Hypoxemia; Causes and Treatment

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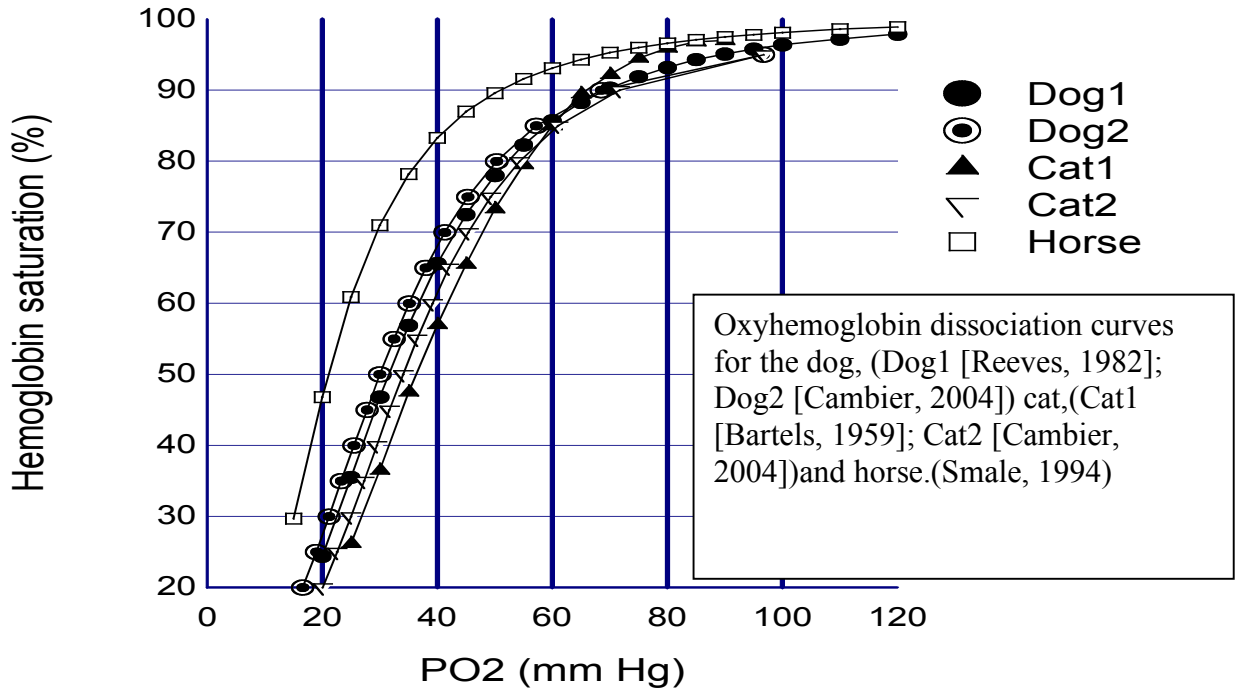
Assessing blood oxygenation

Blood oxygen can be expressed in three different ways: the partial pressure of oxygen dissolved in the plasma (PO_2; units = mm Hg), the percent saturation of the hemoglobin (SO_2; units = %), and the whole blood oxygen content ($ContentO_2$; units = mls of oxygen per 100 mls whole blood).

The Partial Pressure of Oxygen (PO_2)

The PO_2 is the partial pressure (the vapor pressure) of oxygen dissolved in solution in the plasma and is measured with a blood gas machine 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 arterial PO_2 (PaO_2) is a measure of the ability of the lungs to efficiently move oxygen from the atmosphere to the lungs. The normal PaO_2 at sea level ranges between 80 and 110 mm Hg. Hypoxemia is usually defined as a $PaO_2 < 80$ mm Hg. A PaO_2 of less than 60 mm Hg marks severe hypoxemia and treatment should be implemented. There are only two treatments: 1) increase the inspired oxygen concentration; 2) mechanical ventilation; or some combination of the two.

Venous PO_2 reflects tissue PO_2 and bears no correlation to arterial PO_2. Mixed or central venous PO_2 ranges between 40 and 50 mm Hg. Values below 30 mm Hg may be caused by anything that decreases the delivery of oxygen to the tissues (hypoxemia, low cardiac output, vasoconstriction); values above 60 mm Hg (while breathing room air) suggest reduced tissue uptake of oxygen (shunting, septic shock, metabolic poisons). Venous blood for such evaluations must be from a central vein such as the jugular, anterior vena cava, or pulmonary artery. Continuous mixed-venous oxygen-hemoglobin saturation can be measured via a pulmonary artery catheter containing a fiberoptic infrared light source. The reflected light beam is proportional to the degree of hemoglobin oxygenation.



Oxyhemoglobin dissociation curves for the dog, (Dog1 [Reeves, 1982]; Dog2 [Cambier, 2004]) cat,(Cat1 [Bartels, 1959]; Cat2 [Cambier, 2004])and horse.(Smale, 1994)

Oxyhemoglobin saturation

Hemoglobin saturation measures the percent saturation of the hemoglobin and is related to the PaO_2 by a sigmoid curve. The clinical information derived from the measurement of hemoglobin saturation (SaO_2) is similar to that obtained from a PaO_2 measurement in that they are both a measure of the ability of the lung to deliver oxygen to the blood stream. The "numbers of concern" are, however, different:

Correlation between PaO_2 and SaO_2.

PaO_2	SaO_2	Importance
> 80	> 95	Normal
< 60	< 90	Serious hypoxemia
< 40	< 75	Very serious hypoxemia

The percent hemoglobin saturation can be measured with a bench top oximeter or a pulse oximeter, or it can be extrapolated from the measured PO_2 via a standard oxyhemoglobin dissociation curve. Oximetry is based upon the pattern of red to infra-red light absorption of hemoglobin: oxyhemoglobin, reduced hemoglobin, methemoglobin, and carboxyhemoglobin each absorb light differently. At least one wave length of light, preferably one that maximizes the difference between the hemoglobin species of interest and the others, is required to identify each species of hemoglobin. Pulse oximeters use only two wave lengths (660 and 940 nm) and are designed to measure oxygenated and unoxygenated hemoglobin. If methemoglobin or carboxyhemoglobin are present in high concentrations, they will absorb light and will impact the measurement made by the pulse oximeter. Due to the biphasic absorption of methemoglobin at both the 660 and 940 nm wavelengths, abnormal accumulations of this hemoglobin species tends to push the pulse oximeter reading toward 85% (underestimating measurements when SaO_2 is above 85% and overestimating it when below 85%). Carboxyhemoglobin absorbs light like oxyhemoglobin at 660 nm but hardly at all at 940 nm and this would increase the apparent oxyhemoglobin value. Fetal hemoglobin produces very little effect on measured hemoglobin saturation. Indocyanine green dye and methylene blue dye absorb light and will generate falsely low saturation measurements

Tissue, venous and capillary blood, non-pulsatile arterial blood, and skin pigment also absorb infrared light. Pulse oximeters have different ways of separating this background absorption from the change in light absorbance associated with pulsatile arterial blood. There is a fairly narrow spectrum of wavelengths that both pass through skin and yet are absorbed by hemoglobin. Differences in tissue absorption or scatter of light, different thicknesses of tissue, smaller pulsatile flow patterns and small signal to noise ratios, and incompletely compensated light emitting diodes may account some of the inaccuracies associated with pulse oximeters. Inaccuracies may also generate from baseline read errors (motion), differences in sensor location, and electrical or optical interference. The accuracy of a pulse oximeter is greatest within the range of 80 and 95%, and is determined by the accuracy of the empirical formula that is programmed into the instrument.¨ For most clinical purposes, most pulse oximeters are sufficiently accurate *approximations* of hemoglobin saturation, but their accuracy should be verified by an in vitro standard. There are substantial bias and precision variations and response times between products at different levels of saturation.

Pulse oximeters attach to a patient externally (tongue, lips, tail, toenail). A pulse oximeter is an automatic, continuous, audible monitor of mechanical cardiopulmonary function. It specifically measures pulse rate and hemoglobin saturation, and requires reasonable pulmonary and cardiovascular function in order to achieve a measurement. One of the common reasons for poor instrument performance has been peripheral vasoconstriction; the instrument will not be able to pick up a pulse. Its value as an ongoing monitor in detecting hypoxemia has been established. Accuracy should be verified from time to time with an arterial blood gas measurement.

SaO_2 may not be too discriminating when a animal is breathing an enriched oxygen mixture since such measurements would be positioned on the upper plateau of the dissociation curve. The difference between a PaO_2 of 500 and 100 mm Hg in an animal breathing 100% oxygen is very important; the corresponding decrease in SaO_2, from 99 to 98%, would hardly be noticed.

Whole blood oxygen content and oxygen delivery

Oxygen content is dependent upon both hemoglobin concentration and PO_2. Oxygen content is calculated by the formula: $(1.34 \times Hb \times SaO_2) + (0.003 \times PO_2)$ or may be measured with a galvanic cell quantitative analyzer. The relationship between oxygen content and PO_2 is also defined by a sigmoid curve. Hemoglobin concentration is quantitatively the most important contributor to oxygen content (anemia is a far more potent cause of decreased oxygen content than is hypoxemia). The partial pressure of oxygen is important because it provides the driving pressure for the flow of oxygen molecules from the plasma to the mitochondria. Content is important as a reservoir of oxygen to buffer the decrease in PO_2 that would occur when oxygen molecules diffuse out of the plasma. Oxygen delivery is the product of oxygen content and cardiac output.

The PaO_2 at which human hemoglobin is 50% saturated (P_{50}) is about 27 mm Hg. P_{50} is a common way to define the position of the curve; whether it is shifted to the left (a lower P_{50} value due to higher hemoglobin affinity for oxygen) or to the right (a higher P_{50} value due to lower hemoglobin affinity for oxygen). The P_{50} for canine hemoglobin is about 29 mm Hg. The P_{50} for feline hemoglobin, however, is about 36 mm Hg. This represents a rightward shift of the oxyhemoglobin dissociation curve for the cat compared to the dog. Like hemoglobin saturation, oxygen content does not increase much when the PO_2 is raised above 100 mm Hg; the hemoglobin is mostly full and further increases in content are attributed mostly to an increase in dissolved oxygen in the plasma.

Effect of anemia, hypoxemia, and oxygen breathing on PO_2, SO_2, and $ContentO_2$

Condition	F_IO_2	PO_2 (mm Hg)	SO_2 (%)	Hemoglobin (gm/dl)	$ContentO_2$ (ml/dl)
Normal	0.21	100	98	15	19.9
Anemia	0.21	100	98	5	6.9
Hypoxemia	0.21	50	85	15	17.3
Hyperoxemia	1.0	500	99.9	15	21.6
Anemia & hyperoxemia	1.0	500	99.9	5	8.2
Anemia & hypoxemia	0.21	50	85	5	5.8

The treatment for excessive anemia is an infusion of a hemoglobin-containing solution. However, in the interim, between recognition of severe anemia and implementation of the transfusion, does an enriched inspired oxygen concentration benefit the patient? Clearly, even 100% inspired oxygen does not increase the oxygen content very much in the anemic patient and not nearly enough to provide all of the oxygen requirements of the patient. Anemia causes problems for the patient when the blood oxygen content becomes insufficient to meet the metabolic requirements of oxygen consumption. It is no necessary to meet the entire oxygen consumption requirements of the animal with the oxygen therapy. It is only necessary to increase the oxygen content enough to move the animal from a little below the "death line" to a little above it. Sometimes a little bit of help can make a lot of difference to a patient and oxygen therapy is recommended in the anemic patient.

Oxygen delivery is the product of oxygen content and cardiac output. Animals can tolerate a decrease in oxygen content if they can increase their cardiac output to compensate. Animals tolerate anemia and poor cardiac output poorly. Oxygen delivery needs to be sufficient to meet the consumption requirements of the patient. Normally oxygen delivery far exceeds oxygen consumption. Oxygen extraction normally ranges between 20 and 25% of the oxygen delivery. Mixed venous oxygen represents the balance between whole body oxygen delivery and oxygen consumption. Mixed or central venous PO_2 ranges between 40 and 50 mm Hg in normal dogs. When oxygen delivery is decreased (low cardiac output, anemia, hypoxemia,, vasoconstriction), the tissues continue to "draw" their normal amount of oxygen and so oxygen extraction increases and venous oxygen decreases. Venous PO_2 values below 30 mm Hg are usually attributed to excessively low oxygen delivery, but could be caused by high oxygen consumption. Values below 20 mm Hg should be considered life-threatening. When oxygen delivery finally becomes too low to support oxidative phosphorylation, lactic acidosis ensues. Venous PO_2 values above 60 mm Hg (while breathing room air) are primarily suggestive of reduced tissue uptake of oxygen (shunting, septic shock, metabolic poisons, hypothermia), but could also be attributed to high oxygen delivery .

Oxygen delivery (DO_2) (ml/min/M^2) resulting from various combinations of packed cell volume (PCV) and cardiac output (Q) (assuming normal lung function and a PaO_2 of 100 mm Hg)(normal range for Q is 3.5 to 5.5).*

	Q = 6.5	5.5	4.5	3.5	2.5
PCV = 40	1155	977	799	621	444
30	873	739	604	470	336
25	731	618	506	394	281
20	588	498	407	317	226
15	446	378	309	240	172
10	304	257	210	164	117

*It has been recommended to maintain DO_2 above 550-600 ml/min/M^2. Anemic patients can maintain oxygen delivery if they can increase cardiac output to compensate. Anemia and low cardiac output combine to produce very low values for oxygen delivery.

Mechanisms of hypoxemia

Hypoxemia may be caused by a low inspired oxygen concentration, hypoventilation, or venous admixture. A low inspired oxygen must be considered any time an animal is attached to mechanical apparatus such as a face mask, Bain's circuit, anesthetic machine, ventilator, or in an enclosed environment such as oxygen cage or anesthetic induction chamber. Hypoventilation is defined by an elevated $PaCO_2$. Venous admixture represents a reduced efficiency of lung oxygenating ability. Venous admixture is all of the ways in which venous blood can get from the right side of the circulation to the left side of the circulation without being properly oxygenated; this less-than-optimal oxygenated blood then admixes with optimally-arterialized blood flowing from the more normal lung units and dilutes its oxygen content. There are 4 causes of venous admixture: low ventilation/perfusion regions; small airway and alveolar collapse; diffusion defect; anatomic right-to-left shunts. A fifth cause of venous admixture is low venous oxygen content secondary to low cardiac output and increased tissue oxygen extraction.

Low ventilation/perfusion regions occurs secondary to airway narrowing from bronchospasm (reflex or disease induced), fluid accumulation along the walls of the lower airways, or epithelial edema. The effect is hypoventilation of the involved lung units relative to their blood flow and suboptimal arterialization of the blood flowing through the area. This is a common mechanism of hypoxemia in mild to moderate pulmonary disease. This mechanism of hypoxemia is very responsive to oxygen therapy because even though the lung unit is being hypoventilated, the high alveolar oxygen concentrations normalize the oxygenation of the blood flowing through the area (PaO_2 values may reach expected values (500 mmHg) when breathing 100%). A low V/Q disturbance could also be attributed to an increase in blood flow to the area. This may be part of the explanation for hypoxemia in pulmonary thrombo-emolism.

Small airway and alveolar collapse (a no ventilation but still-perfusion scenario) is caused by spontaneous collapse of small airways and alveoli caused by either positional stasis or by an increase in airway fluids which increases surface tension and collapsing tendency. The effect is that the blood flowing through the area will not be arterialized at all (it will remain venous blood when it exits the gas exchange area). This is a common mechanism of hypoxemia in moderate to severe pulmonary disease. This mechanism is not responsive to oxygen therapy - oxygen cannot get down to the gas exchange area. These small airways and alveoli must be opened by positive pressure ventilation if they are to become functional gas exchange units.

Diffusion impairment, due to a thickened respiratory membrane, is an uncommon cause of hypoxemia. I, however, the type 1 pneumocytes are damaged by inhalation inflammatory injury, they are replacement by the thick, cuboidal type 2 pneumocytes (which will eventually become type 1 pneumocytes). This mechanism of hypoxemia is partially responsive to oxygen therapy.

Anatomical shunts are right-to-left extra- or intrapulmonary shunts where the blood flows from the right side of the circulation to the left side without ever coming into contact with a functional gas exchange unit. This is not a common mechanism of disease in clinical medicine. It is responsive to neither oxygen therapy nor positive pressure ventilation, and requires surgical correction.

In pulmonary parenchymal disease, lungs often perform poorly for the purposes of oxygenation and yet are well able to eliminate carbon dioxide (hypocapnic hypoxemia). This is due to the fact that alveolar-capillary units that are working relatively well can compensate for those that are working relatively poorly with respect to carbon dioxide elimination, but not for oxygen intake. The reason for this is that at the normal alveolar/arterial blood PCO_2 values are positioned on the steep, linear portion of the PCO_2/CO_2 content curve and an increase in alveolar ventilation causes a proportional decrease in CO_2 content. In contrast, normal alveolar/arterial blood PO_2 values are

positioned on the upper, flat portion of the oxyhemoglobin dissociation curve and an increase in alveolar ventilation causes very little increase in O2 content.

Estimating the magnitude of venous admixture

1) The alveolar - arterial PO_2 gradient (A-a PO_2) is the difference between the calculated alveolar partial pressure of oxygen (P_AO_2) and the measured arterial partial pressure of oxygen (PaO_2). Alveolar PO2 is calculated as inspired PO_2 (P_IO_2) – $PaCO_2$ (1.1). The 1.1 is 1/RQ, assuming RQ = 0.9, which is a reasonable average for critically ill patients. Inspired PO_2 is not the oxygen concentration of air that is breathed in; it is the concentration of oxygen in the air entering the alveoli after it has been fully humidified. It is calculated as barometric pressure – 50) x F_IO_2 (which is the concentration of oxygen [expressed as a fraction] in the air that is breathed in). The 50 is the vapor pressure of water when the air is 100% saturated at body temperature. At sea level, breathing 21% oxygen, P_IO_2 is invariably about 150 mm Hg. The A-a PO_2 is normally about 10 mm Hg when breathing 21% oxygen at sea level and is about 100 mm Hg when breathing 100% oxygen. If the calculated A-a PO_2 is greater than 15 mm Hg when the animal is breathing room air or greater that 150 mm Hg when the animal is breathing 100% oxygen, the animal has venous admixture. The greater the A-a PO_2 , the greater the venous admixture. The expected A-a PO_2 at intermediate inspired oxygen concentrations has not been established and must be extrapolated.

2) There are only four gases of note in the alveoli (nitrogen, water vapor, oxygen, and carbon dioxide). Barometric pressure and the partial pressures of nitrogen and water vapor do not change. Blood flowing through the lungs adds carbon dioxide to and removes oxygen from the alveoli at a rate that is comparable to metabolic production and consumption of these two gases. Alveolar PCO_2 and PO_2 are, therefore, approximately reciprocally related in the normal lung. A change in $PaCO_2$ should be associated with an opposite and approximately equal (1.0 to 1.2 x) change in the PaO_2. The PaO_2 + $PaCO_2$ added value for arterial blood should be about 140 ± 20 mm Hg, when the animal is breathing 21% oxygen at sea level. A discrepancy develops with progressive venous admixture such that the added PaO_2 and $PaCO_2$ values drop progressively below 120 mm Hg; the greater the discrepancy between the added value and 120 mm Hg, the greater is the venous admixture.

3) When breathing 21% oxygen, changes in $PaCO_2$ have an important impact on PaO_2 and must be taken into account when calculating the expected PaO_2. With progressively higher inspired oxygen concentrations, changes in $PaCO_2$ have a progressively less important effect on PaO_2 and, for clinical purposes, can legitimately be ignored at inspired oxygen concentrations over about 40%. A common rule-of-thumb is that the anticipated PaO_2 should be at least 5 times the inspired oxygen concentration (50% x 5 = an anticipated PaO_2 > 250 mm Hg). If the PaO_2 is only 3 to 5 x the inspired oxygen concentration, a mild oxygenating inefficiency exists; between 2 and 3 x = moderate lung inefficiency; while < 2 = severe venous admixture.

The PaO_2/F_IO_2 ratio is frequently used in the medical literature and represents this same idea except F_IO_2 is expressed as a fraction instead of a whole number (0.50 instead of 50%). This represents only a decimal point change in the dividend. In normal lungs the PaO_2/FiO_2 ratio is > 500 mm Hg; values between 300 and 500 represent mild oxygenating inefficiency; values between 200 and 300 represent moderate lung inefficiency; and values below 200 represent severe venous admixture.

4) If mixed venous blood can be obtained, the venous admixture can be calculated: $Q_S/Q_T = (CcO_2 - CvO_2) / (CcO_2 - CaO_2)$. Q_S/Q_T = venous admixture expressed as a percent of cardiac output that would have to traverse the lung venous blood, assuming the rest is optimally arterialized. CcO_2 = oxygen content of end-capillary blood; CvO_2 = oxygen content of mixed venous blood; and CaO_2 = oxygen content of arterial blood. Oxygen content (ml/dl) is calculated

as: (1.34 x Hb x SO_2) + (0.003 x PO_2) where SO_2 is percent hemoglobin saturation with oxygen. Venous admixture is normally less than 5% of the cardiac output; values over 10% are considered to be increased. Venous admixture may increase to over 50% in diffuse lung disease.

Treatment considerations

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 or anatomical shunt.

A high inspired oxygen concentration can easily be attained with a face mask. Dyspneic patients commonly do not tolerate a tight fitting face mask, in which case 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 or glued 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 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.

The effectiveness of the oxygen therapy is not judged to be effective, a more efficient means of providing oxygen therapy could be attempted, but if ineffective, positive pressure ventilation may be efficacious.

Positive pressure ventilation

Positive pressure ventilation (PPV) is indicated whenever an animal cannot ventilate adequately due to neuromuscular disease or pulmonary parenchymal disease, or when a patient is not sufficiently responsive to oxygen therapy. PPV improves blood oxygenating efficiency of the lungs, which allows for the reduction of the inspired oxygen concentration. PPV is also indicated when the animal is having to work excessively hard to breath. Muscle fatigue and exhaustion causes these patients to deteriorate rapidly and they are very difficult to resuscitate.

The goals 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.

Airway access is usually accomplished with an orotracheal tube. Unless the central nervous system is severely depressed, heavy sedation or light general anesthesia is usually required to introduce and maintain an endotracheal tube. Notwithstanding co-existent cardiovascular disease, any fast acting anesthetic would suffice for the introduction of the tube (ketamine/diazepam; propofol; etomidate; or thiopental). A tracheostomy tube is used if the animal is quadriplegic or there is reason to believe that the ventilation procedure can be accomplished without the use of drugs. Weaning from ventilator support will be facilitated if you do not have to simultaneously

wean from the sedative drugs. A commercial tracheostomy tube with an inner cannula is superior to a single lumen endotracheal tube. The inner cannula can be removed for easy cleaning.

The general guidelines for positive pressure ventilation of animals with relatively normal lungs(regardless of the method or brand of ventilator utilized) are: 1) a peak proximal airway pressure of 10 to 20 cm H_20; 2) a tidal volume of 10 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 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 should receive 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 needs of the patient or 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); 2) the tidal volume should probably not be increased in an animal with diffuse lung disease. Pulmonary disease is associated with a reduced vital capacity (fig. 1) due to a reduced inspiratory and expiratory reserve volume. What would be a normal tidal volume for a normal lung could easily over-distend the reduced number of remaining lung units, contributing to volutrauma. Protective lung strategies currently aim for very small tidal volumes, e.g. 5 ml/kg; 3) the ventilatory cycle rate could be increased in a step-wise fashion up to 60 breaths per minute; 4) the inspiratory time or the inspiratory plateau could be increased. The inspiratory/expiratory [I/E] ratio must allow suffieicent time for exhalation of all of the last breath, otherwise air trapping and auto-PEEP will occur; and 5) the PEEP can be increased. Lung units are easier to ventilate when they are kept open after the last breath rather than having to start from a collapsed position.

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 short periods of time or up to 60% for prolonged periods of time; or 3) the end-expiratory pressure could be increased up to 20 cm H_20. PEEP 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.

Canine oxyhemoglobin dissociation curve*

PO2 (mmHg)	SO2 (%)	Oxygen content (ml/dl) Hb**	dslvd**	total	PO2 (mmHg)	SO2 (%)	Oxygen content (ml/dl) Hb	dslvd	total	PO2 (mmHg)	SO2 (%)	Oxygen content (ml/dl) Hb	dslvd	total
20	24.4	4.90	0.06	4.96	71	90.7	18.23	0.21	18.44	102	96.6	19.41	0.31	19.72
22	28.7	5.77	0.07	5.84	72	91.0	18.30	0.22	18.51	103	96.7	19.43	0.31	19.74
24	33.2	6.68	0.07	6.75	73	91.3	18.36	0.22	18.58	104	96.8	19.45	0.31	19.76
26	37.8	7.60	0.08	7.68	74	91.7	18.42	0.22	18.64	105	96.8	19.47	0.32	19.78
28	42.3	8.51	0.08	8.59	75	91.9	18.48	0.23	18.71	106	96.9	19.48	0.32	19.80
30	46.8	9.40	0.09	9.49	76	92.2	18.54	0.23	18.77	107	97.0	19.50	0.32	19.82
32	51.0	10.25	0.10	10.35	77	92.5	18.59	0.23	18.82	108	97.1	19.51	0.32	19.84
34	55.0	11.06	0.10	11.16	78	92.8	18.64	0.23	18.88	109	97.2	19.53	0.33	19.86
36	58.8	11.82	0.11	11.93	79	93.0	18.69	0.24	18.93	110	97.2	19.54	0.33	19.87
38	62.3	12.53	0.11	12.64	80	93.2	18.74	0.24	18.98	112	97.4	19.57	0.34	19.91
40	65.6	13.18	0.12	13.30	81	93.5	18.79	0.24	19.03	114	97.5	19.60	0.34	19.94
42	68.5	13.78	0.13	13.90	82	93.7	18.83	0.25	19.08	116	97.6	19.62	0.35	19.97
44	71.3	14.32	0.13	14.46	83	93.9	18.87	0.25	19.12	118	97.7	19.65	0.35	20.00
46	73.7	14.82	0.14	14.96	84	94.1	18.91	0.25	19.16	120	97.9	19.67	0.36	20.03
48	76.0	15.27	0.14	15.42	85	94.3	18.95	0.26	19.20	122	98.0	19.69	0.37	20.05
50	78.0	15.69	0.15	15.84	86	94.5	18.99	0.26	19.24	124	98.0	19.71	0.37	20.08
52	79.9	16.06	0.16	16.21	87	94.6	19.02	0.26	19.28	126	98.1	19.73	0.38	20.10
54	81.6	16.39	0.16	16.55	88	94.8	19.05	0.26	19.32	128	98.2	19.74	0.38	20.13
56	83.1	16.70	0.17	16.86	89	95.0	19.09	0.27	19.35	130	98.3	19.76	0.39	20.15
58	84.4	16.97	0.17	17.15	90	95.1	19.12	0.27	19.39	140	98.6	19.82	0.42	20.24
60	85.7	17.22	0.18	17.40	91	95.3	19.15	0.27	19.42	150	98.9	19.88	0.45	20.33
61	86.2	17.34	0.18	17.52	92	95.4	19.18	0.28	19.45	160	99.1	19.91	0.48	20.39
62	86.8	17.45	0.19	17.63	93	95.5	19.20	0.28	19.48	170	99.2	19.94	0.51	20.45
63	87.3	17.55	0.19	17.74	94	95.7	19.23	0.28	19.51	180	99.3	19.97	0.54	20.51
64	87.8	17.65	0.19	17.84	95	95.8	19.26	0.29	19.54	190	99.4	19.99	0.57	20.56
65	88.3	17.74	0.20	17.94	96	95.9	19.28	0.29	19.57	200	99.5	20.00	0.60	20.60
66	88.7	17.84	0.20	18.03	97	96.0	19.30	0.29	19.60	300	99.9	20.07	0.90	20.97
67	89.2	17.92	0.20	18.12	98	96.2	19.33	0.29	19.62	400	99.9	20.09	1.20	21.29
68	89.6	18.00	0.20	18.21	99	96.3	19.35	0.30	19.65	500	100.0	20.09	1.50	21.59
69	90.0	18.08	0.21	18.29	100	96.4	19.37	0.30	19.67	600	100.0	20.10	1.80	21.90
70	90.3	18.16	0.21	18.37	101	96.5	19.39	0.30	19.69	700	100.0	20.10	2.10	22.20

PO2 = partial pressure of oxygen; SO2 = hemoglobin saturation; *from Reeves, J Appl Physiol 1982;53:87 (((38848/(PO2^3+202*PO2+1.17*PO2^2))+1)^-1)*100; **Hb = oxygen bound to hemoglobin; assumes a hemoglobin concentration of 15 gm/dl (Hb x 1.34 x saturation); ***dslvd = oxygen in solution (0.003 x PO2)

Feline oxyhemoglobin dissociation curve*

PO2 (mmHg)	SO2 (%)	Oxygen content (ml/dl) Hb**	dslvd**	total	PO2 (mmHg)	SO2 (%)	Oxygen content (ml/dl) Hb	dslvd	total	PO2 (mmHg)	SO2 (%)	Oxygen content (ml/dl) Hb	dslvd	total
20	19.9	2.94	0.06	3.00	71	89.7	13.23	0.21	13.44	102	95.5	14.08	0.31	14.39
22	23.7	3.50	0.07	3.56	72	90.1	13.28	0.22	13.49	103	95.6	14.10	0.31	14.41
24	27.8	4.10	0.07	4.18	73	90.4	13.33	0.22	13.55	104	95.7	14.11	0.31	14.42
26	32.2	4.74	0.08	4.82	74	90.7	13.37	0.22	13.60	105	95.8	14.12	0.32	14.44
28	36.6	5.40	0.08	5.48	75	91.0	13.42	0.23	13.64	106	95.9	14.13	0.32	14.45
30	41.1	6.06	0.09	6.15	76	91.3	13.46	0.23	13.69	107	95.9	14.14	0.32	14.46
32	45.5	6.71	0.10	6.81	77	91.6	13.50	0.23	13.73	108	96.0	14.15	0.32	14.48
34	49.9	7.35	0.10	7.45	78	91.9	13.54	0.23	13.77	109	96.1	14.16	0.33	14.49
36	54.0	7.96	0.11	8.07	79	92.1	13.58	0.24	13.81	110	96.2	14.17	0.33	14.50
38	57.9	8.53	0.11	8.65	80	92.3	13.61	0.24	13.85	112	96.3	14.19	0.34	14.53
40	61.5	9.07	0.12	9.19	81	92.6	13.64	0.24	13.89	114	96.4	14.21	0.34	14.55
42	64.9	9.57	0.13	9.70	82	92.8	13.68	0.25	13.92	116	96.5	14.23	0.35	14.57
44	68.0	10.03	0.13	10.16	83	93.0	13.71	0.25	13.95	118	96.6	14.24	0.35	14.60
46	70.9	10.45	0.14	10.58	84	93.2	13.73	0.25	13.99	120	96.7	14.26	0.36	14.62
48	73.4	10.82	0.14	10.97	85	93.4	13.76	0.26	14.02	122	96.8	14.27	0.37	14.63
50	75.8	11.17	0.15	11.32	86	93.5	13.79	0.26	14.05	124	96.9	14.28	0.37	14.65
52	77.9	11.48	0.16	11.63	87	93.7	13.81	0.26	14.07	126	97.0	14.29	0.38	14.67
54	79.8	11.76	0.16	11.92	88	93.9	13.84	0.26	14.10	128	97.0	14.30	0.38	14.69
56	81.5	12.01	0.17	12.17	89	94.0	13.86	0.27	14.13	130	97.1	14.31	0.39	14.70
58	83.0	12.23	0.17	12.40	90	94.2	13.88	0.27	14.15	140	97.4	14.36	0.42	14.78
60	84.3	12.43	0.18	12.61	91	94.3	13.90	0.27	14.18	150	97.6	14.39	0.45	14.84
61	85.0	12.53	0.18	12.71	92	94.5	13.92	0.28	14.20	160	97.8	14.42	0.48	14.90
62	85.6	12.61	0.19	12.80	93	94.6	13.94	0.28	14.22	170	98.0	14.44	0.51	14.95
63	86.1	12.70	0.19	12.89	94	94.7	13.96	0.28	14.24	180	98.1	14.46	0.54	15.00
64	86.7	12.78	0.19	12.97	95	94.8	13.98	0.29	14.26	190	98.2	14.47	0.57	15.04
65	87.2	12.85	0.20	13.05	96	94.9	14.00	0.29	14.28	200	98.3	14.49	0.60	15.09
66	87.7	12.92	0.20	13.12	97	95.1	14.01	0.29	14.30	300	98.8	14.56	0.90	15.46
67	88.1	12.99	0.20	13.19	98	95.2	14.03	0.29	14.32	400	99.0	14.60	1.20	15.80
68	88.6	13.05	0.20	13.26	99	95.3	14.04	0.30	14.34	500	99.2	14.62	1.50	16.12
69	89.0	13.11	0.21	13.32	100	95.4	14.06	0.30	14.36	600	99.3	14.64	1.80	16.44
70	89.4	13.17	0.21	13.38	101	95.5	14.07	0.30	14.37	700	99.4	14.65	2.10	16.75

PO2 = partial pressure of oxygen; SO2 = hemoglobin saturation; *derived by iterative processing of the data of Cambier Res Vet Sci 2004;77:83. 100*((((-2554.074)*(PO2))+((387.8873)*(PO2)^2)+((-15.12568)*(PO2)^3)+((PO2)^4)) (((-437538.2))+((58785.11)*(PO2))+((245.5008)*(PO2)^2)+ ((-10.18485)*(PO2)^3)+((PO2)^4))); **Hb = oxygen bound to hemoglobin; assumes a hemoglobin concentration of 11 gm/dl (Hb x 1.34 x saturation); ***dslvd = oxygen in solution (0.003 x PO2)