Reexpansion pulmonary edema (RPE) is an uncommon but potentially fatal complication after reexpansion of a chronically collapsed lung lobe in a dog or cat. RPE has been reported in humans and small animals when rapid reinflation of a chronically collapsed lung lobe via spontaneous ventilation or positive-pressure ventilation (PPV) is preceded by pleural suction and/or removal of space-occupying lesions. To improve understanding of this intricate secondary complication, this article gives a brief review of lung anatomy and physiology, explains what RPE is, raises awareness of which patients are at risk, offers techniques to help prevent RPE, and describes the available treatments.

Anatomy and Physiology
Air moves down the trachea and enters the lower respiratory tract, which starts at the bronchi where the trachea ends. As the bronchi enter the lungs and divide into smaller diameters, the cartilage support stops; these are the bronchioles. The bronchioles further divide and become smaller and lined with alveoli, becoming alveolar ducts.

The left and right lung lobes in cats and dogs, situated in their respective pleural cavities, are separated from the abdominal cavity by the diaphragm. During inspiration, the diaphragm and external intercostal muscles contract (increasing the thoracic cavity volume), the intrapleural pressure falls, and air is pulled into the lungs. During expiration, the respiratory muscles relax and the volume of the thoracic cavity decreases, increasing pressure inside the lungs and forcing out air. The right and left lungs have cranial (apical), middle (cardiac), and caudal (diaphragmatic) lobes. The right lung has a fourth lobe called the accessory lobe. Each pleural cavity is vacuum lined with a pleural membrane, which secretes a small amount of fluid to reduce friction between the surfaces in the pleural space when the lungs move during respiration. Buildup of this fluid between the layers of tissue that line the lungs and the chest cavity results in pleural effusion. The pleural space loses its vacuum when it is filled with air—a condition called pneumothorax. Expansion of the lungs is limited when there is blood, pus, or lymphatic fluid in the pleural space—conditions called hemothorax, pyothorax, and chylothorax, respectively. When fluid formation in the lung tissue surpasses the ability of the lymphatic system to remove it, pulmonary edema results. Additionally, the pleural cavity can be filled with abdominal organs if the diaphragm is not intact, which could result from a diaphragmatic hernia. When the pleural space is filled and/or the vacuum is lost, the ability of the lungs to inflate decreases secondary to lung compression, which can lead to collapse of one or more lobes—a condition called atelectasis.¹ The severity of the space occupation generally correlates with the degree of respiratory distress; however, if the condition is chronic, the affected animal may adjust over time.

Pulmonary Edema
Pulmonary describes the lung tissue, and edema refers to an excessive fluid volume in the vessels that leaks into interstitial spaces. Pulmonary edema describes lung tissue that contains an abnormal amount of extravascular fluid.² Pulmonary edema can be cardiogenic or noncardiogenic depending on whether it is associated with heart failure or something else. Pulmonary edema develops when fluid moves out of the vessels and into the surrounding space (interstitial space). In cardiogenic pulmonary edema, such as heart failure, this fluid movement is secondary to an increase in pressure within the vessel. Noncardiogenic pulmonary edema is caused by (1) an increase in fluid leaking from the vessels (as in pneumonia, acute respiratory distress syndrome, and iatrogenic fluid overload caused by administration of excessive intravenous fluids or blood transfusion) or (2) a decrease in the protein level in the blood (e.g., as in a protein-losing enteropathy or neuropathy). Other mechanisms that can contribute to noncardiogenic pulmonary edema include diogenic pulmonary edema.

Glossary

- **Atelectasis**—a collapsed state of a lung
- **Dyspnea**—difficult or labored respiration
- **Extravascular**—outside a vessel
- **Hypoxia**—lack of adequate oxygenation of the blood in the lungs
- **Ischemia**—restriction of blood to tissues
- **Pulmonary parenchyma**—lung tissue in animals
- **Pathogenesis**—the origin and development of a disease
- **Thoracocentesis**—removal of fluid or air from the thoracic cavity using a needle
include, but are not limited to, sepsis, ventilator-induced lung injury, and rapid reexpansion of a chronically collapsed lung lobe. Pulmonary edema reduces arterial oxygenation because edema increases the gap between the alveoli and the capillaries, inhibiting diffusion of oxygen from the lungs to the capillaries. Pulmonary edema can also reduce arterial oxygenation by filling the alveoli with fluid, thereby preventing air from filling the alveoli. Additionally, chronic atelectasis, associated with the underlying disease process, reduces lung compliance (elasticity), decreasing overall lung volume. Atelectasis of the alveolar sacs also leads to a loss of surface area for gas exchange, resulting in less oxygen-saturated hemoglobin circulating throughout the body. Signs of pulmonary edema include coughing, difficulty breathing, exercise intolerance, and collapse. Some patients with pulmonary edema have harsh lung sounds or crackles on auscultation. Some patients have less dyspnea if the degree of edema is mild, lung compliance is poor, and/or the respiratory muscles are fatigued.

**Reexpansion Pulmonary Edema**

RPE is a rare form of noncardiogenic pulmonary edema that can result after prompt reexpansion of a chronically collapsed lung lobe. It is not well documented in small animals; however, in humans, the mortality rate has been reported to be as high as 21%. It is thought that the first report of RPE was by a physician in 1853 when pulmonary edema occurred following reexpansion of a lung after pleural effusion was evacuated via thoracocentesis. Other reports of RPE followed, such as in 1959, when pulmonary edema occurred in a patient with pneumothorax after a physician performed thoracocentesis. This suggested to physicians that RPE is possible when rapid reexpansion of the lungs follows any disease process that causes chronic lung collapse. The potential for RPE in small animals was first recognized in a 1965 study, when 32 dogs and cats underwent surgical repair of diaphragmatic hernias.

Chronically (>72 hours) collapsed lung lobes are at greater risk for RPE because the longer a lung is collapsed, the more the structures within the parenchyma thicken, harden, and become inflexible. Because of the decreased flexibility, the microvessels are more likely to be damaged during stretching as the lung reexpands to normal size. This damage causes leakage of fluid into the interstitial space and a reduction in perfusion. It has been reported that blood flow can be reduced up to 72% during atelectasis.

Reexpansion of the lungs allows alveolar ventilation and perfusion to resume. This rapidly reoxygenates the lungs, which were hypoxic and vasoconstricted before reinfalation. RPE can be compared with ischemia-reperfusion injury. It is thought that cats are more susceptible than dogs to RPE, but the reason for this is unknown.

**Prevention**

To help prevent RPE, complete and/or immediate expansion of a chronically collapsed lung lobe should be avoided. Abrupt evacuation of air and/or fluid from the pleural cavity by thoracocentesis or large expansion of the lung lobe by manual or mechanical PPV should be avoided. To avoid RPE during thoracocentesis, fluid and/or air should be removed slowly. Safe volumes and rates for removal of fluid and/or air in small animals largely depend on the individual case and the comfort level of the clinician.

Manual or mechanical PPV of diseased lungs can begin a chain of events leading to complications. When collapsed lung lobes, which are less compliant than normal lobes, are suddenly ventilated with a normal tidal volume, they can become overdistended, resulting in barotrauma or volutrauma. Barotrauma happens when air is forced into a lung under pressure that is too high; volutrauma happens when the volume of air forced into a lung is too great. Both conditions can lead to pneumothorax when air leaks from the lungs into the pleural cavity. Even in the absence of pneumothorax, pulmonary edema can occur.

When diseased lungs are artificially ventilated, a lower-than-normal tidal volume and a higher-than-normal pressure are needed to push air into the noncompliant tissue without overstretching and damaging it. The tidal volume should be equal to the volume of a normal breath of the patient being ventilated. Normal lung ventilation requires a pressure of 8 to 15 cm H$_2$O (without exceeding 20 cm H$_2$O) and a tidal volume of 10 to 15 mL/kg. Diseased lungs require a tidal volume of 6 to 10 mL/kg and an initial pressure of 10 to 15 cm H$_2$O with adjustments up to 30 to 35 cm H$_2$O, according to pulmonary function. Pulmonary function can be monitored in various ways, such as end-tidal carbon dioxide (ETCO$_2$), blood oxygen saturation (SpO$_2$), mucous membrane color, or blood gas analysis.

When mechanical ventilation is available for an anesthetized or intubated patient, values such as peak inspiratory pressure and tidal volume can be preset. During manual ventilation, only peak inspiratory pressure can be monitored with a pressure manometer; therefore, care must be taken to avoid overinflation of collapsed lung lobes. A ventilometer or respirometer can be attached to the anesthetic circuit to measure tidal volume, but this equipment is expensive and not commonly used.

A valuable method of ventilating diseased lungs is positive end-expiratory pressure (PEEP), which works by maintaining constant pressure in the breathing circuit during exhalation, preventing the patient from exhaling completely. The constant pressure holds the alveoli open and may help to improve oxygenation by acting as a recruitment maneuver to reduce atelectasis. Patients with lung disease may also require increased PEEP. Healthy lungs require a PEEP of 0 to 5 cm H$_2$O; diseased lungs require 3 to 8 cm H$_2$O.

PEEP valves that connect to anesthesia machines are commercially available. ETCO$_2$ should always be monitored by capnography during PPV to ensure that a patient is

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**Suggested Reading**


not overventilated or underventilated. A normal ETCO$_2$ range is 35 to 45 mm Hg. ETCO$_2$ is decreased by hyperventilation and increased by hypoventilation.

**Treatment**
Prevention of RPE is well documented (Suggested Reading) and easier than treatment. Because RPE is complex, treatment is limited to supportive care based on the severity and signs of pulmonary edema. Treatment protocols typically include diuretics, correction of acid-base disturbances, and hypertonic solutions, with the chief supportive measure being oxygen supplementation. In conscious patients, oxygen supplementation can be delivered by various methods, including oxygen cage, nasal oxygen, and flow-by oxygen via facemask. In more severe cases, intubation may be necessary. In humans, differential lung ventilation is beneficial in managing further damage when mechanical ventilation is used to maintain an adequate oxygen level. Differential lung ventilation has been used in patients with unilateral RPE lung involvement and involves placing a double-lumen endobronchial tube in lieu of an endotracheal tube. The unaffected lung is mechanically ventilated based on healthy lung parameters, and the affected lung is ventilated separately using PEEP only. Although this technique is standard in human medicine, in veterinary medicine, it is limited to hospitals that offer an advanced level of care. Treatment of RPE in small animals may be difficult because it requires special equipment and knowledge of advanced techniques.

**Conclusion**
Although RPE has a complicated pathogenesis and a very low incidence, it can be fatal; therefore, veterinary technicians should know which patients are at risk for RPE and how it can be prevented and treated to improve a patient's chance of survival.

**References**
1. During respiration, gas exchange occurs in the
   a. bronchi.
   b. alveolar sacs.
   c. trachea.
   d. bronchioles.

2. The fourth lobe of the right lung is called the ________
   lobe.
   a. cardiac
   b. apical
   c. accessory
   d. diaphragmatic

3. When pulmonary edema is present, which of the following does not reduce arterial oxygenation?
   a. filling of the alveoli with fluid
   b. an increased gap between the alveoli and the capillaries
   c. increased surface area of the lung tissue
   d. a reduction in lung compliance

4. In RPE, atelectasis is considered to be chronic when it has been present for ________ hours.
   a. 12 to 24
   b. >36
   c. 36 to 48
   d. >72

5. Which of the following is the appropriate pressure and volume for ventilating diseased lungs?
   a. high pressure; high volume
   b. low pressure; low volume
   c. high pressure; low volume
   d. low pressure; high volume

6. When a patient with diseased lungs is artificially ventilated, the
   a. respiratory rate may need to be decreased.
   b. pressures may need to be decreased.
   c. tidal volume may need to be increased.
   d. pressures may need to be increased.

7. Which of the following does not increase the risk for RPE in chronically collapsed lung lobes?
   a. thickening and hardening of structures within the parenchyma
   b. positive end-expiratory pressure ventilation
   c. decreased flexibility of microvessels
   d. rapid reoxygenation of hypoxic tissues

8. The chief supportive effort during treatment of RPE is
   a. fluid therapy.
   b. oxygen supplementation.
   c. administration of diuretics.
   d. artificial ventilation.

9. RPE is most likely to develop after
   a. rapid reinflation of chronically collapsed lungs.
   b. gradual reinflation of chronically collapsed lungs.
   c. intermittent fluid removal via a chest tube.
   d. intermittent air removal via a chest tube.

10. Which of the following cannot be used to monitor pulmonary function?
    a. ETCO₂
    b. mucous membrane color
    c. blood gas analysis
    d. capillary refill time