Inhalation/Inspiration is initiated by the respiratory center in the brain and is normally triggered by an increased level of PaCO₂ (carbon dioxide in the arterial blood, normal = 35-45 mmHg). The intercostal muscles and diaphragm are stimulated, expanding the thorax, creating negative pressure, causing the lungs to expand. Air then moves through the breathing passages into the alveoli. After the lungs reach an adequate volume, the brain signals the stop of active respiration which causes the intercostal muscles and diaphragm to relax and exhalation/expiration occurs passively. During exhalation, PaCO₂ begins to increase again inhalation is initiated again. Respiratory rate (RR) is defined by the number of breaths taken in one minute. On expiration, there is still some air left in the lungs to prevent the alveoli from collapsing. This is known as the functional residual capacity (FRC).

Ventilation, in the anesthetized patient, is the physical movement of air and anesthetic gases into and out of the lungs and upper respiratory passageways. Gas exchange occurs at the alveolar-capillary membrane when oxygen moves into and carbon dioxide moves out of the bloodstream. The volume of gas that passes in and out of the lungs in one single breath is referred to as tidal volume (TV). The total amount of gas that moves in and out of the lungs in one minute is defined as respiratory minute volume (TV x RR).

There are 2 components of the anesthetized patient that are considered dead space volume where there is little or no diffusion of oxygen and carbon dioxide through membranes. Anatomical dead space refers to the nose, nasal passage, nasopharynx and trachea. This space assists in humidifying and tempering inhaled air and it cools the body as in panting. Panting is predominantly dead space ventilation due to an increased RR and decreasing TV, which keeps alveolar ventilation almost constant. This is the reason many patients that pant under anesthesia very often wake up. Mechanical dead space is related to equipment that adds to the anatomical dead space of the patient by extending the airway. This can include malfunctioning, missing or stuck exhalation one-way valve, improper filling of CO2 absorbent, cracked inner tube of a coaxial circuit, using too many adaptors between endotracheal tube and breathing system, and endotracheal tube extending beyond the patient’s incisors.

Ventilation differs significantly in the anesthetized patient from normal ventilation in the awake patient:

- **Respiratory depression** caused by tranquilizers and general anesthetics, despite PaCO₂ level becoming elevated. Normal RR in anesthetized patient = 8-20 Bpm. Normal RR in awake patient = 20-30 Bpm.

- **Reduced TV** is caused by many factors that restrict or relax intercostal muscles and diaphragm, causing them to expand less than normal during inhalation. In the anesthetized patient, breathing spontaneously, TV is often reduced to 8ml/kg or less as compared to the awake patient at 10-20 ml/kg.

Patient factors that contribute to a decrease in RR and TV:

- Anesthetic agents (inhalants most profound)
- Anesthetic episode that lasts more than 90 minutes (prolonged)
- Pain
- Patient position during surgery (decreased FRC and atelectasis in dorsal or lateral recumbency)
- Obese patients
- Patients with large abdominal masses or excessive abdominal distention (GDV, dystocia, ascites)
- Patients that have received a neuromuscular blocking agent (NMBAs - paralytics)
- Patients with pre-existing pulmonary disease (pneumonia, effusion)
- Patients with recent head trauma
- Patients undergoing surgical procedures involving the thorax or diaphragm

Because the patient’s TV and RR may be decreased due to the above mentioned factors, respiratory minute volume is also decreased. The anesthetist must be aware of the following potential complications:

**Hypoventilation** – is the insufficient elimination of carbon dioxide (CO2) from the body relative to CO2 production. This is the most common complication of anesthetized animals because most patients are allowed to spontaneously ventilate on their own during the anesthetic procedure. Causes of hypoventilation may include: excessive mechanical dead space volume, anesthetic overdose, pain, pleural effusion, pulmonary edema, pneumonia, endobronchial intubation, abdominal distention, diaphragmatic hernia/rupture, severe hypotension, hypothermia, cervical disease affecting the phrenic nerve, muscle paralysis due to neuromuscular blocking agents (NMBAs) or disease.

Management options for hypoventilation include: decrease mechanical dead space, proper endotracheal intubation, reduce anesthetic depth, utilize patient warming devices, use multimodal anesthesia, thoracocentesis, implement IPPV (intermittent positive pressure ventilation) if indicated.

**Hypercapnia** (or hypercarbia) – is an increase in PaCO2 (>60mmHg) in the circulating blood due to rebreathing CO2 or hypoventilation. Rebreathing of CO2 does not directly cause hypoventilation, but it contributes to a build-up of CO2 and can result in hypercapnia. Factors that contribute to rebreathing of CO2 include exhausted CO2 absorbent, malfunctioning scavenging system and inadequate oxygen flow rates for non-rebreathing circuits. It is important to keep these factors in mind because an elevated CO2 might not be due to hypoventilation, but rather the rebreathing of CO2.

**Mild hypercapnia** (PaCO2 45-60mmHg) may actually be beneficial to some degree because it acts as a mild sympathetic nervous system stimulant which causes a release of catecholamines. These circulating catecholamines will cause an increase in blood pressure and cardiac output. This is known as permissive hypercapnia. The effects of mild hypercapnia include myocardial depression if the patient's catecholamines are depleted due to stress or trauma and possible arrhythmias due to circulating catecholamines. Permissive hypercapnia is contraindicated in patients with elevated intracranial pressure because even a slight increase in CO2 will cause dilation of the cerebral blood vessels which can lead to an increase in blood flow to the brain and further increase intracranial pressure.

**Moderate hypercapnia** (PaCO2 60-90 mmHg) can lead to respiratory acidosis, myocardial depression, ventricular arrhythmias and a further increase in intracranial pressure.

**Severe hypercapnia** (PaCO2 90-120 mmHG) will result in CNS depression, bradycardia and unconsciousness even without anesthesia.
Management options for hypercapnia include: decrease mechanical dead space, reduce anesthetic depth, implement IPPV (intermittent positive pressure ventilation).

**Hypoxemia** – Is a reduced oxygen concentration in the blood. Less O2 and inhaled anesthetic enters the lungs and less is available to be absorbed into the blood. If the anesthetized patient has been pre-medicated and is still breathing room air, PaO2 (oxygen in the arterial blood) may fall below normal values (<80-90mm Hg) as a result of decreased respiratory minute volume. This is equivalent to oxygen saturation (SaO2) of less than 95%. It may be beneficial to provide flow-by O2 in the peri-induction period to saturate the patient’s hemoglobin with O2 to prevent SaO2 dropping below 95% during the potentially apneic phase of induction and intubation. Patients that are intubated and breathing 100% O2 should maintain a PaO2 of 400-500 mmHg (SaO2 98-100%). Causes for hypoxemia include:

- **Ventilation/Perfusion Mismatch (V/Q mismatch)** – Ventilation and blood flow (perfusion) are mismatched at the level of the alveoli resulting in inefficient gas exchange between the lungs and pulmonary blood. This is the most common cause of reduced oxygenation in an anesthetized patient due to dorsal or lateral recumbencies and vasodilation or vasoconstriction caused by disease or anesthetic drugs. Maximum gas exchange occurs when the ratio between ventilation (V) and perfusion (Q) is equal to 1. A V/Q ratio of <1 means that perfusion is occurring but ventilation is not. In other words, blood does not become fully oxygenated as it passes through the lungs. Causes include atelectasis and bronchial intubation. A V/Q of >1 means that ventilation is present, but perfusion is not. This is also indicative of dead space ventilation. Causes may include pulmonary thromboembolism and severe hypovolemia or hypotension.

- **Other conditions that may cause hypoxemia:** diaphragmatic hernia/rupture, hypothermia, anemia, hypovolemia, pulmonary edema, pneumonia, pulmonary fibrosis, congenital heart abnormalities. Patients experiencing pulmonary or cardiac conditions are not good anesthetic candidates and should receive a very thorough examination, cardiology consult and/or treatment prior to undergoing anesthesia and should be managed and monitored closely by a veterinary specialist.

Management options for hypoxemia include: pre-oxygenate patient prior to induction, proper placement of endotracheal tube, administer adequate oxygen flow rates for the specific breathing system selected, “sigh” for patient every 5 minutes to increase alveoli surface area, minimize patient placement in dorsal or lateral recumbencies, implement IPPV, administer bronchodilators (albuterol).

**Atelectasis** – Because the patient’s TV is reduced, the alveoli do not expand fully, as normal on inspiration. The alveoli in some sections of the lung may partially collapse. This subsequently leads to a decreased uptake of inhaled anesthetic into the bloodstream. Atelectasis can be seen in patients in lateral or dorsal recumbency when alveoli can be compressed by the weight of the overlying lung tissue, mediastinal structures, and by the weight of the abdominal contents pressing against the diaphragm. Patients with large abdominal masses or excessive abdominal distention (GDV, dystocia, ascites) can be significantly prone to atelectasis.

Management options for atelectasis include: proper placement of endotracheal tube, “sigh” for patient every 5 minutes, minimize patient placement in dorsal or lateral recumbencies, implement IPPV,
increase peak airway pressure (up to 40 cmH2O) every 1-2 breaths during IPPV, provide positive end expiratory pressure (PEEP) to increase alveolar volumes and recruit collapsed alveoli. Use extreme caution when providing IPPV to a patient experiencing long-term atelectasis to avoid re-expansion pulmonary edema (RPE) which is a result of consistent high inspiratory pressures cause the collapsed alveoli to rupture and “weep”, causing fluid to leak out into the bronchioles leading to edema.

**Apnea** - is the absence of breath usually caused by anesthetic induction agents (Propofol), increased anesthetic depth, anesthetic agent overdose. Other causes of respiratory arrest include head trauma, hypoxia, severe pulmonary disease, cardiac arrest, iatrogenic ventilation of the patient, neuromuscular blocking agents, neuromuscular diseases and equipment failure (APL valve closed). There is no gas exchange occurring, therefore, patients will either wake up or respiratory arrest will occur.

Management options for apnea include: carefully titrate anesthetic induction agents, rapid intubation, assisted breathing, implement IPPV, avoid hypercapnia, set-up and monitor anesthetic equipment carefully, initiate CPCR if respiratory arrest occurs.

**Patient factors that may contribute to an increase in RR and/or TV:**

- Hyperthermia
- Pain
- Upper airway disease (elongated soft palate, stenotic nares, laryngeal paralysis, tumor)
- Inadequate anesthetic depth
- Hypoxia (obstructed airway, asthma, anemia)
- Pulmonary disease or trauma (edema, effusion, pneumothorax)

Because the patient’s TV and RR may be increased due to the above mentioned factors, the anesthetist must be aware of the following **potential complications**:

**Hyperventilation** – is an increase in minute volume due to an increase in TV and RR. Common causes are pain, inadequate anesthetic depth, overzealous manual ventilation, no or inadequate oxygen delivery flow rate, hypoxia, hypotension, or hyperthermia (panting), upper airway disease, upper airway obstruction.

Management options for hyperventilation include: increasing anesthetic depth, administering analgesic agents, provide adequate oxygen flow rate, reduce IPPV (RR and/or TV), treat hypoxia, hypotension and hyperthermia. Patients with suspected pulmonary trauma should be ventilated very cautiously and conservatively, or when possible, should breathe spontaneously to avoid additional trauma.

**Hypocapnia** – is a decreased amount of CO2 in the blood (PaCO2 of <35mmHg) caused by hyperventilation.

In many cases, it may be necessary to provide intermittent positive pressure ventilation (IPPV) during anesthesia. This refers to artificial inspirations applied to the airway. This can be done manually or mechanically. The most common mechanical ventilator used in veterinary medicine is known as a pressure limited/time cycled design during which breaths are given at fixed intervals and allows for a certain maximum airway pressure to be chosen so that when this pressure is reached, inspiration is terminated. The minute volume is chosen by adjustment of the inspiratory flow control and the respiratory rate control.
A driving gas enters the bellows chamber that, when pressurized, compresses the bellows and delivers a preset TV to the patient. The bellows are filled with O2 and anesthetic inhalant. To put it simply, a ventilator is nothing more than a mechanical reservoir bag. The Inspiratory to Expiratory Ratio (I:E) is used to determine the time of inspiration versus the time allowed for expiration by the patient. The standard acceptable setting is 1:2. An I:E ratio that is too short could potentially cause a decrease in venous return and subsequently blood pressure (decreased cardiac output). To prevent alveolar collapse and keep terminal airways partially inflated, it may be necessary to apply Positive End Expiratory Pressure (PEEP). This refers to a valve that is applied directly to the anesthesia machine to maintain a degree of pressure in the lungs at the end of exhalation. It can affect cardiac output by maintaining constant positive pressure in the thoracic cavity, which can affect venous return to the heart.

Indications for mechanical ventilation:
- Surgery requiring an open thorax
- If NMBAs are used (paralytic agents)
- Neuromuscular disease (myasthenia gravis, myopathies)
- Thoracic wall problems
- Abdominal distention
- Pulmonary parenchymal disease
- Increased intracranial pressure

Risks of mechanical ventilation:
- Excessive airway pressure may rupture alveoli, causing pneumothorax or mediastinal emphysema
- Decreased cardiac output
- Hypocapnia (decreased ETCO2) which could lead to respiratory alkalosis, which, if severe, can cause cerebral vasoconstriction and decreased cerebral blood flow.
- Hypercapnia (elevated ETCO2) which could lead to respiratory acidosis, which, if severe, can cause cerebral vasodilation and increased cerebral blood flow and intracranial pressure.
- Ventilation/Perfusion problems (V/Q mismatch) due to decreased pulmonary blood flow.
- Tension pneumothorax (if undetected) will become worse.

Prior to each use, it is very important to choose the appropriately sized bellows for your patient (calculated TV + 20%), properly pressure check the anesthesia machine and pressure test the ventilator. Always refer to the manufacturer’s guide for the proper setup and directions on the use of your particular model.

Peak Inspiratory Pressure (PIP) is the numeric pressure value of gas delivery to the patient at the peak of inspiration. PIP is monitored by watching the pressure manometer on the anesthesia machine during peak inspiration. To avoid trauma to lung tissues, PIP in healthy animals should not exceed 20 cmH2O. A PIP of 12-15 cmH2O in the closed thorax patient is sufficient for adequate respiratory function given that the RR is acceptable. Feline patients may require less pressure to adequately ventilate, usually 10-15 cmH2O. There are instances in which higher pressures are needed to deliver adequate tidal volumes such as obese patients, dorsal recumbency, abdominal distention and an open thorax.
Generally, a respiratory rate of 8-14 breaths per minute and a tidal volume of 10-20 ml/kg are considered within normal limits for a small animal patient on a ventilator. Keep in mind that slow, deep breaths are usually more beneficial for alveolar ventilation. IPPV is more efficient than spontaneous breathing, therefore, close monitoring of anesthetic depth is a must and lower respiratory rates and reduced inhalant concentrations are often warranted. Consider that larger, younger and deep-chested patients can have a normal TV closer to 20-25ml/kg. Conversely, smaller, older, flat-chested patients can have a normal TV closer to 10 ml/kg. Be prepared to make changes to these settings as they are only values to be set as a baseline. Monitoring the patient physical and mechanically will allow you to adjust ventilator settings as the patient’s status indicates.

Physically monitoring includes closely observing your patient’s thorax during inspiration. Does this patient’s breathing (chest rising and falling) seem similar to as if he were lying asleep on the family room rug but the fireplace? If you are manually ventilating (hand bagging) the patient, evaluate how it feels when you are administering a breath. Do you feel any resistance? This can be defined as anything that causes an increase in work to attain adequate ventilation. This can occur in either the upper or lower airways. Examples of resistance are pneumonia (due to congestion), endotracheal kink due to patient’s neck position, overinflated endotracheal tube cuff (crushing inner lumen of the tube), geriatric patient (difficult thoracic expansion), and accumulation of salivary secretions in the endotracheal tube (partial or complete obstruction). Can you appreciate the thoracic compliance or flexibility? A decrease in compliance may indicate disease processes and upper or lower airway obstructions (asthma) or atelectasis.

Mechanical monitoring should include capnography, at minimum to verify efficiency in gas exchange and adequate ventilation. Blood gas analysis can be a useful tool, but is not usually available in private practice due to cost. Capnography is a noninvasive method to assess systemic metabolism, cardiac output, pulmonary perfusion, and the adequacy of patient ventilation. Capnographs typically provide a numerical value for end-tidal CO2 (ETCO2 – the amount of CO2 in the exhaled breath) and a graphic representation of exhaled CO2 in respiratory gases over time. The capnograph often also includes a numerical value for RR and inspired CO2 (FiCO2). Capnography is superior over pulse oximetry for the prompt identification of apnea and airway mishap. It can also be an early and reliable indication of impending cardiovascular collapse or cardiac arrest. Since delivery of CO2 from the lungs requires blood flow, cellular metabolism, and alveolar ventilation, the presence of ETCO2 can also be used to assess the effectiveness of CPCR.

A normal capnograph is represented by 4 phases: 3 expiratory and 1 inspiratory. The illustrated result of a normal capnograph should appear as a “top hat” with the inspiratory plateau landing at 0 mmHg and the expiratory plateau falling between 35-45 mmHg (ETCO2). (Fig. 1)
Fig. 1 Normal Capnograph (Reuss-Lamky, Anesthesia for Veterinary Technicians 2010; Drawing by Mele Tong)

Fig. 2 Hypoventilation (Increased ETCO2) (Reuss-Lamky, Anesthesia for Veterinary Technicians 2010; Drawing by Mele Tong)
Fig. 3 Hyperventilation (Decreased ETCO2) (Reuss-Lamky, Anesthesia for Veterinary Technicians 2010; Drawing by Mele Tong)

Fig. 4 Spontaneous breaths taking during ventilated breaths. (Reuss-Lamky, Anesthesia for Veterinary Technicians 2010; Drawing by Mele Tong)
Fig. 5 Rebreathing CO2, Inspired CO2 doesn’t reach 0 mmHG. (Reuss-Lamky, Anesthesia for Veterinary Technicians 2010; Drawing by Mele Tong)

Fig. 6 Artifact due to strong heartbeat. (Reuss-Lamky, Anesthesia for Veterinary Technicians 2010; Drawing by Mele Tong)
Fig. 7 Airway Obstruction. (Reuss-Lamky, Anesthesia for Veterinary Technicians 2010; Drawing by Mele Tong)
Troubleshooting mechanical ventilation:

1) Bellows will not stay inflated
   a. Is the O2 on?
   b. Is the APL valve closed?
   c. Is there a leak in the endotracheal tube cuff?
   d. Are the bellows attached to the ventilator correctly?
   e. Is the bellows canister secured to the ventilator?
2) When I increase the patient’s respiratory rate, why does the TV decrease? 
Remember that a Time Cycled ventilator takes the minute volume you’ve calculated for your patient and divides it into the number of breaths chosen. When you adjust either the TV or RR, the opposite will compensate to match your original minute volume. Therefore, you will need to adjust both to change your patient’s ventilation status. For example, if you increase your RR, you will need to also increase your TV to its original setting and vice versa.

3) Why do I hear a single beep alarm at the end of each inhalation?
   a. Double check your ventilator’s maximum pressure alarm setting and make sure it is set at 20cm.
   b. Evaluate your patient’s inspiratory pressure on the manometer and make sure that it is within normal limits (<20cmH2O)

4) The big, loud, scary alarm is going off and won’t stop. HELP!
   a. Are the ventilator bellows delivering a breath to your patient? Is it a reasonable TV?
   b. Is the APL valve closed to keep gases in the ventilator? Are the bellows staying inflated?
   c. Is the patient breathing spontaneously around the ventilator settings?
   d. Is your patient’s thorax rising and falling with each ventilation? Could your ET tube be kinked? Or could there be an obstruction?

5) What if my patient’s ETCO2 level is high (hypercapnia)? What adjustments do I make to my ventilator settings?
   a. Increase RR
   b. Increase TV
   c. Increase O2 flow rate?? (20-40 ml/kg/min rebreathing system) (200-400 ml/kg/min non-rebreathing system)

You can do the opposite for low ETCO2 (hypocapnia)

6) My patient is “bucking” the ventilator, what do I do?
   a. When a patient begins “bucking” the ventilator or breathing spontaneously, you may see spontaneous breaths on the capnograph or the bellows “bobbing” up and down at intervals other than your set respiratory rate. An alarm may also sound due to changes in peak inspiratory pressure.
   b. Check patient’s anesthetic depth and increase inhalant/injectable anesthetic and/or provide additional analgesia agents as indicated.
   c. Increase patient RR and/or TV if necessary or indicated by capnography.

Mechanical ventilation is not intended to relieve the anesthetist of the necessity for patient monitoring. The anesthetist must closely monitor all patients in which ventilation is controlled or assisted to ensure that patient anesthetic depth and vital signs are maintained within acceptable limits. Mechanical ventilation can be an extremely useful tool in ensuring that an anesthetized patient receives adequate oxygen, inhaled anesthetics and is able to exhale adequate amounts of CO2.