A Breath of Fresh Air – Clinical Applications of Oxygenation and Ventilation in the Emergency Room

Christin Reminga, DVM, DACVECC

Lungs allow the exchange of oxygen (O2) and carbon dioxide (CO2) between blood and room air. Respiratory gas transport is essential for cellular metabolism and thus organism sustainability, requiring ventilation, pulmonary gas exchange, and O2 transport to and from the tissues. Oxygenation and ventilation are distinct but interdependent physiological processes.

Ventilation is the movement of gas into and out of the alveoli and is under the control of brainstem respiratory centers in spontaneously breathing animals and skeletal muscle (diaphragm and intercostal muscles) generating the mechanical force responsible for lung expansion. Air from the atmosphere starts at the nares and is conducted through the nasopharynx, larynx, trachea, and into the mainstem bronchi. The bronchi split several times ending in the terminal respiratory units of the lung that are lined with alveoli. ***Minute Ventilation (MV)***, which is the total volume of air moved into and out of the lungs in one minute, is determined by the respiratory rate and tidal volume (or volume of air each breath). It is controlled by blood carbon dioxide levels, which alter cerebrospinal fluid pH that directly stimulates the respiratory center to alter ventilation in order to maintain CO2 within a narrow range. ***Hypercapnia*** is defined as an elevated arterial partial pressure of CO2 (PaCO2) and the body's response is to increase MV by hyperventilating and recruiting secondary muscles to aide in respiration. ***Hypocapnia*** is a low arterial CO2 and is combated by hypoventilation.

While ventilation can be thought of as the delivery system that presents oxygen-rich air to the alveoli, ***oxygenation*** is the process of delivering O2 from the alveoli to the tissues in order to maintain cellular activity. Oxygenation is a complex process that involves the respiratory, cardiovascular, hematologic, and cellular transport systems. The mode of O2 transport is by way of diffusion, where the O2 molecule passively travels from areas of high concentration to lower concentration. It diffuses from the alveoli into the erythrocytes and binds to hemoglobin in order to travel to the tissues, ultimately entering the cellular mitochondria for aerobic cellular metabolism. CO2 is then the main end product of this metabolism and, since it's accumulation results in systemic effects due to respiratory acidosis, it is promptly removed by the body by again diffusion into the plasma and exits the body by way of alveolar ventilation.

The rate of O2 and CO2 diffusion to and from the alveoli and mitochondria is dependent on both transmembrane partial pressures of that molecule as well as the solubility prosperities of that gas molecule in the tissues. That concentration gradient between high atmospheric O2 and lower venous O2 allows for rapid diffusion in the healthy lung. Hemoglobin (Hg) is an incredible structure that leads to

highly efficient oxygen transport to tissues. If it was up to plasma O2 concentrations alone, the O2 would fully saturate in the bloodstream very quickly in low concentrations and an animal would never be able to keep up with the demands of the tissues. Instead, the red blood cells contain many Hg molecules, each with up to 4 O2 binding sites, which continues to provide a new place for the O2 molecules to continue diffusing from the plasma. The attachment of one O2 molecule to its binding site results in a confirmational change of Hg, opening up the other 3 sites and making it even more efficient for the rest of the oxygen molecules to attach. This means that the relationship between the concentration of O2 dissolved in the plasma (PaO2) and the percent saturation of Hg (SaO2) with oxygen is actually non-linear, and is instead characterized by a sigmoid bend.

Types of Hypoxemic Hypoxia

Hypoxemic hypoxia: Caused by a fall in the PaO2 in the bloodstream and is primarily driven by disorders of respiration. Most common type of hypoxia.

1. **Low Inspired Oxygen (FiO2):** low inspired oxygen content can occur with anesthetized patients, patients in chambers that are not receiving ample fresh gas flow, smoke inhalation, and high altitudes. O2 supplementation resolves this form of hypoxia.
2. **Hypoventilation:** delivery of low volume inspired air to the alveoli causing hypercapnia. Supplementary O2 can bring more high rich oxygen to the alveoli but it doesn't affect the volume of oxygen in each breath so it may improve the PaO2 but may not resolve hypoxia or hypercapnia. Establishing an airway and providing improved MV is necessary.
3. **Ventilation/perfusion (V/Q) mismatch:** There are two main reasons for a mismatch, being either dead space ventilation or intrapulmonary shunting. This disorder tends to be the most common reason for poor oxygen delivery to tissues.
    a. **Dead space ventilation (V/Q >** 1): areas of the respiratory system that do not participate in gas exchange. ***Anatomic dead space*** is the conducting airways prior to respiratory units that participate in gas exchange. ***Physiologic dead space*** refers to areas in which inspired air reaches the respiratory units but is not able to equilibrate with the flowing capillary blood. The three main causes of physiologic dead space ventilation include pulmonary thromboembolism (PTE), decreased blood flow to the lungs, and alveolar overdistension preventing capillary blood flow during positive pressure ventilation (PPV). Supplemental O2 helps drive the diffuse gradient to improve increased uptake of O2 to the bloodstream.
    b. **Intrapulmonary shunting (V/Q < 1):** more perfusion than ventilation. Blood flow that is not exposed to ventilated areas cannot participate in gas exchange. There are two forms of this condition: **true shunts** and **venous admixture**.
        i. **True shunt:** complete lack of gas exchange (V/Q = 0). Does not improve with supplementary O2.
        ii. **Venous admixture (0 < V/Q < 1):** decreased ventilation to perfusion ratio due to incomplete equilibration between the capillary blood flow and alveolar O2

levels. Consider any form of alveolar and lower airway disease, including atelectasis as a cause. The other more normal lungs will benefit from O2 supplementation.

4. **Diffusion Impairment:** Any condition leading to thickening of the pulmonary interstitium because close contact is required to allow diffusion of molecules down their concentration gradient. This condition usually improves with supplementary O2.

Diagnostic Workup

Initial point of care testing should be conducted to better determine the cause of a respiratory disorder and include packed cell volume/total protein, complete blood count, blood chemistry, urine specific gravity, coagulation profile, and a blood gas analysis. The **venous blood gas** is useful to understand the patient's ventilation status and subsequent acid-base disturbances since the venous CO2 level (PvCO2) is directly proportional to the PaCO2 and MV, which quickly alters the acid-base balance. Hypercapnia results in a respiratory acidosis and hypocapnia causes a respiratory alkalosis. This values can be obtained from a direct venipuncture or a peripheral/central venous catheter. Venous O2 levels (PvO2) do not correlate to PaO2 levels so should not be interpreted. **Arterial blood gas** provides invaluable and specific data regarding the patient's respiratory status. The PaO2 and PaCO2 are directly measured and is the gold standard for the diagnosis for respiratory failure because it can quantify the severity of disease, and sometimes even allows categorization of the type of respiratory dysfunction. Preventing exposure of the sample to air in all blood gas measurements is important because it will rapidly falsely lower the CO2 measurement.
Additional testing include pulmonary thromboembolism testing, Baermann fecal exam, Bronchoscopy, thoracic radiographs, CT scan, ultrasound, pleural effusion and airway wash cytology/fluid analysis & culture, and infectious PCR testing.

Focal assessment of sonography for trauma (FAST) in patients with respiratory distress has gained popularity due to the increased availability of bedside ultrasounds and noninvasive nature of the exam. One can become easily proficient in both FASTthoracic scans and the newly developed VetBLUE (Veterinary Bedside Lung Exam) to examine the heart, lungs, and pleural space for concerns of left atrial enlargement and congestive heart failure, pericardial effusion, pulmonary edema, pneumothorax, pleural effusion, and sometimes pulmonary masses.

Machine Monitoring

Pulse-oximetry is a noninvasive tool that provides rapid, continuous assessment of oxygenation, allowing detection of minor changes in respiratory status. It measures the saturation of Hb with O2 using pulsatile flow detection (SpO2) which is dependent and correlates with PaO2 levels without obtaining an arterial blood gas. Normal value is 95% or above. Drastic changes in the patient's PaO2 level will result in only small changes in the SpO2. If SpO2 is 93% the patient is already hypoxemic (PaO2

80 mmHg) and SpO2 90% or below represents severe hypoxemia (correlated with a 60 mmHg PaO2). Caveats to this monitoring device is that it is not always tolerated in an awake patient, is no longer accurate once SpO2 reaches below 86% or patient had pigmented tissue, and does not read accurately when there is excessive motion, vasoconstriction, or severe anemia (PCV < 15%). It also does not provide the ventilatory status of the patient.

Capnography is a device that attaches to an endotracheal tube (ET) and measures the CO2 level on expiration. It reveals the CO2 level at every phase of the respiratory cycle and the number to best indicate alveolar CO2 levels (PACO2) is the end-tidal CO2 (ETCO2). Normal ETCO2 is 35-45 mmHg. The ETCO2 is directly correlated with the PaCO2 and PvCO2 levels. It's use is now considered standard of care for any critically ill intubated patient. It can be the first sign of inappropriate ET placement into the esophagus or respiratory arrest in patients under general anesthesia or getting PPV, because the reading will quickly drop substantially close to zero. This device is also utilized during CPCR, confirming adequate compressions and associated with improved patient return to spontaneous circulation when readings are 15 mmHg or above.

Intervention Strategies

Stabilizing animals suffering from hypoxemia and/or hypoventilation often require three main initiatives while starting the previously discussed diagnostics. That being anxiolytics/analgesics to decrease stress and improve MV, assure a patent airway and either re-establish or bypass the obstruction, and provide supplemental oxygen. Removal of large volume pleural effusions comes promptly after initial treatments.

Supplemental oxygen comes in many forms, by way of both invasive and non-invasive techniques. It is important to provide humidification immediately whenever bypassing the nasal passage and within a few hours whenever providing oxygen gas without bypassing efforts. Non-invasive forms of oxygen supplementation can be placed with little or no sedation or procedures, but they have limitations depending on the patient tolerance, disease process, and supply accessibility.

1. **Flow-by oxygen:** O2 tubing is placed close to the mouth or nose. Flow rates 1-5 L/min and provide FiO2 21-35%. Since it can cause a negligible improvement in FiO2 it is recommended only for the initial stabilization/assessment of the patient.
2. **Oxygen mask:** reservoir system that should be slightly larger than the muzzle/head to retain O2 levels, while minimizing accumulation of CO2, heat, and humidity. The flow rate is 2-10 L/min and provide FiO2 levels of 30-90%. Frequent monitoring is required to ensure placement and tolerance.
3. **Oxygen hoods:** commercially available or made in hospital. Flow rate is 2-10 L/min and provide FiO2 30-50%. It can be used for long-term management but larger panting patients can become hyperthermic.

4. **Oxygen cage:** come in many varieties from fully enclosed climate-controlled, CO2-scavenging, accurate FiO2 monitoring/adjusting units to collapsible portable chambers with no ability to scavenge CO2 or know or control FiO2 or humidity levels. Separate monitors can be purchased to monitor these important parameters. FiO2 levels can reach up to 60-90% depending on the manufacturer. Disadvantages of this device is the inability to handle the patient, rapid re-equilibration with room air when opening the door, difficult temperature control in large panting patients, and inability to hear patients during upper airway obstructions.
5. **Nasal prongs:** provides nasal supplementary oxygen for long-term management of patients that cannot tolerate a cage or hood. The flow rates can go up to 150 ml/kg/min to achieve FiO2 up to 70% when nasal breathing. Patients may not tolerate this type of O2 supplementation due to discomfort of oxygen through the nostril at higher rates, even with humidification. If the patient is open-mouth breathing or panting it may not provide adequate improvements in FiO2.
6. **Nasal catheters:** catheters can be placed with little or no sedation and allows for less dislodgement and potentially improved FiO2 rates (up to 70-80% with bilateral catheters in closed-mouth breathing). It is important to premeasure and advance the catheter into the ventral meatus to the point of the ocular medial canthus. Flow rates are identical to nasal prongs. Bilateral placement will improve FiO2.
7. **Nasopharyngeal catheters:** catheters can also be advanced farther down the ventral meatus to the level of the caudal nasopharynx. These catheters are premeasured at the mandibular ramus.
8. **Endotracheal intubation:** this is an invasive form of providing improved oxygenation and ventilation and is indicated for general anesthesia, hypoventilation causing hypoxemia, respiratory fatigue or apnea, and patients not responding to maximum levels of less invasive forms of O2 supplementation. Preparation is key before starting this intervention. Obtain all intubation supplies (even for difficult intubations), CPR crash cart, monitoring equipment, and appropriate medications, if able. Difficult intubations more often occur in cats, small patients, brachycephalic dogs, patients with suboptimal positioning, and those with oropharyngeal or orofacial obstructions, trauma, or other disease. A rapid assessment of the upper airway during intubation increases the changes of successful intubation and identification of abnormal structures, upper airway secretions, and dynamic functional changes of the upper airway.
9. **Transtracheal intubation:** this technique can be a life-saving procedure during an upper airway obstruction. It can also be used for O2 supplementation and a patent airway in an awake patient, to facilitate removal of airway secretions, to provide O2 and gas inhalant to patients undergoing upper airway procedures where ET tube placement is contraindicated, and to decrease anatomical dead space during PPV. Temporary procedures include tracheal needle insertion and catheterization or surgical tracheostomy. Cervical anatomy must be understood to avoid vital cervical structures.
    a. **Transtracheal catheterization:** Tracheal catheter placement is performed as a temporary means to establish partial control over the airway and provide oxygen.
    b. **Temporary tracheostomy:** A transtracheal tube placed that is the same diameter as an ET tube but bypassing the upper airway.

Pleural space disease results in respiratory distress due to the inability to fully insufflate the lungs. Stabilization is initiated the same as any other form of hypoxemia, with anxiolysis/analgesics and O2 supplementation. The additional immediate intervention is a therapeutic thoracocentesis if there is large volume fluid or air appreciated. Any patient exhibiting signs of respiratory distress and has a large volume of air or fluid in the pleural space warrants an emergency thoracocentesis prior to transport. Risks involved should be discussed with the client. If multiple procedures are required or a very large volume is to be removed it is best to consider a thoracostomy tube placement procedure. This is also utilized for medical management of a pyothorax, intrapleural chemotherapy, blood pleurodesis for a refractory pneumothorax, and post-thoracic surgeries.

References

- Harpster NK. Physical examination of the respiratory tract. In: Textbook of Respiratory Disease in Dogs and Cats. King LG, ed. St Louis: Saunders, 2004: pp 67–72.
- Tseng LW, Waddell LS. Approach to the patient in respiratory distress. Clin Tech Small Anim Pract. 2000;15(2):53–62.
- Sigrist NE, Adamik KN, Doherr MG, et al. Evaluation of respiratory parameters at presentation as clinical indicators of the respiratory localization in dogs and cats with respiratory distress. J Vet Emerg Crit Care 2011;21(1):13–23.
- Creedon, Burkitt J, Davis H. Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care. Chichester: Wiley-Blackwell, 2012.
- Silverstein D, Hopper K, eds. Small Animal Critical Care Medicine. St Louis: Saunders Elsevier, 2015.
- Ilkiw JE, Rose RJ, Martin ICA. A comparison of simultaneously collected arterial, mixed venous, jugular venous and cephalic venous blood samples and the assessment of blood-gas and acid-base status in the dog. J Vet Intern Med. 1991;5:294.
- Boysen SR, Lisciandro GR. The use of ultrasound for dogs and cats in the emergency room: AFAST and TFAST. Vet Clin North Am Small Anim Pract. 2013;43(4):773–97.
- Lisciandro G. The VetBLUE lung scan. In: Focused Ultrasound Techniques for the Small Animal Practitioner. Ames: Wiley & Sons, 2014: pp 166–88.
- Fairman NB. Evaluation of pulse oximetry as a continuous monitoring technique in critically ill dogs in the small animal intensive care unit. J Vet Emerg Crit Care 1992;2(2):50–6.
- Hendricks JC, King LG. Practicality, usefulness, and limits of pulse oximetry in critical small animal patients. J Vet Emerg Crit Care 2003;l3(1):5–12.
- Raffe M. End tidal capnography. In: Textbook of Respiratory Disease in Dogs and Cats. King LG, ed. St Louis: Saunders, 2004: pp 198–201.
- Sanders AB, Kern KB, Otto CW, et al. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation. A prognostic indicator for survival. J Am Med Assoc. 1989;262:1347–51.

- Tseng LW, Drobatz KJ. Oxygen supplementation and humidification. In: Textbook of Respiratory Disease in Dogs and Cats. King LG, ed. St Louis: Saunders, 2004: pp 205–13.
- Loukopoulos P, Reynolds W. Comparative evaluation of oxygen therapy techniques in anaesthetized dogs: intranasal catheter and Elizabethan collar canopy. Aust Vet Pract. 1996;26:199–204.
- Mort TC. Preoxygenation in critically ill patients requiring emergency tracheal intubation. Crit Care Med. 2005;33(11):2972–5.
- Colley P, Huber M, Henderson R. Tracheostomy techniques and management.Compend Contin Educ Pract Vet. 1999;21(1):44–52.
- Janocik SE, Roy Ted, Killeen TR. Re-expansion pulmonary edema: a preventable complication. J Ky Med Assoc. 1993;91:143–6.
- Hawkins EC, Fossum TW. Medical and surgical management of pleural effusion. In: Kirk's Current Veterinary Therapy, vol. 13. Bonagura JD, ed. Philadelphia: WB Saunders, 2000.
- Sigrist NE. Thoracostomy tube placement and drainage. In: Small Animal Critical Care Medicine. Silverstein DC, Hopper K, eds. Philadelphia: Saunders, 2009: pp 134–7.