Pulmonary Monitoring and Support

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The lung has many important functions, two of which are the transfer of oxygen from the environment to the blood and the removal of carbon dioxide from the blood to the atmosphere. Respiratory failure encompasses either the inability of the lung to properly oxygenate the blood (hypoxemia) or the inability of the lung to properly remove carbon dioxide from the blood (hypercapnia), or both. Disease and therapy can easily impact one function more than the other, and it is necessary to consider each function separately.

Breathing Rate

The central pattern generator (the inspiratory and expiratory neurons) in the medulla are influenced by many ascending and descending afferent signals. One of these is the central chemoreceptors located near the surface in the medulla which are very sensitive to the hydrogen ion changes associated with changes in CSF carbon dioxide. Medullary disease can impair the ability of these sensors to detect an increasing carbon dioxide or can impair the central pattern generator directly, resulting in hypoventilation or apnea. The breathing rate, per se, is of limited value without some reference to tidal volume and previous trends because normal rates can vary so widely. A change in breathing rate is, however, a sensitive indicator of a change in the underlying disease process.

Breathing Rhythm

Arrhythmic breathing patterns are indicative of a problem in the medullary respiratory control center and may be manifested by "central neurogenic" tachypnea, apneustic (inspiratory hold) breathing pattern, cyclic hyper/hypoventilation or cyclic tachypnea/bradypnea, or cyclic bradypnea/apnea. Most of these abnormal breathing patterns are associated with the clinical picture of a decreased breathing effort. A central medullary dysfunction is often associated with other signs of intracranial disease. Agonal gasping, characterized by a gaping of the mouth and, sometime, spasmodic contraction of the diaphragm, is not real breathing even if there is movement of some air. It usually occurs sometime after the animal has arrested and is truly a terminal medullary sign.

Breathing Nature

Breathing is normally a rather subtle event. Inspiration and exhalation are normally less than a second each. Inspiration is normally accomplished with simultaneous expansion of both the chest and the abdomen (diaphragm). The volume of the chest and abdominal expansion are usually minimal. Prolonged inspirations may be indicative of upper airway obstructive disease. Prolonged exhalations may be indicative of lower airway collapse disease. Large excursions of the chest and abdomen indicate large tidal volumes or exaggerated effort. Grossly audible noise indicates upper or lower airway obstructive disease. Upper thoracic spinal cord disease may cause intercostal motor paralysis (breathing is characteristically abdominal or diaphragmatic). Phrenic

nerve "irritability" is associated with hiccoughs - the diaphragm "twitches" with each heart beat. In both situations the animal breathes satisfactorily, however the appearance of the breathing pattern is altered. Cervical cord disease may interfere with both intercostal and diaphragmatic function resulting in weak or absent ventilatory efforts.

Breathing Effort

A normal animal breathes free and easy. If the animal is putting extra effort into breathing, it's respiratory centers are being driven by hypoxemia or hypercapnia, or some other non-respiratory problem such as hypotension, hyperthermia, excitement, or sepsis. Or the animal is having difficulty moving a tidal volume (upper or lower airway obstruction, intrathoracic or intra-abdominal space-filling abnormality (pneumothorax, diaphragmatic hernia, gastric dilation, severe ascites).

Mucous Membrane Color

Mucous membrane color should be evaluated for ashen or cyanotic discoloration as an indicator of hypoxemia. Cyanosis is always a late sign and is unreliable in that it may not appear in an animal that is anemic. It takes an absolute amount of unoxygenated hemoglobin (about 5 gm/dl) in the tissue bed to show cyanosis. If an animal is very anemic, it could die of hypoxemia without showing any cyanosis simply because there is not enough hemoglobin to show it. An animal may have cyanotic discoloration of the mucous membranes if there is sluggish flow through the capillary bed (congestive, end-stage of hypovolemic or septic shock; cardiac arrest) or if there is methemoglobinemia. In methemoglobinemia, the PaO_2 would be normal but the hemoglobin oxygenation (%) and blood oxygen content would be low. The animal may appear to be cyanotic with poor or fluorescent lighting.

Auscultation

Air moving through the normal trachea and bronchi are easiest to hear (rough, high pitched sounds). The sounds made by air flowing through the lobar and segmental bronchi are heard as soft, low-pitched, gentle rustling sounds in the periphery of the lungs. Abnormal sounds that you might hear include crackles which are caused by the sudden opening of small airways during inspiration and the sudden closing of these airways during exhalation. Crackles suggest an increase in airway fluid causing premature closure of airways during exhalation and their re-opening during the next inspiration. Loud, rough pleural friction rubs, caused by inflammation of the serosal surfaces, are generally of a lower pitch and a longer duration than crackles and the pattern of the sound occurs in reverse order during inspiration vs exhalation. Very loud, low pitch, snoring sounds suggest an upper airway obstruction and is due to the vibration of tissues primarily during inspiration. Very loud, high-pitched, squeaky noises, primarily during inspiration, are caused by the turbulence of air being forced through a narrowed airway and suggest a very severe upper airway obstruction. Wheezes, or asthmatic breathing sounds, heard both during inspiration and expiration are caused by turbulent airflow through narrowed lower airways. They may have a musical quality and may be high-pitched or low-pitched, and may be loud and polyphonic or subtle and monophonic.

Radiography

A chest radiograph is often very diagnostic in chest disease and should be procured if the patient is sufficiently stable to tolerate the stress and the time delay of the procedure (some patients are presented in such severe life-threatening condition that it would not be appropriate to obtain a chest radiograph prior to stabilizing the patient). The chest radiograph should reveal the problem if it involves the pleural space, lung parenchyma, or lower airways. The chest radiograph may look relatively normal in neuromuscular and upper airway obstruction disease, look-alike disorders, and pulmonary thromboembolism. An abnormal chest radiograph defines that an abnormality exists, but does not necessarily define the physiologic performance of the lung. The lung can compensate for a great deal of disease and still function normally . So, depending upon the nature of the disease and the compensation of the patient, there may be a disparity between how they look on the radiograph and how they perform as defined by arterial blood gases.

Ventilometry

Ventilation volume can be estimated by visual observation of chest or rebreathing bag excursions, or measured by ventilometry. Normal tidal volume ranges between 10 and 20 ml/kg. A small tidal volume may be acceptable if the breathing rate is fast enough to accomplish normal alveolar minute ventilation. Normal total minute ventilation ranges between 150 and 250 ml/kg/min.

Arterial PCO_2 ($PaCO_2$)

The average $PaCO_2$ is between 35 and 40 mm Hg in dogs and 30 to 35 mmHg in cats. Below-normal range values represent hypocapnia due to hyperventilation while above-normal values represent hypercapnia due to hypoventilation. A $PaCO_2$ in excess of 60 mmHg may be associated with excessive respiratory acidosis, and hypoxemia (when breathing room air) and is considered to represent sufficient hypoventilation to warrant therapy. Because hypercapnia causes cerebral vasodilation and increases cerebral blood flow, $PaCO_2$ values should be maintained within the normal range in animal with intracranial disease. $PaCO_2$ values below 20 mmHg are associated with excessive respiratory alkalosis and a decreased cerebral blood flow which may impair cerebral oxygenation.

$PvCO_2$ is usually 3-5 mmHg higher than $PaCO_2$ and can usually be used as a surrogate marker of $PaCO_2$. The arterial/venous gradient may be greater when there are problems with carbon dioxide carriage such as during anemia, sluggish peripheral blood flow or during carbonic anhydrase inhibitor therapy, or during transition states of changed ventilation. End-tidal PCO_2 is usually 3-5 mmHg lower than $PaCO_2$ and can also be used as a surrogate marker of $PaCO_2$. End-tidal PCO2 can be variably lower than PaCO2 with pulmonary thromboembolism and hypovolemia, with tachypnea.

Arterial PO_2 (PaO_2)

The amount of oxygen in the blood can be expressed three different ways: 1) the partial pressure of oxygen dissolved in the plasma (PO_2; units = mm Hg), 2) the percent saturation of the hemoglobin (SO_2; units = %), and 3) the whole blood oxygen content

($Content O_2$; units = milliliters of oxygen per 100 milliliters of whole blood). An arterial blood sample is required; the dorsal metatarsal or femoral/medial saphenous arteries are most commonly used, however, the brachial, radial, aural, and lingual arteries may also be accessed. Free-flowing capillary blood has also been used to approximate an arterial blood sample. The venous blood is separated from arterial blood by a metabolically active tissue bed which consumes oxygen. In contrast to venous carbon dioxide, venous oxygen measurements bear no correlation to arterial oxygen measurements and cannot be used to assess pulmonary function. Venous oxygen measurements is interpreted by an entirely different set of criteria. The PO_2 is the vapor pressure of oxygen dissolved in solution in the plasma. It is independent of hemoglobin and is not affected by anemia. It is measured in a blood gas analyzer with a silver anode/platinum cathode system in an electrolyte solution (polarography) separated from the unknown solution (the blood) by a semipermeable (to oxygen) membrane. The normal PaO_2 at sea level ranges between 80 and 110 mm Hg. These values vary at altitude due to the decrease in barometric pressure. A $PaO_2 > 110$ represents hyperoxemia while a $PaO_2 < 80$ mm Hg represents hypoxemia. Severe hypoxemia, which requires intervention, is usually defined as a PaO_2 of less than 60 mm Hg.

The percent saturation of the hemoglobin (SaO_2 when measured in arterial blood and SpO_2 when measured by a pulse oximeter) is related to the PaO_2 (oxyhemoglobin dissociation curve) and can be used as a surrogate marker of PaO2 in normal to low range, but not high, PO2 values.

PaO_2	SaO_2 or SpO_2
500	100
100	98
80	95
60	90
45	75
30	50

Oxygen therapy

Oxygen is often administered when a patient with respiratory distress is first presented. Oxygen therapy may be beneficial when the predominant cause of the hypoxemia is ventilation-perfusion mismatching or diffusion impairment. Oxygen therapy would not be expected to be substantially beneficial if the predominant cause of the hypoxemia is small airway and alveolar collapse. The patient's response to the oxygen therapy should be evaluated at periodic intervals (improvement in mucous membrane color, a decrease in restlessness, decrease in the magnitude of the respiratory distress, decrease in breathing rate and/or heart rate, an improvement in PaO_2 or SpO_2 to an acceptable level.

A high inspired oxygen concentration can easily be attained with a face mask. The animal's nose and face should fill the mask as much as possible to reduce the dead space within the mask. The mask does not necessarily need to make an airtight seal around the animal's muzzle; leaks may, however, allow room air to be drawn into the mask during inspiration and decrease the inspired oxygen concentration. Since dyspneic patients commonly do not tolerate a tight fitting face mask, the mask or oxygen outlet should be held as close to the animal's nose as possible.

Oxygen cages are commercially available or can be homemade. Oxygen tents and infant incubators may be available from used medical equipment suppliers. In any enclosed environment, it is important to regulate and control the oxygen concentration in order to optimize the therapy, to control the carbon dioxide concentration, and to control

the humidity and the temperature. High humidity is acceptable as long as the temperature is controlled at a comfortable level.

A convenient way to increase the inspired oxygen concentration is via insufflation. A soft, flexible catheter can be inserted into the nasal cavity, about to the level of the medial canthus of the eye. The catheter should exit the nose via the lateral alae and then sutured at this exit point and at points on the side of the face or the top of the head to keep the catheter out of the patient's view. An intravenous catheter could also be placed transtracheally through the cricothyroid membrane or between tracheal rings. Additional side holes can be placed in the catheter near the tip to facilitate the diffusion of the oxygen into the airway and to minimize the jetting of the gas against one spot on the tracheal epithelium. The catheter should be sutured and bandaged to the patient's neck in the same fashion as a jugular catheter. The catheter should be long enough so that it can't accidentally slip out of the tracheal lumen and into the subcutaneous tissues; serious subcutaneous emphysema, pneumomediastinum, and pneumothorax can develop very rapidly. An oxygen flow rate of 50 to 100 ml/kg effectively maximizes the inspired oxygen concentration; flow rates should be subsequently adjusted to the needs of the patient. Medical oxygen is anhydrous and should be bubbled through warm water so that it will be at least partially humidified by the time it reaches the patient.

Positive pressure ventilation

Positive pressure ventilation (PPV) is indicated whenever an animal cannot ventilate adequately due to neuromuscular disease or pulmonary parenchymal disease. PPV is also indicated when hypoxemia does not respond to oxygen therapy. PPV is also indicated when the animal is having to work excessively hard to breath and is in danger of muscle exhaustion. This is a subjective assessment, but if the animal's condition is serious enough to incite you to be concerned about the breathing effort, it is probably serious enough to warrant PPV.

The goal of positive pressure ventilation are to stabilize ventilation ($PaCO_2$ 35 to 60 mmHg) and oxygenation (PaO_2 80 to 120 mmHg) at modest inspired oxygen concentrations (< 60%) while minimizing the deleterious effects of the procedure.

The general guidelines for positive pressure ventilation of animals with relatively normal lungs are: 1) a peak proximal airway pressure of 10 to 20 cm H_20; 2) a tidal volume of 10 to 15 ml/kg; 3) an inspiratory time of about 1 second (or just long enough to achieve a full tidal volume); 4) a ventilatory rate of about 10 to 15 times per minute; 5) a minute ventilation of about 150 to 250 ml/kg/minute; and 6) a 0 to +2 end-expiratory pressure. Some ventilated patients may require a deep breath (a sigh) at an airway pressure of 30 cm H_20 at regular intervals (30 minutes) to minimize small airway and alveolar collapse.

Diseased lungs are stiffer (less compliant) than normal lungs, and are therefore much more difficult to ventilate. It is a common finding that the above recommended guidelines are insufficient to adequately oxygenate or ventilate a patient with diffuse pulmonary parenchymal disease. Whenever ventilator settings do not seem to meet the aforementioned goals: 1) make sure that the ventilator settings are indeed what you had planned (including the inspired oxygen); 2) make sure that there is patient synchrony; and 3) make sure that other untoward events are not present (hyperthermia, pneumothorax). After these conditions are met, adjust ventilator settings. There is no particular order in

which ventilator settings should be adjusted. To improve ventilation: 1) the proximal airway pressure could be increased in a step-wise fashion up to 60 cm H_20 (or to the limit of the ventilator). It is not a goal of PPV to increase the tidal volume (pulmonary disease is associated with a reduced vital capacity due to a reduced inspiratory and expiratory reserve volume and smaller-than-normal tidal volumes have been demonstrated to be lung protective), however, it is inevitable that increasing peak airway pressure will increase tidal volume. 2) the ventilatory cycle rate could be increased in a step-wise fashion up to 60 breaths per minute (be sure that the last breath is completely out of the patient before the next breath starts to minimize air trapping). 3) the inspiratory time or the inspiratory plateau could be increased. 4) the PEEP can be increased (lung units are easier to ventilate when they are kept open after the last breath).

If oxygenation must be improved: 1) all of the above techniques to improve ventilation will also improve oxygenation; 2) the inspired oxygen could be increased up to 100% for 12 to 24 hours (afterwhich oxygen toxicity is a concern) or up to 60% for prolonged periods of time; or 3) PEEP can be increased (increases transpulmonary pressure and functional residual capacity, and keeps small airways and alveoli open during the expiratory phase and improves ventilation and oxygenation). PEEP also minimizes the repetitive collapse and re-opening of small airways, a process which contributes to ventilator-induced injury. Positive end-expiratory pressure (PEEP) is available on many ventilators as an adjustable knob but can also be achieved by attaching a corrugated breathing tube to the exhalation port of the ventilator and placing the other end a few centimeters under water (the depth to which the end of the tube is submerged determines the airway pressure at the end of exhalation).