Anesthesia
for Veterinary Technicians

1 Review of Cardiovascular and Respiratory Physiology

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Most anesthetic drugs affect the cardiovascular and/or pulmonary systems in some way. It is important for the anesthetist to have at least a basic understanding of how these systems function and what impact anesthetic drugs are likely to have on them. This chapter reviews the basic physiology of the cardiovascular and respiratory systems, and some of the terminology related to them, as they relate to anesthesia.

The Cardiovascular System

Anatomy and physiology of the heart

The cardiovascular system consists of the heart, which is a muscular pumping device, and a closed system of vessels: the arteries, veins, and capillaries. The heart is responsible for pumping blood around the body, carrying nutrients to all parts of the body, and carrying waste away for removal. The heart consists of four chambers: the right atrium, the right ventricle, the left atrium, and the left ventricle. Arteries are the vessels that carry blood from the heart, and veins carry blood to the heart. Sodium, chloride, potassium, and calcium are the electrolytes that are most important for normal cardiac function. Depolarization of the cell occurs when sodium channels in the cell membrane open increasing sodium permeability. Resting membrane potential becomes less negative due to an influx of positive sodium ions. Cells begin repolarizing when the sodium gates close and negatively charged chloride ions begin to move into the cell. This causes calcium channels to open, allowing an influx of these ions. Final repolarization occurs when the calcium channels close and potassium permeability increases. Any alterations of normal plasma concentrations of these electrolytes can affect cardiac muscle function.

The *sino-atrial (SA) node* in the wall of the right atrium initiates the heartbeat. Impulses from this node transmit to the *atrioventricular (AV) node*. Other impulses in the heart are transmitted by the bundle of HIS, the bundle branches, and the purkinje fibers. Any damage to the cardiac muscle can result in unsynchronized impulse transmission, irregular heart contractions, and reduced cardiac output. The SA node acts as an intrinsic pacemaker and controls the rate of contractions. Both parasympathetic and sympathetic nervous systems innervate the SA node. Acetylcholine and noradrenaline are nervous system mediators that affect sodium,

calcium, and potassium channels and can increase or decrease depolarization. Many drugs used for anesthesia purposes can affect heart rate, and therefore monitoring is strongly indicated.

Cardiac cycle

The *cardiac cycle* is the complete series of events that happens in the heart during one heartbeat. Blood flows into the atria from the vena cava and pulmonary veins. The cycle starts with depolarization at the SA node leading to atrial contraction. The atrioventricular valves, called the *mitral* and *tricuspid* valves, open when atrial pressure exceeds ventricular pressure. While the atria contract, blood flows into the relaxed ventricles. This is *diastole*, when the ventricles are relaxed and filling. Next, the atria relax and the ventricles contract (*systole*) pushing blood out the aortic and pulmonary valves. Ventricular systole causes closure of the atrioventricular valves and this action is the first heart sound heard on auscultation. The second heart sound is generated when ventricular relaxation occurs and the pulmonic and aortic valves close. *Murmurs* are abnormal cardiac sounds and usually result from malfunction of the valves (Reece 1997).

Electrical activity

As the heart undergoes depolarization and repolarization, the electrical currents that are generated (as described above) spread not only within the heart, but also throughout the body. This electrical activity generated by the heart can be measured by electrodes placed on the body surface. The recorded tracing of this activity is called an *electrocardiogram* (ECG or EKG). The different waves that comprise the ECG represent the sequence of depolarization and repolarization of the atria and ventricles. The complete cardiac cycle that is portrayed on the ECG is represented by waves that are identified as P wave, QRS complex, T wave.

The *P wave* represents the wave of depolarization that spreads from the SA node throughout the atria. The brief isoelectric period after the P wave represents the time in which the impulse is traveling within the AV node and the bundle of HIS. The period of time from the onset of the P wave to the beginning of the QRS complex is termed the *P-R interval*. This represents the time between the onset of atrial depolarization and the onset of ventricular depolarization. If the interval is prolonged or no QRS complex follows (the impulse is unable to be conducted to the ventricles), there is an *AV conduction block* (1st, 2nd, or 3rd degree AV block).

The *QRS complex* represents the ventricular depolarization. The duration of the QRS complex is normally of relatively short duration, which indicates that ventricular depolarization occurs very rapidly. If the QRS complex is prolonged, conduction is impaired within the ventricles. This can occur with bundle branch blocks or whenever an abnormal pacemaker site becomes the pacemaker driving the ventricle. Changes in the height and width of the QRS complex can indicate left heart enlargement. The shape of the QRS complex can also change depending on placement of the electrodes. The isoelectric period following the QRS (ST segment) is the time at which the entire ventricle is depolarized. The ST segment is important in the diagnosis of ventricular ischemia or hypoxia because under those conditions, the ST segment can become either depressed or elevated.

The *T wave* represents ventricular repolarization and is longer in duration than depolarization. The *Q-T interval* represents the time for both ventricular depolarization and repolarization to occur and therefore roughly estimates the duration of an average ventricular action potential. At high heart rates, ventricular action potentials shorten in duration, which increases the Q-T interval. Prolonged Q-T intervals can be diagnostic for susceptibility to certain types of tachyarrhythmias.

Determining heart rate from an ECG strip

Heart rate can be determined by examining an ECG rhythm strip. The ventricular rate can be

determined by measuring the time intervals between the QRS complexes, which is done by looking at the R-R intervals. Assuming a recording speed of 25 mm/sec and a lead II ECG, one method is to divide 1500 by the number of small squares on the recording paper between two R waves. Or one can divide 300 by the number of large squares between waves (Blaze and Glowaski 2004). If the heart rate is irregular, it is important to determine a time-averaged rate over a longer interval. Changes in heart rate can affect the function of the heart. Very fast heart rates can reduce cardiac output by not allowing the ventricles to fill adequately (Clark 2003). Bradycardia can also affect cardiac output. Troubleshooting heart rate abnormalities should include identifying and correcting the underlying cause if possible. Treatment with fluid therapy and/or additional analgesics may be necessary for tachycardic patients. Lightening anesthetic depth and/or treatment with an anticholinergic may be necessary for bradycardic patients. Arrhythmias should be identified and their effect on cardiovascular function should be determined before treatment therapy is decided on.

Contractility is the intrinsic ability of cardiac muscle to develop force for a given muscle length. It is also referred to as *inotropism*. *Preload* is the force acting on a muscle just before contraction, and it is dependent on ventricular filling (or end diastolic volume). Preload is related to right atrial pressure. The most important determining factor for preload is venous return. Hypovolemia, vasodilation, and venous occlusion decrease preload.

Afterload is the tension (or the arterial pressure) against which the ventricle must contract. If arterial pressure increases, afterload also increases. Afterload for the left ventricle is determined by aortic pressure; afterload for the right ventricle is determined by pulmonary artery pressure.

Blood pressure is the driving force for blood flow (perfusion) through capillaries that supply oxygen to organs and tissue beds of the body. Blood pressure is needed to propel blood through high-resistance vascular beds, including those of the brain, heart, lungs, and kidneys. Blood pressure variations are detected by baroreceptors that are present throughout the cardiovascular system. These baroreceptors are capable of stimulating the autonomic nervous system in response to increases and decreases in blood pressure. If blood pressure falls, the sympathetic nervous system is stimulated and outflow will be increased, causing an increase in heart rate and blood pressure. If blood pressure increases, the parasympathetic system works to slow the heart rate and decrease pressure (Fraser 2003).

Blood pressure values are expressed in millimeters of mercury (mm Hg) and as three measurements: *systolic*, *mean*, and *diastolic*. Remember that the systolic pressure is the pressure generated when the left ventricle is fully contracted. Diastolic pressure is the pressure measured when the left ventricle relaxes. Pulse pressure felt on peripheral arteries is the difference between the two numbers. Mean arterial pressure (MAP) is calculated as diastolic pressure + 1/3 systolic pressure (systolic pressure – diastolic pressure) (Smith 2002). Mean blood pressure determines the average rate at which blood flows through the systemic vessels. It is closer to diastolic than to systolic because, during each pressure cycle, the pressure usually remains at systolic levels for a shorter time than at diastolic levels. Most times, under anesthesia, a patient's mean pressure is what the anesthetist focuses on. A mean arterial pressure of at least 60 mm Hg (70 in horses) is needed to properly perfuse the heart, brain, and kidneys. Mean arterial blood pressures consistently below 60 mm Hg can lead to renal failure, decreased hepatic metabolism of drugs, worsening of hypoxemia, delayed recovery from anesthesia, neuromuscular complications, and central nervous system abnormalities, including blindness after anesthesia (Smith 2002). Prolonged hypotension (> than 15–30 minutes) can lead to nephron damage. Although the effects may not be immediately apparent because 65–75% of nephrons need to be damaged before renal disease becomes clinically observable, the effects may play a role in the onset of renal disease later in a pet's life. Severe untreated hypotension can lead to cardiac and respiratory arrest. Hypertension, or excessively high blood pressure, can lead to problems as well. Ideally, any animal under anesthesia

should have should have regular blood pressure monitoring because most anesthetic drugs affect blood pressure in some way.

Mean arterial blood pressure (MAP) = cardiac output (CO) × systemic vascular resistance (SVR). *Cardiac output* is defined as the amount of blood pumped by the heart in a unit period of time. Cardiac output is a term that is often used in anesthesia because it is extremely important in the overall function of the cardiovascular system. In general, the term applies to how well the cardiovascular pump (heart) is working. Many factors affect CO, directly or indirectly, including some anesthesia drugs. CO = heart rate (HR) × stroke volume (SV). *Contractility* is the amount of force and velocity that the ventricles can exert to eject the volume within them (Hamlin 2000). *Systemic vascular resistance* is the amount of resistance to flow through the vessels. Some vessels may be dilated and therefore allow more flow at less resistance. Constriction of vessels may limit blood flow and require more pressure to get blood through. It's important to know that many of the drugs used for anesthesia affect one or more of these systems in some way.

Normal systolic blood pressures in the conscious awake patient are 100–160 mm Hg, normal diastolic pressures are 60–100 mm Hg, and normal mean arterial blood pressure ranges are 80–120 mm Hg. Hypotension is classified as MAP of less than 60 mm Hg. It is important to be able to identify the cause of a blood pressure abnormality to know how to begin treatment for it. There are generally three things to consider when looking for causes of hypotension. Look for drugs or physiological/pathological factors that may reduce systemic vascular resistance (SVR), look at heart rate, and look for things that affect stroke volume (preload/contractility) (Smith 2002). As mentioned earlier, many of the drugs used in anesthesia cause some degree of hypotension, and less often, hypertension. Knowing the side effects of these drugs and how they work will help in determining treatment. Drugs that decrease SVR (and cause vasodilation) in a dose-dependent manner include acepromazine, thiobarbiturates, propofol, and the inhalants. Other physiologic factors that may cause a decrease in blood volume or vascular tone include hemorrhage, inadequate volume administration or replacement, dehydration, shock, sepsis, anaphylaxis, or severe hypercapnia (high CO_2) (Smith 2002). Patients with acid/base abnormalities should be stabilized prior to anesthesia if possible to help reduce the possibility of hypotension. Drugs that can decrease heart rate include opioids, alpha 2 agonists, and the inhalant drugs isoflurane and sevoflurane. Patients with intracranial disease, hypothermic patients, and extremely fit pets may have low heart rates (bradycardia). Anesthetic drugs affecting the contractility of the heart include the inhalants, thiobarbiturates, propofol, and alpha-2 agonists. The inhalant drugs are potent vasodilators, with up to a 50% reduction in cardiac contractility at surgical planes of anesthesia as well. The other drugs' effects on contractility are more transient and less profound. Alpha-2 agonists and phenylephrine cause vasoconstriction of blood vessels, which results in hypertension. The effects of hypertension from the alpha-2 agonists are transient, lasting only a few minutes before the vessels relax and hypotension can result. The dissociative drugs, ketamine and Telazol, have indirect positive effects on the cardiovascular system and thus increase heart rate, but this can cause a reduction in stroke volume depending on how severely heart rate is affected. Patient positioning can affect blood pressure. Obese or bloated patients or patients with large abdominal masses placed in dorsal recumbency may be hypotensive due to excessive pressure on the caudal vena cava. This pressure may compromise venous return and result in hypotension. The same can happen when positive pressure ventilation is used.

Certain disease states can cause hypertension, including renal disease, pheochromocytomas, pulmonic stenosis, heartworm disease, and hyperthyroidism. Ideally, these patients will have their hypertension well controlled before surgery. The exception may be the pheochromocytoma patient whose hypertension may spike up during surgery when the tumor is manipulated. A nitroprusside CRI may be indicated for these patients if systolic pressure exceeds 200 mm Hg. If a patient develops hypertension under anesthesia that is not related to a disease state, the cause is most likely related to inadequate anesthetic

depth and/or inadequate analgesic administration. Adjusting anesthetic depth and providing additional pain medications should result in normotension.

Changes in blood volume affect arterial blood pressure by changing cardiac output. During general anesthesia, the rate of normal ongoing losses of fluid from the body is increased by high oxygen flow rates. The oxygen dries the respiratory system and causes rapid evaporation from mucous membranes. To offset this loss, intravenous fluid administration of crystalloids at a rate of 10 mL/kg/hr is recommended to help "fill the space" caused by vasodilation and to replace normal ongoing losses that occur in patients (with normal cardiovascular and renal function; patients with certain cardiac diseases may not be able to "handle" excessive fluid overload) under anesthesia. Fluid therapy is best begun before hypotension exists. For suspected hypovolemia a fluid bolus of "1 hour's worth" of the patient's maintenance rate may be given (i.e., 35 kg pet = 350 mL bolus, along with maintenance fluids). Reassess following the bolus. If the patient is instrumented with a Doppler monitor you may be able to hear the improvement and "stronger" flow. Blood loss should be replaced with 2–3 times the suspected amount of loss. One mL of blood loss should be replaced with 2–3 mL of crystalloid. Excessive hemorrhage will require replacement with colloids and blood products. The best way to prevent hypotension is to detect changes in blood pressure as soon as they begin and start treatment to restore it as soon as possible.

Volume status

Central venous pressure is the blood pressure within the intrathoracic portion of the caudal or cranial vena cava. It can be measured to gain information about intravascular blood volume and cardiac function. CVP correlates with the volume reaching the right atrium during diastole. A single measurement of CVP yields little information, but serial measurements are useful. Measuring CVP can be helpful in determining hypovolemia or fluid overload. Normal CVP should be 2–7 cm H_2O in anesthetized patients (Muir et al. 2000).

Urine output can also be measured to determine volume status. Normal urine output should be 1–2 mL/kg/hr.

Respiratory Physiology

Every cell in the body needs a constant supply of oxygen to produce energy to grow, repair, or replace itself and to maintain normal vital functions. The respiratory system is the body's link to its supply of oxygen. It includes the diaphragm and chest muscles, the nose and mouth, the pharynx and trachea, the bronchial tree and the lungs. The bloodstream, heart, and brain are also involved. The bloodstream takes oxygen from the lungs to the rest of the body and returns carbon dioxide to them to be removed. The heart creates the force to move the blood at the right speed and pressure throughout the body. The smooth functioning of the entire system is directed by the brain and the autonomic nervous system.

Anatomy of the respiratory system

The main function of the lungs is to provide continuous gas exchange between the inspired air and the blood in the pulmonary circulation. The lungs supply oxygen during inspiration and remove carbon dioxide (CO_2) during expiration. During inspiration air containing oxygen (at sea level, atmospheric air contains 21% oxygen) enters the body through the nose and mouth. From there it passes through the pharynx on its way to the trachea. The trachea divides into two main *bronchi* upon reaching the lungs. One bronchus serves the right lung and the other serves the left lung. The bronchi subdivide several times into smaller bronchi, which then divide into smaller and smaller branches called *bronchioles*. After many subdivisions, the bronchioles end at the alveolar ducts. At the end of each alveolar duct are clusters of *alveoli*. The oxygen transferred through the system is finally

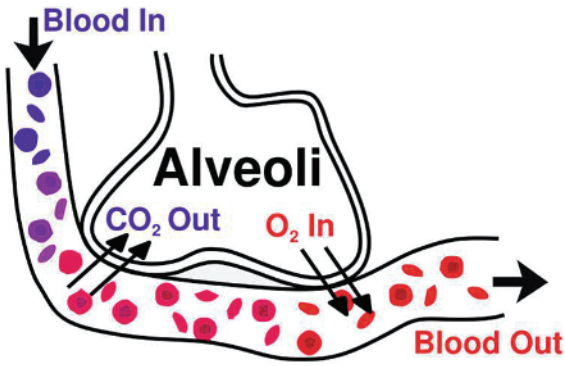

Figure 1.1. Illustration of perfusion. (Drawing by Melanie Tong.)

transferred to the bloodstream at the alveoli. Blood vessels from the pulmonary arterial system accompany the bronchi and bronchioles. These blood vessels also branch into smaller and smaller units ending with *capillaries*, which are in direct contact with each alveolus. Gas exchange occurs through this *alveolar-capillary membrane* as oxygen moves into and carbon dioxide moves out of the bloodstream (perfusion) (Fig. 1.1).

In the blood, oxygen is transported in two forms: dissolved in plasma (which is measured by PO_2) and bound to hemoglobin (measured by SpO_2). The amount of oxygen bound to hemoglobin is much larger than the amount dissolved in plasma (Fig. 1.2).

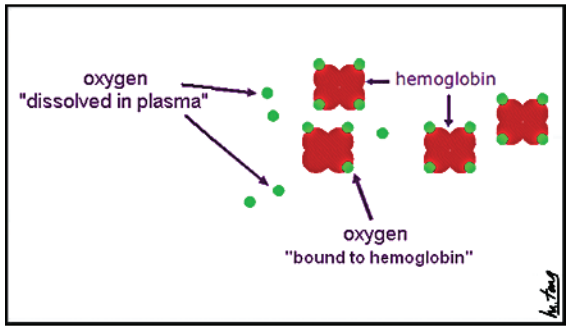

Figure 1.2. The amount of oxygen bound to hemoglobin is much larger than the amount dissolved in plasma. (Drawing by Melanie Tong.)

The "matching" of ventilation and perfusion is important to proper lung function. It does no good to ventilate an alveolus that is not being perfused (*alveolar dead space*) or to perfuse an alveolus that is not being ventilated because of *atelectasis* (collapse of alveoli). In the normal lung, ventilation (V) and perfusion (Q) are not evenly matched (known as *V/Q mismatch*), and this worsens with lung disease and dorsal or lateral recumbencies. Both ventilation and perfusion increase toward the dependent regions of the lung, but since blood is heavier than lung parenchyma, perfusion increases at a faster rate than ventilation. Vasodilation or vasoconstriction, caused by disease or anesthetic drugs, enhances V/Q mismatching and hypoxemia. From an anesthetic point of view, *alveolar ventilation* is very important because it will control the amount of volatile or gaseous anesthetic agent that can diffuse into the bloodstream. Any increase in alveolar ventilation will increase anesthetic uptake into the pulmonary blood (Fraser 2003).

The movement of air into and out of the lungs is called *ventilation*. The contraction of the inspiratory muscles, mainly the diaphragm, causes the chest cavity to expand, creating negative pressure. This is inspiration. During maximal inspiration, the diaphragm forces the abdominal contents ventrally and caudally. The external intercostal muscles are also involved. These muscles contract and raise the ribs during inspiration, increasing the diameter and volume of the chest cavity. Ventilation is the process by which gas in closed spaces is renewed or exchanged. As it applies to the lungs, it is a process of exchanging the gas in the airways and alveoli with gas from the environment. Breathing provides for ventilation and oxygenation.

Normal expiration is a passive process resulting from the natural recoil or elasticity of the expanded lung and chest wall. However, when breathing is rapid, the internal intercostal muscles and the abdominal muscles contract to help force air out of the lungs more fully and quickly. At the end of inspiration, the elasticity of the lung causes it to return to its smaller, unexpanded size. The ability to do this is called *elastic recoil*. The volume of air remaining in the lung at the end of a normal breath (the end expiratory lung

volume) is called the *functional residual capacity* (FRC). FRC is composed of the expiratory reserve volume and the residual volume. On expiration, there is still air left in the lungs; if there weren't, all of the alveoli would collapse. FRC decreases slightly in supine and lateral recumbency compared to prone. Certain conditions such as pregnancy, obesity, and abdominal distention due to gas-filled organs or masses can exacerbate decreases in FRC. FRC is diminished with small airway diseases. At FRC the alveoli in the nondependent lung sections are larger than those in the dependent regions because of gravity and the weight of the lung. Alveoli in the dependent regions are squashed and compressed by the weight of the overlying lung tissue. In lateral or dorsal positions the dependent alveoli are also compressed by the weight of the mediastinal structures and by the weight of the abdominal contents pressing against the diaphragm. In patients with large abdominal masses or excessive bloating (such as gastric dilatation volvulus GDV or obstructive colic in horses), the problem becomes especially significant.

In spite of many protective mechanisms in place in the lungs, including FRC, small airway and alveolar collapse (atelectasis) still occurs in the normal animal. Atelectasis is especially prominent in the dependent lung regions when an animal is recumbent. Intermittent deep positive pressure breaths in the anesthetized patient can help minimize small airway and alveolar collapse. The use of *positive end expiratory pressure* (PEEP) is known to help as well. PEEP increases airway pressure and FRC to help keep small airways and alveoli open during expiration (Battaglia 2001). PEEP valves are commercially available (in 5, 10, and 15 cm H_2O) and can be added to the anesthesia machine for this purpose. Unfortunately, once atelectasis has occurred, it is very difficult to open the closed alveoli. Applying excessive pressure in an attempt to open alveoli (as in the case of attempting to reinflate a packed-off lung during a thoracotomy) tends only to damage the already working tissue (barotrauma) before reinflation takes place. Reinflation is a slow, delicate process and will happen on its own in healthy tissue over time or once recumbency or insult changes.

The degree of stiffness or *compliance* of the lung tissue affects the amount of pressure needed to increase or decrease the volume of the lung. With increasing stiffness, the lung becomes less able to return to its normal size during expiration. Virtually all diseases cause the compliance of the lungs to decrease to some extent.

The amount of airflow resistance can also affect lung volumes. *Resistance* is the degree of ease in which air can pass through the airways. It is determined by the number, length, and diameter of the airways. An animal with a high degree of resistance may not be able to exhale fully, thus some air becomes trapped in the lungs.

Tidal volume is the volume of gas passing into and out of the lungs in one normal respiratory cycle. Normal tidal volume for mammals is 10–20 mL/kg. *Minute volume* is used to describe the amount of gas moved per minute and is approximately 150–250 mL/kg/minute. *Minute volume = tidal volume × respiratory rate*. It is *alveolar ventilation* that is important for gas exchange, however. Alveolar ventilation is the portion of ventilation that contributes to gas exchange (McDonnell and Kerr 2007). Tidal volume is used to ventilate not only the alveoli, but also the airways leading to the alveoli. Because there is little or no diffusion of oxygen and carbon dioxide through the membranes of the airways, they comprise what is known as *dead space ventilation* (Fig. 1.3).

The other part of dead space is made up of alveoli with diminished capillary perfusion. Ventilating these alveoli is ineffective and will do nothing to improve blood gases. The nonperfused alveoli and the airways are known as *anatomic dead spaces*. Therefore tidal volume has a dead space component and an alveolar component. Dead space ventilation is about 30–40% of tidal volume and minute volume in a normal patient breathing a normal tidal volume. Dead space ventilation has a purpose. It assists in humidifying and tempering inhaled air, and it cools the body, as in panting. Panting is predominantly dead space ventilation. During panting, the respiratory frequency increases and the tidal volume decreases so that alveolar ventilation remains approximately constant. This is the reason that when animals under anesthesia

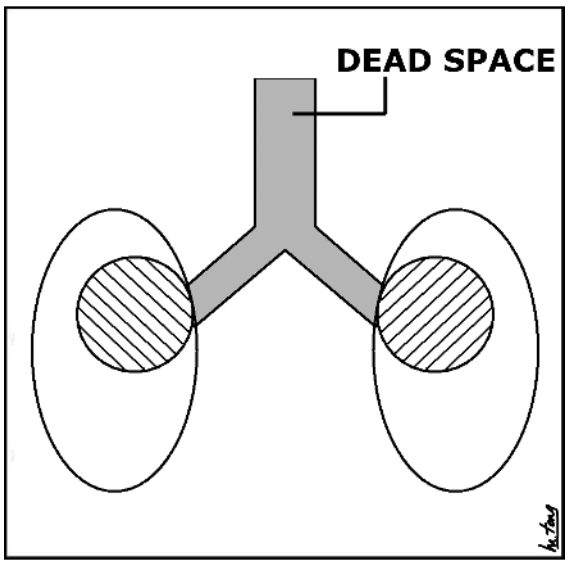

Figure 1.3. Anatomical dead space where no gas exchange takes place. (Drawing by Melanie Tong.)

pant, they very often wake up. They are not effectively ventilating their alveoli and exchanging gas well. Often times these patients will be hypercarbic because they are not able to effectively reduce their carbon dioxide levels. Slower, deeper breaths are usually more efficient. Certain pieces of anesthesia equipment can add to the anatomical dead space of a patient by "extending" its airway. This is called *mechanical dead space*. Endotracheal tubes that are too long and extend far beyond the patient's nose would be an example. Adding this dead space presents a further challenge to patients trying to effectively ventilate.

Monitoring ventilation on patients under anesthesia can be done a number of ways. Ventilation is assessed in terms of rate, rhythm, and tidal volume. First of all, a good look at the patient's chest excursions should be done to evaluate for quality and effort. Auscultation of the lungs should be performed prior to sedating or anesthetizing any patient. Normal lung sounds should be heard on both sides of the chest. Any abnormal sounds should be investigated prior to moving forward with anesthesia because anesthetic drugs can depress respiration and ventilation and may worsen existing problems.

Mucous membrane color should be assessed regularly. The tongue and gums should be pink. Any change in color, especially blue or purple tingeing, can indicate hypoxemia.

Spirometers or ventilometers can be used to measure tidal volume and minute volume. Apnea or respiratory monitors detect the movement of gas through the proximal end of the endotracheal tube. They sound an alarm when no gas movement is detected. They provide no information on tidal volume or the physiologic state of the patient. They can be falsely activated by pressure on the chest or abdomen of the patient or by cardiac oscillations that cause gas movement in the trachea.

Positive pressure ventilation (PPV) is indicated when an animal cannot ventilate adequately on its own. This indication may be defined as one or more of the following: *hypercarbia* (increased $CO_2 > 60$ mm Hg), *desaturation* ($SpO_2 < 95\%$) in spite of oxygen therapy, *hypoxemia* (PaO_2 of less than 100 mm Hg on oxygen), or a low observed or measured minute volume. PPV is always indicated in any surgery requiring an open chest, whenever paralytic neuromuscular blocking drugs are to be used, neuromuscular diseases, chest wall problems, abdominal enlargements, or pulmonary parenchymal disease. Any patient that is to be anesthetized with potentially increased intracranial pressure should be mechanically ventilated. Positive pressure ventilation is of great benefit to many patients, but it is not without potential complications. These can be avoided with careful monitoring, attention to detail, and a good understanding of the underlying physiological processes. A major contraindication for positive pressure ventilation is a closed pneumothorax, because positive pressure ventilation will make it worse. Positive pressure ventilation can decrease arterial blood pressure and reduce cardiac output, especially if airway pressures are consistently more than 10 mm Hg or if circulating blood volume is low. Artificial ventilation decreases pulmonary blood flow and therefore may lead to ventilation-perfusion abnormalities. These depressant effects can be seen on the arterial wave form during direct blood pressure measurement and on the waveform when measuring

oxygen saturation. It is seen as a dampening of the wave form following an artificial breath. The changes in circulatory flow during IPPV are caused by prolonged increases in mean airway pressures and decreases in CO_2 (Muir, et al., 2000). Hypovolemia worsens these effects.

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