

Anaphylaxis**Oxygen toxicity****Pulmonary embolus**

Conditions that cause pulmonary edema are relatively common in veterinary medicine. Previously, noncardiogenic pulmonary edema (NCPE) was defined as a type of permeability edema, but experimental studies support the association of some cases of NCPE with local elevations of hydrostatic (i.e., vascular) pressure, not just changes in vascular or epithelial permeability. Cardiogenic pulmonary edema (CPE) and NCPE both cause interstitial edema, which is associated with perivascular and peribronchial expansion and increased lymphatic flow. Interstitial edema may progress into alveolar edema with alveolar flooding and secondary respiratory compromise.⁴ Treatment options and prognosis vary greatly based on the underlying disease.

Pulmonary Anatomy and Physiology

To properly diagnose and support patients with pulmonary edema, it is essential to understand the Starling forces at work in the pulmonary capillary-alveolar space and the lungs' innate defenses against the accumulation of edema.

Within the normal lung, fluid constantly moves between the compartments of the capillary-alveolar space due to a higher net permeability of the capillary endothelium compared with other tissues. The capillary-alveolar space has three anatomic regions: the alveolar wall, the capillary endothelium, and the intermediary interstitial space. Alveoli are small air sacs with thin walls comprising type I pneumocytes. The tight junctions between the cells are permeable mainly to gases and water. A large force is required to damage these tight junctions and allow nonselective movement of solutes. In contrast, the junctions between the capillary endothelial cells are loose

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(highly permeable), which allows for filtration of fluids and colloids. The interstitial space contains connective tissue, fibroblasts, macrophages, small arteries, veins, and lymphatic channels.⁴⁻⁶ As lymph drains from alveoli to the hilus of the lungs, the net hydrostatic pressure in the pulmonary interstitium decreases and the potential space for fluid accumulation increases.

Starling Forces

Under normal conditions, fluid movement is governed by the equilibrium between the net force of the capillary hydrostatic pressure and the net force of the capillary oncotic pressure. Net capillary hydrostatic pressure is the driving force for fluid flux out of the capillary, and net capillary oncotic pressure is the driving force to keep fluid within the capillary. Albumin and its associated sodium ions are the main solutes contributing to oncotic pressure both inside and outside the capillary.^{7,8} The Starling equation describes these forces at work (**FIGURE 1**).

Compared with skin or muscle tissue, the pulmonary capillary endothelium is more permeable to albumin, and a strong oncotic gradient between intravascular and interstitial spaces cannot be maintained. This inherent “leakiness” allows the lungs to be more resistant than peripheral tissues to edema secondary to low oncotic states, such as hypoalbuminemia. Increases in net pulmonary capillary hydrostatic forces are more likely to cause fluid flux out of the capillaries given that the pulmonary capillaries have a very low pressure gradient in normal conditions.^{7,9} Overall, the net Starling forces of the lung favor reabsorption of water into the intravascular space, keeping the lungs “dry.” Under normal circumstances, the lung efficiently keeps water in the intravascular space despite the low interstitial oncotic pull by matching it against a small hydrostatic “push.”

Although the Starling equation is useful for explaining fluid movement, it is of relatively limited clinical use because many of the variables cannot be directly measured. Colloid osmotic pressure (COP) can be measured directly with the use of a colloid osmometer, which is the preferred method in ill animals, or calculated indirectly using several formulas such as the Landis-Pappenheimer formula ($COP = 2.1 TP + [0.16 TP^2] + [0.009 TP^3]$; TP = total protein concentration).¹⁰

Pulmonary Defenses

The lungs have many protective mechanisms against alveolar edema. Two factors limit the movement of fluid out of the capillary space. First, as fluid flows out of the capillary space and into the interstitium, COP decreases secondary to a dilutional effect. Second, as the interstitial hydrostatic pressure increases, the net filtration pressure decreases.^{4,11} As interstitial fluid continues to accumulate, lymphatic flow also increases. Ventilation itself pumps fluid along the lymphatics with the help of one-way valves. Once the interstitial space distends and the alveolar lymphatic system becomes overwhelmed, excess filtrate flows toward the loose interlobular, perivascular, and peribronchovascular interstitial spaces. The interstitial space is able to accommodate a certain amount of fluid, away from the alveoli, with little effect on gas exchange.^{4,12,13}

Several other pulmonary defense mechanisms play a role in maintaining effective gas exchange. As mentioned, the tight junctions of alveolar type I pneumocytes must be compromised before edema forms. Once the barrier has been damaged, fluid that moves into the alveolar lumen can be removed via active transport of sodium by the pneumocytes.^{5,14} Type II pneumocytes play a major role in the removal of the fluid from the alveoli, but type I cells also contribute to fluid transport. Both cell types move sodium out of their cytosol via Na^+/K^+ -ATPase apical pumps, creating an active gradient that drives osmotic absorption of water from the alveolar lumen. Several water transporting proteins, called *aquaporins*, have been identified in the pulmonary system. One, AQP5, which is found on the apical surface of type I pneumocytes, transports water across the cell; another, AQP4, found at the basolateral membrane of the airway epithelium, transports water across the alveolar barrier^{15,16} (**FIGURE 2**).

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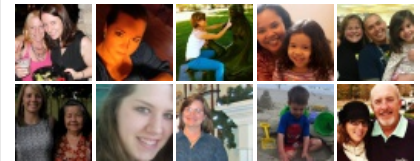
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Causes of Pulmonary Edema

In CPE, edema occurs if the lung's protective mechanisms are overwhelmed by systemic increases in hydrostatic pressure. In cases of NCPE, there may be (1) a local increase in hydrostatic pressure without increases in left atrial pressure or underlying cardiac disease, (2) permeability changes in the alveolar or capillary endothelial surfaces, or (3) a combination of changes in local hydrostatic forces and permeability (**FIGURE 3**).

Despite similar physical examination findings, veterinary patients presenting with NCPE and CPE tend to have different signalment and history. Drobatz et al reported in 1995¹⁷ that of 23 dogs and three cats considered to have NCPE, 11 dogs had neurologic disease (seizures or head trauma), and six dogs and one cat had sustained an electric shock. Six dogs and two cats had had a temporary airway obstruction. Two of these dogs were bulldogs, and the authors suspected brachycephalic syndrome as the cause of NCPE in these animals. Nineteen of the 23 animals were younger than 1 year.¹⁷ A predilection for some forms of NCPE may be age related due to behavior (chewing electric cords, pulling at a leash), but in the human medical literature, NCPE secondary to laryngospasm is a specific risk for pediatric patients compared with adults.¹⁸

Upper Airway Obstruction

NCPE secondary to transient occlusion of the upper airway is also known as *postobstructive pulmonary edema (POPE)* or *negative-pressure pulmonary edema*.^{19,20} POPE can have a wide range of causes, including strangulation, airway collapse, and acute occlusion secondary to a foreign body or other mass. This is evident after examining the history of the eight animals with POPE in the Drobatz review; specific causes included leash strangulation, nasal occlusion, restraint, and brachycephalic disease.¹⁷ Clients of young dogs with POPE typically report low-force strangulation or rough play. Pediatric animals may suffer from complete occlusion of the trachea or larynx due to softness of the supporting tissues and cartilage.

In a review of nine dogs with POPE,²¹ laryngeal pathology, including laryngeal cancer, polyp, edema, or paralysis of the vocal folds, played the dominant role. Only one of these dogs was younger than 5 years. POPE can be secondarily complicated by aspiration pneumonia in patients with laryngeal dysfunction. The laryngeal muscles normally open and close the arytenoids during respiration. When these muscles become paralyzed, the airway may be partially or completely obstructed. A vicious cycle of obstruction and resultant airway inflammation further exacerbates clinical signs.

Brachycephalic patients may be predisposed to upper airway obstruction. Structural abnormalities of the upper airway in brachycephalic animals can include stenotic nares, elongated soft palate, everted laryngeal saccules, everted tonsils, and a hypoplastic trachea.²² A single anatomic or combination of structural abnormalities within the upper airway may lead to a chronic partial obstruction. Acute stresses such as heat, exercise, intubation, or leash strangulation may increase the risk for acute airway obstruction, POPE, and aspiration pneumonia.

Proposed causes of POPE include excessive negative intrathoracic pressure, hypoxia, and sympathetic overstimulation.²⁰ Breathing against an obstructed airway generates a large negative intrathoracic pressure. The abrupt decrease in pressure promotes increased pulmonary venous flow to the heart, thereby acutely increasing pulmonary intravascular volume and pressures. Experimentally, there is evidence of increased afterload stress on the ventricles and increases in both systolic and end-diastolic volumes.^{20,23} With this transient increase in pulmonary capillary hydrostatic pressure, fluid moves from the pulmonary capillary system into the pulmonary interstitial space and the airway. Surfactant is depleted as fluid floods the alveoli, leading to increased work of breathing.²³ Furthermore, mechanical stress on the pulmonary tissue may cause microlesions within the alveolar epithelium that allow solutes and proteins to flow into the alveolar lumen.²³ Transient hypoxia may directly damage the alveolar epithelium and cause pulmonary

vasoconstriction.^{21,24} Acute sympathetic stimulation also promotes pooling of blood within the pulmonary circulation and, subsequently, transiently increases capillary hydrostatic pressure.²¹

POPE patients may present with overt evidence of upper airway disease, an obvious obstruction, or a suspicious history and signalment. Typical physical examination findings include dyspnea with stridor noted on inspiration and/or expiration. On auscultation, increased lung sounds or crackles may be evident. Many patients with POPE are sufficiently hypoxic to require supplemental oxygen ($P_{aO_2} < 80$ mm Hg, $S_{aO_2} < 95\%$). Some of these patients may require sedation and/or immediate intubation. Animals with laryngeal paralysis or foreign body obstruction and brachycephalic animals may benefit from anxiolytics to decrease hyperventilation and allow irritated tissues to heal. If a foreign object is present, it must be removed immediately. Clinicians should be aware that sedation may compromise some of these patients. It has been suggested that bulldogs develop a compensatory hyperactivity of the upper airway dilating muscles to keep the airway open. With sedation, this mechanism is removed, and the resultant relaxation of the dilating muscles could worsen upper airway collapse.^{21,22}

Neurogenic Pulmonary Edema

Neurogenic pulmonary edema occurs secondary to seizures, head trauma, subarachnoid hemorrhage, and subdural hematomas, as well as other central nervous system pathology. The pathogenesis of neurogenic pulmonary edema is not well understood; however, it appears that a combination of hydrostatic and permeability changes are responsible.^{5,25–28} In this model, known as the *blast theory*, an increase in systemic and pulmonary pressures is associated with a surge of sympathetic stimulation, or “catecholamine storm,” initiated by the medulla. This causes a volume shift from the systemic circulation to the pulmonary circulation. The resulting massive systemic vasoconstriction causes pulmonary and systemic hypertension, which may increase capillary hydrostatic pressure upstream in the pulmonary microcirculation. Theoretically, the acute and extensive increase in pulmonary capillary hydrostatic pressure damages the capillary-alveolar epithelium and tight junctions, causing vascular leakage and pulmonary edema.

There is also evidence that neuropeptide Y and endothelin-1 may play a role in edema formation. Neuropeptide Y may directly increase the permeability of the pulmonary vasculature.²⁸ Endothelin-1, as a potent vasoconstrictor, increases pulmonary vascular pressure. Experimentally, intrathecal injections of endothelin-1 and endothelin-3 also enhance vascular permeability.²⁹

NCPE secondary to electrocution is occasionally seen in young dogs and cats. How electrocution causes pulmonary edema is the subject of some debate, but the edema is commonly categorized as neurogenic. In a review of 36 electrocuted animals (29 dogs and seven cats),³⁰ approximately 75% were dyspneic and had evidence of pulmonary edema. Respiratory distress was noted within 1 hour of electrocution by owners. Pulmonary crackles were noted in most patients. All cats in this study survived; however, 38% of the dogs died, reportedly due to respiratory dysfunction.³⁰ Since this report, there have been advances in treatment options that could alter the prognosis.

Physical examination findings include evidence of pulmonary edema shortly after a neurologic episode or trauma to the central nervous system. The onset of respiratory distress is often seen within an hour of the neurogenic event.^{29–31} If neurogenic pulmonary edema is suspected, it is important to evaluate the oral cavity for burns.

Acute Lung Injury/Acute Respiratory Distress Syndrome

Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are severe forms of respiratory dysfunction. They are referred to as “sequel[ae] of diffuse damage to the pulmonary parenchyma within hours to days by a variety of local or systemic insults,” including pneumonia, sepsis, systemic inflammatory response syndrome, and shock.³² In ALI/ARDS, increased permeability is due to widespread pulmonary endothelial and epithelial disruption accompanied by a diffuse inflammatory reaction.³³ At the onset of injury, ALI/ARDS pathology is mainly secondary to

NCPE due to permeability and inflammatory cell infiltration, which occurs in the exudative phase. As the edema resolves, further pathology develops in the proliferative and fibrotic stages of ALI/ARDS.³²

Diagnostic criteria have been established to identify ALI/ARDS patients. Patients must meet at least four of the following five criteria to be diagnosed with ALI/ARDS³⁴: (1) arterial hypoxemia refractory to oxygen therapy ($P_{aO_2}:F_{iO_2}$ ratio ≤ 300 mm Hg for ALI, ≤ 200 mm Hg for ARDS), (2) an acute onset of respiratory distress within the past 72 hours, (3) evidence of pulmonary capillary leakage without evidence of increased capillary hydrostatic pressure (e.g., bilateral infiltrates on thoracic radiographs, proteinaceous fluid within the conducting airways, increased extravascular lung water), (4) evidence of pulmonary inflammation, and (5) presence of a known risk factor.^{32–34}

Clinical signs may be delayed between 1 to 4 days after the local or systemic insult. Risk factors include primary pulmonary diseases such as pneumonia and systemic disorders such as sepsis. Physical examination findings may include harsh lung sounds that may develop into crackles, tachypnea, or respiratory distress or failure. A cough may be present along with pink, foamy sputum in severely affected patients.

Other Causes

Another cause of NCPE is smoke inhalation. Smoke inhalation damages the lungs through three main mechanisms: tissue hypoxemia, thermal damage, and irritation.¹¹ Smoke inhalation can cause damage to the entire respiratory tract.³⁵ Thermal injury is usually associated with the upper airway, leading to extensive edema and ulceration.³⁶ Airway obstruction can occur secondary to extensive damage; in humans, airway obstruction is related to the amount of smoke inhaled.³⁷ Inhalation of toxins and particles present in smoke may cause direct damage to the capillary-alveolar system. This in turn may cause an increase in pulmonary microvascular permeability, ultimately leading to inflammation and edema within the pulmonary alveoli. Peak microvascular damage is seen about 24 hours after exposure. Secondary inflammatory changes occur after the initial pulmonary edema, and many patients suffer from secondary pneumonia.³⁶

In humans, transfusion-related acute lung injury (TRALI) usually occurs 2 to 6 hours after a blood transfusion.^{38,39} TRALI is an immune/inflammatory response associated with all blood products that contain plasma.⁴⁰ It usually resolves within 96 hours.^{38,39} TRALI has not been documented in veterinary medicine; however, it is easily missed in critically ill human patients with preexisting respiratory dysfunction, coagulopathies, or other risk factors for ARDS.

Near-drowning or submersion injury is another cause of NCPE. The factors that contribute to morbidity and mortality in these patients include the type of water aspirated (salt or fresh), water temperature, and contaminants.⁴¹

Diagnostic Testing

CPE and NCPE are differentiated by obtaining a complete patient history, performing a thorough physical examination, and conducting some key diagnostic tests (**TABLE 1**). The history should include questions about recent trauma, exposure to drugs or inhalants, electric cord access, potential choking, recent blood transfusion, or history of cardiac disease. The physical examination can be performed in stages, depending on the degree of respiratory compromise. The oral cavity should be evaluated for burns, lesions, and foreign objects. The patient's respiratory rate, effort, and pattern should be observed carefully, with attention paid to evidence of stridor or inspiratory or expiratory effort. Evaluation of the cardiovascular system should include the capillary refill time, checking for the presence of a heart murmur or arrhythmia, and evaluation for femoral pulse abnormalities and jugular venous distention. Patients with underlying heart disease may not have a heart murmur, especially feline patients.

Thoracic radiography should be performed for all patients with respiratory dysfunction. However, patients in severe respiratory compromise may need respiratory support before images are taken. Depending on the status of the patient, single views (e.g., one lateral or dorsoventral) may be less stressful, but three views are often needed to determine the severity and distribution of pulmonary changes. During radiography, flow-by oxygen can be supplied; the patient should be kept calm by minimal restraint if possible. The heart size, pulmonary pattern, and the pulmonary vasculature should be evaluated. A vertebral heart score can be calculated to objectively evaluate suspected cardiomegaly. For patients with questionable pulmonary edema, a cardiac evaluation, including electrocardiography, may be helpful. Radiographic findings with NCPE include a normal cardiac silhouette and a patchy or peripheral alveolar or interstitial pattern, commonly noted in the caudodorsal lung field (**FIGURE 4** and **FIGURE 5** ; **BOX 2**). Pulmonary infiltration may become diffuse with severe disease such as ALI or ARDS.

Box 2. Radiographic Signs of Noncardiogenic Pulmonary Edema

Heart size: Normal or small

Vasculature size: Normal or small

Pulmonary infiltrates

- Alveolar and/or interstitial or bronchial pattern
- Caudodorsal lung region most commonly affected
- Diffuse infiltrate in all fields in ARDS/SIRS

Arterial blood gas can be measured; however, it does not differentiate NCPE from CPE. A $P_{aO_2} < 80$ mm Hg is considered hypoxic, but P_{aO_2} should not be evaluated alone. Compensatory hyperventilation ($P_{aCO_2} < 35$ mm Hg) can effectively increase the P_{aO_2} to normal levels, leaving the patient at risk for exhaustion and respiratory arrest if not supplemented with oxygen. Other parameters such as the alveolar-arterial gradient (normal, < 20 on room air) and $P_{aO_2}:F_{iO_2}$ ratio (normal, > 400) are useful in further evaluating affected patients. It is also helpful to use repeated P_{aO_2} measurements to monitor the patient's response to oxygen supplementation. Pulse oximetry can also be helpful, with a $S_{aO_2} < 93\%$ considered hypoxic. Some anxious patients tolerate a pulse oximetry probe better than an arterial blood draw.^{42,43}

Evaluation of the PCWP, which is an indirect measurement of left ventricular end-diastolic pressure and left atrial pressure, is not a common practice in veterinary medicine; however, it is considered the gold standard for defining the cause of pulmonary edema. To evaluate the PCWP, a Swan-Ganz catheter is placed from the jugular vein into the pulmonary artery. A pressure sensor at the tip of the catheter measures the pressure in the lung on the pulmonary venous side of the balloon and estimates the pressure in the left atrium. As this is usually a low-pressure system, normal PCWP ranges between 5 and 10 mm Hg.³ A PCWP > 18 mm Hg can indicate cardiac dysfunction or volume overload, including iatrogenic fluid overload.⁴⁴ In patients with NCPE, PCWP is within the normal range.^{2-4,11}

Other useful diagnostic tools include a complete blood count, a chemistry panel, electrolyte levels, a urinalysis, and clotting times. With these tests, the objective is to identify systemic contributions to the pulmonary compromise. NCPE can be hard to differentiate from pulmonary hemorrhage; therefore, clotting times are indicated in all patients, especially those with possible exposure to anticoagulant rodenticides.

Brain natriuretic peptide (BNP) concentration may help distinguish between CPE and NCPE and is commonly used in human medicine to identify patients with congestive heart failure. Secretion of

BNP is increased with pressure overload and left ventricular pathology.⁴⁵ In humans, if BNP is <100 pg/mL, heart function is likely normal and observed pulmonary edema is unlikely to be of cardiac origin.^{9,45} A recently published multicenter, cross-sectional study⁴⁶ supports the use of NT-proBNP measurement in dogs and determined a cutoff concentration that can discriminate dogs with CPE from dogs with primary respiratory disease with moderately good sensitivity and specificity (NT-proBNP, >1158 pmol/L).

Another diagnostic option for intubated patients is the pulmonary edema fluid to plasma ratio (EF:PL ratio). Undiluted pulmonary edema fluid and plasma samples are taken at the same time and analyzed to calculate their protein ratio. In medical literature, a ratio >0.65 is indicative of NCPE.⁴⁷ It is important to collect edema fluid at the onset of clinical signs; as patients begin to heal, the fluid from the airway is absorbed, leaving the protein within the lumen and falsely elevating the edema fluid protein measurement. A long catheter tip is required to get fluid samples from the distal airways. A small pilot study performed by Rozanski et al⁴⁸ in dogs and cats compared edema fluid and plasma protein in intubated patients and found that, compared with patients with CPE, patients with NCPE had not only higher actual edema fluid protein concentrations (4.2 g/dL versus 2.3 g/dL), but also higher EF:PL ratios (0.83 versus 0.34; $P < .05$).^{2,48}

Treatment

Treatment for NCPE patients is largely supportive. The outcome depends on the underlying primary issue and the individual response to therapy. Patients may need oxygen support and should be evaluated serially. Oxygen therapy (Fio₂ between 40% and 70%) can be provided by a simple flow-by system such as nasal insufflation, which is tolerated well by many dogs. Oxygen should always be humidified. Oxygen cages are easy to use and may provide less stress for the patient; however, there are some potential drawbacks, such as the requirements for a high oxygen flow and a significant amount of time for the oxygen level in the cage to reach a therapeutic level of at least 40% to 60%. Also, with each opening of the oxygen cage, the oxygen level drops, making serial examination of the patient difficult.^{49,50}

Patients may require intubation or a tracheostomy to establish an airway or may require ventilation therapy. Ventilation can provide the patient with a Fio₂ of 100%. Prolonged exposure to 100% oxygen therapy is toxic and may lead to pulmonary dysfunction and NCPE. Appropriate caution should be used. Patients that are not able to maintain a Pao₂ >60 mm Hg with oxygen supplementation and/or a Paco₂ <60 mm Hg should be considered as candidates for ventilator support.^{51,52}

Fluid therapy may be required to maintain cardiac output, help maintain hydration, and replace fluid losses. However, IV fluids could increase the pulmonary capillary hydrostatic push or increase leakage in patients with microvascular damage and thereby make the pulmonary edema worse. Monitoring weight helps in assessing patients; however, it does not account for the status of the cardiovascular system or tissue perfusion. Central venous pressure and PCWP are more sensitive indicators of increased hydrostatic pressure but are more technically difficult in an awake and dyspneic patient. COP should be monitored via direct or indirect methods.

Colloid support may also be required and can be guided by serial evaluation of COP. A main concern when using colloid therapy is endothelial integrity. If the capillary endothelium is damaged, the colloid solution may leak into the interstitium and pull more fluid into the interstitium.⁵³⁻⁵⁵ Hemodynamic stabilization is an important part of resuscitation and support in these patients, but patients receiving combined crystalloid and colloid therapy must be closely monitored.

The use of diuretics, such as furosemide, has been proposed to lower pulmonary capillary hydrostatic pressure and decrease "flooding" in an NCPE patient with permeability edema. Furosemide could be used to treat bronchospasm and acts as a bronchodilator.^{11,56,57} The use of

β_2 adrenergic agonists to help increase alveolar fluid clearance has been the subject of some research. β_1 and β_2 receptors exist within the apical and basolateral surface of the alveolar epithelium. Some studies indicate that alveolar clearance significantly increases when β agonists such as terbutaline are administered. The efficacy of these medications is unclear at this time.^{11,58}

Conclusion

NCPE is a relatively common respiratory complication in veterinary medicine due to its wide definition. NCPE must be differentiated from CPE in order to treat the patient properly. Patient history, physical examination, and thoracic radiography can be used to help distinguish the two categories of pulmonary edema and guide ongoing therapy. Outcome depends on the underlying disease and response to therapy.

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Sharon Bell, DVM

Friday, November 16, 2012

The use of acronyms in every article and especially this one detracts from the readability and enjoyment. Some journals list the acronyms at the bottom of each page, which is a big time saver. However my preference would be for no acronym use with an online format.

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Muchas gracias!! excelente articulo

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