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When is Respiratory Distress not Respiratory Distress

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Respiratory distress is a common emergency problem characterized by an increased respiratory rate and effort. Animals are often uncomfortable and restless; they may extend their neck and have increased respiratory noise. The causes of respiratory distress can be divided into eight broad categories;

1. Upper airway disease
2. Lower airway disease
3. Pulmonary parenchymal disease
4. Pleural space disease
5. Chest wall disease
6. Pulmonary thromboembolism
7. Abdominal distension
8. “Look alike” diseases

The first 6 causes all involve primary respiratory tract pathology and are commonly associated with abnormalities in oxygenation and ventilation. The last two causes are not primary respiratory tract disease processes and are discussed further below.

**Abdominal distension:**
Abdominal distension causes anterior displacement of the diaphragm leading to respiratory compromise. Common causes of severe abdominal distension include gastric dilation volvulus, heavy pregnancy, ascites and intra-abdominal masses. Significant abdominal distension in a patient with respiratory distress should be decompressed as possible. In cases of severe abdominal distension pulmonary atelectasis can occur.

**“Look alikes”:**
There are several causes of increased respiratory rate and/or effort that are due to stimulation of the central respiratory center in animals with no primary respiratory tract disease. These patients will have normal oxygenation and no abnormalities on auscultation or thoracic radiographs. They require no treatment of their respiratory signs but may benefit from treatment of their primary disease process.

Many brain diseases can cause stimulation of the medullary respiratory center. Diseases such as neoplasia, inflammatory brain disease and trauma can all lead to respiratory changes. These patients usually have other signs of brain disease such as obtundation, cranial nerve defects and behavioral changes. They may also have abnormal respiratory patterns such as apneustic breathing and cheyne stokes breathing.

Hyperthermia is also a potent stimulus of respiratory rate and effort, especially in dogs. Hyperthermia is the consequence of high ambient temperatures and/or exertion and should be differentiated from fever. Fever describes an elevation in body temperature due to endogenous pyrogens and will not trigger increases in respiratory rate and effort. Measurement of body temperature in animals with respiratory distress is very important. Hyperthermia may be the primary cause or a contributor to increased respiratory rate and effort and cooling will tend to improve these clinical signs.

Drugs such as opioids will directly stimulate the central respiratory centre producing rapid panting. On examination these animals will have a normal or lower than normal body temperature and a normal or higher than normal PCO\(_2\).
Metabolic acidosis will also stimulate increases in respiratory rate and depth of breathing in an effort to lower PCO₂ and return pH towards normal.

**Hypoventilation:**
This is a form of respiratory compromise that does not present with increased respiratory rate and effort. Hypoventilation is associated with reduced respiratory rate and effort that is often clinically indistinguishable from normal breathing.

Ventilation is considered to be the tidal movement of air in and out of the lungs. It is quantified as minute ventilation (MV) which is the total volume of gas exchanged in one minute and equals the respiratory rate (RR) multiplied by the tidal volume (TV) of each breath. \( \text{MV} = \text{RR} \times \text{TV} \)

Hypoventilation occurs when there is insufficient fresh gas inhaled down to the level of the functional alveoli to adequately remove carbon dioxide. The end result is an elevation in blood carbon dioxide levels (hypercapnia). This ‘effective alveolar ventilation’ equals the MV less the volume of dead space. Dead space is any portion of the tidal volume that does not participate in gas exchange. For example tidal volume that fills the upper airways and bronchi is considered dead space and is not effective alveolar ventilation. By definition hypoventilation will cause an elevation in blood carbon dioxide levels – hypercapnia.

**Clinical Signs**
Hypoventilation is often clinically silent. It can be due to an inadequate RR, an inadequate TV or perhaps both. Cases with shallow breathing will often have an increased RR and there can be exaggerated abdominal movements to compensate for insufficient thoracic wall movements.

If hypercapnia is severe it can lead to central nervous system depression and in extreme cases narcosis and coma can occur. Systemically hypercapnia tends to increase heart rate and blood pressure. Intracranially hypercapnia causes cerebral vasodilation which can result in increases in intracranial pressure. For this reason patients with primary brain disease often cannot tolerate a carbon dioxide level any higher than normal.

**Causes**
Carbon dioxide levels are normally very tightly maintained within a narrow, acceptable range. If there is any change in carbon dioxide levels noted the respiratory system will instantly alter ventilation in response. Hypoventilation is most commonly the result of neuromuscular disease processes that impair this pathway.

Neuromuscular Disease:
- Central respiratory center depression eg. Anesthetic & sedative drugs, brain trauma, mass lesions etc
- Cervical spinal cord disease eg. intervertebral disc protrusion, trauma, mass lesions etc
- Lower motor neuron / neuromuscular junction abnormalities eg. myasthenia gravis, botulism, polyradiculoneuritis etc.
- Respiratory muscle problems eg. Myopathy, chest wall trauma, respiratory muscle fatigue

Other:
- Pulmonary thromboembolism
- Excessive breathing circuit dead space (anesthetized animals)
- Exhausted soda lime, machine malfunction (anesthetized animals)

**Diagnosis**
Hypoventilation may be suspected from the clinical signs present but cannot be confirmed without measuring arterial or venous PCO₂. Normal arterial carbon dioxide levels are approximately 37 mmHg in the dog and 32 mmHg in the cat. Normal venous carbon dioxide levels are 5-10 mmHg higher than the arterial levels and provide an acceptable indication of arterial carbon dioxide in most cases. A PCO₂ of greater than 45 mmHg in dogs and 40 mmHg in cats is considered hypercapnia. A PCO₂ of greater than 60 mmHg is severe hypercapnia and requires intervention.

In intubated patients endtidal carbon dioxide (ETCO₂) measurements can be performed. In normal animals the ETCO₂ is approximately 2-6 mmHg less than arterial PCO₂ and is considered an accurate representation of arterial PCO₂ in most situations.

**Treatment**
The initial priority in the hypoventilating patient is to ensure the animal has a patent airway and is breathing. If the airway is compromised it should be immediately secured with an orotracheal tube, if this cannot be placed
successfully a tracheostomy is indicated. If the animal is not breathing or has an insufficient respiratory rate manual ventilation should be started. Oxygen therapy is essential for any animal that has significant hypercapnia.

When possible, specific therapy for the primary disease process should then be provided in order to lower the PCO₂. For example decompression of a cervical intervertebral disc prolapse or reversal of an overdose of opioid drugs. When there is no specific therapy available mechanical ventilation is indicated in patients with persistent severe hypercapnia. Mechanical ventilation will be able to provide a satisfactory alveolar minute ventilation to ensure adequate CO₂ elimination. The goal of mechanical ventilation is to stabilize the patient while diagnostic and therapeutic interventions are provided to resolve the primary disease process.

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