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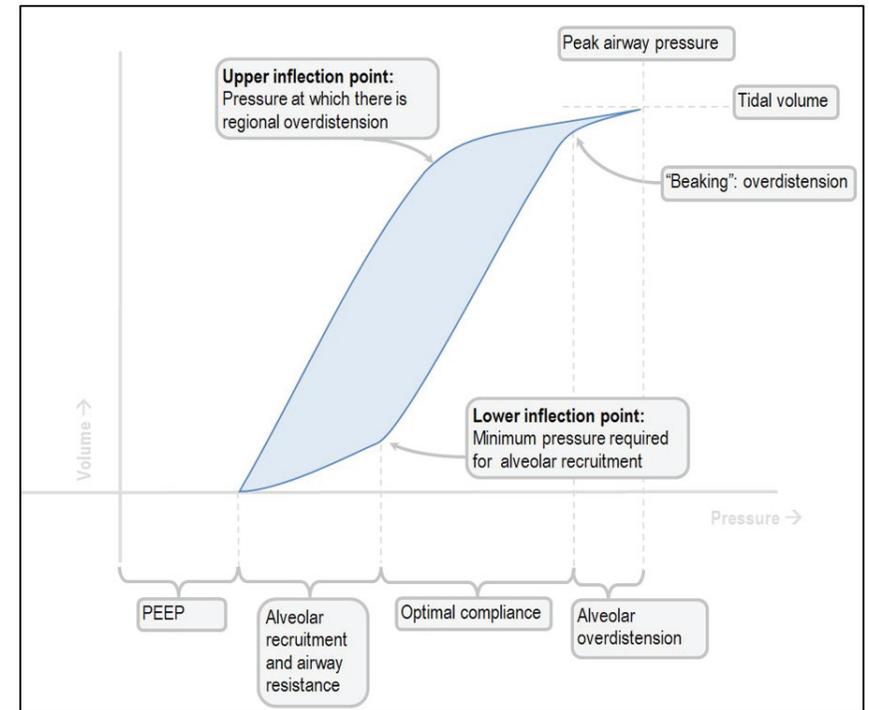
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PULMONARY PARENCHYMAL DISEASE

Considering respiratory pathology in general, pulmonary parenchymal disease is the respiratory condition which is most likely to cause hypoxia. Conditions causing pulmonary parenchymal disease generally lead to fluid accumulation within the alveoli which directly compromises gas exchange. As oxygen does not diffuse as easily across the vascular and alveolar walls, any impairment of gas exchange can lead to some degree of hypoxia. Although carbon dioxide exchange can be impaired, due to its solubility, it is transported easily within the circulation and readily diffuses. Therefore, the development of hypercarbia is relatively late in most disease processes.

Fundamentally, diseases involving the pulmonary parenchyma lead to a decrease in compliance. Compliance is defined simply as the change in volume divided by the change in pressure.

$$\text{Compliance} = \frac{\Delta \text{Volume}}{\Delta \text{Pressure}}$$

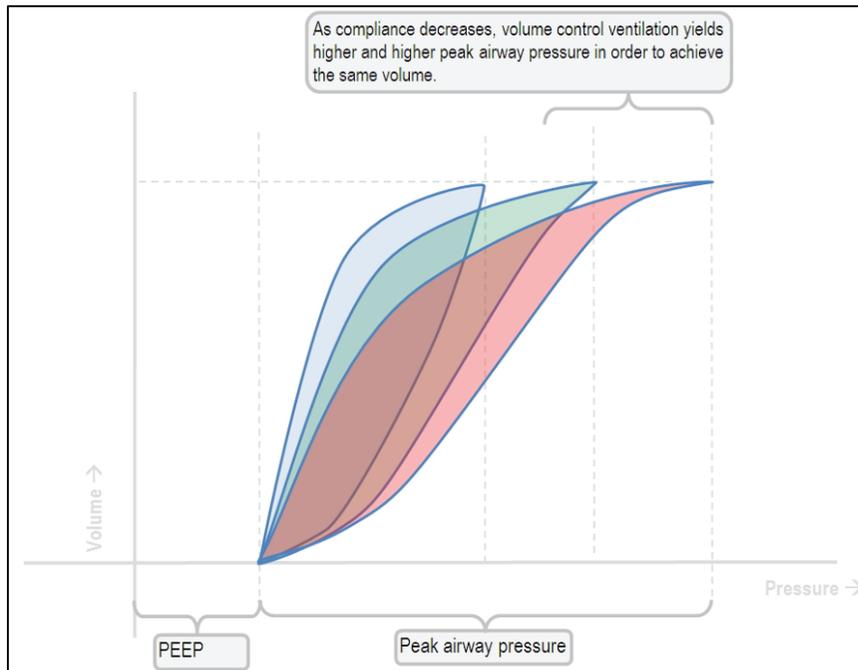


<http://www.derangedphysiology.com/main/core-topics-intensive-care/mechanical-ventilation-0/Chapter%205.1.3/interpreting-shape-pressure-volume-loop>

As pulmonary parenchymal disease develops and compliance decreases, the patient must generate greater pressure (meaning the patient must exert more effort) to create the former change in volume. This decrease in compliance means the patient now must work harder on both inspiration and expiration. As expiration is typically a passive process in both dogs and cats, any degree of expiratory exertion (meaning, any detectable active effort during expiration) while at rest can often be appreciated by the observer. As inspiration is an active process in both dogs and cats, small increases in inspiratory exertion may be more difficult for the observer to appreciate.

COMPANION ANIMAL

THORACOLOGY



<http://www.derangedphysiology.com/main/core-topics-intensive-care/mechanical-ventilation-0/Chapter%205.1.3.1/pressure-volume-loops-presence-lung-pathology>

Pulmonary parenchymal disease generally occurs due to one of two reasons: alveolar fluid accumulation or consolidation and diffuse replacement of the normal pulmonary tissue as may occur with granuloma formation or metastatic/diffuse neoplasia. Fluid accumulation almost exclusively occurs in the form of edema, blood or purulent material. Edema may develop secondary to cardiogenic causes, non-cardiogenic causes (e.g. strangulation, electrocution, near-drowning, seizures, head trauma), or vasculitis. Blood may accumulate into the alveolar space due to trauma (e.g. pulmonary contusions) or a hemostatic disorder (e.g. thrombocytopenia is more likely than a secondary hemostatic disorder). Purulent material is generally found in the alveolar

space secondary to infection (bacterial most common) but can occur secondary to a sterile pneumonitis (such as aspiration of sterile gastric contents) or an immune-mediated process (e.g. eosinophilic bronchopneumopathy).

Thoracic radiographs are frequently the most useful tool in the diagnostic approach to patients with pulmonary parenchymal disease. A diffuse interstitial or alveolar pattern is a common radiographic finding and the distribution of such a pattern can be useful in determining the underlying cause:

- Cardiogenic edema: generally, begins in the perihilar region
- Non-cardiogenic edema: classically, presents with bilateral caudodorsal infiltrates
- Pneumonia: most often affects the cranioventral lobes (especially right middle lobe)
- Blood: often patchy distribution but no classic location

Airway sampling (either via endotracheal wash or bronchoalveolar lavage) may be useful in confirming a suspicion, but frequently, a diagnosis can be made or highly suspected based on a combination of the patient's signalment, geography, history reported by owners, physical examination, and radiographic findings.

In the emergent setting, cardiogenic edema is typically treated with furosemide, oxygen, mild sedation, and occasionally pimobendan. Further direction usually follows a more thorough cardiology evaluation. Non-cardiogenic edema, however, has little in the way of treatment options outside of oxygen support and nursing care. There is no evidence that such patients benefit from furosemide, antibiotic, or steroid therapy. Ideally, patients with suspected bacterial pneumonia undergo an airway wash to differentiate bacterial pneumonia from sterile pneumonitis as well as target antibiotic therapy once culture results have returned. In practice, however, these patients are generally treated with broad spectrum antibiotics along with oxygen support, if indicated. There is some evidence to suggest that septic patients may benefit from gastric antacids in the form of either proton pump inhibitors or H₂-receptor antagonists. Following trauma, patients with pulmonary contusions are frequently treated for hypovolemic shock. There is an argument for limited volume resuscitation in these patients to minimize aggravation and worsening hemorrhage of pulmonary

contusions. Although not an immediate concern, it is important to consider that people with pulmonary contusions have an increased risk of developing pneumonia and more severe contusions are associated with the development of acute respiratory distress syndrome.

If geography, history, and examination create concern for granulomatous disease, testing may be performed for serology or the presence of antigen. Treatment is generally initiated while waiting for laboratory results as the treatment course is usually protracted (often 9-12 months for fungal disease). In the instance that a patient is diagnosed with diffuse or metastatic neoplasia, treatment is supportive or aimed at secondary complications as the primary disorder cannot reasonably be treated or controlled.

References

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