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Chapter 1: Anatomy and Physiology of the Respiratory System

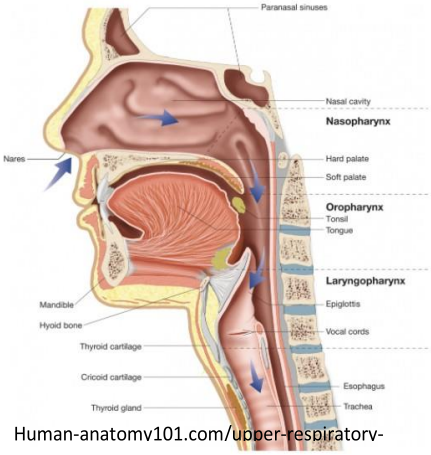
Anatomy of the airway and respiratory system

The anatomy of the respiratory system consists of all structures that air must travel through down to the smallest part of the lungs. Its role is to filter, warm, and moisten air as it is conducted into the lungs as well as provide an efficient environment by which oxygen (O_2) and carbon dioxide (CO_2) can be exchanged into the blood stream. Respiration refers to the utilization of oxygen and production of carbon dioxide by the body as a whole, or by individual cells, to create energy and complete cellular/metabolic tasks. For the study airway management and pulmonary function as it pertains to the SuperNO_2VA™ device, the anatomy of the respiratory system can be divided into two main categories: the upper airway and the lower airway.

Upper Airway Anatomy

The upper airway is made up of all aspects of the respiratory tract that is above the vocal cords, aka supra-glottic ("above the glottis"). These structures include: oral cavity, nasal cavity, pharynx, and larynx. The structures of the upper airway do **not** participate in gas exchange.

- ***Oral cavity:*** mouth, tongue, soft palate
- ***Nasal cavity***: nostrils, nasal mucosa
- ***Pharynx:*** back of the throat; divided into 3 regions
    - Nasopharynx: connects nasal cavity to pharynx
    - Oropharynx: connects oral cavity to pharynx
    - Laryngopharynx/Hypopharynx: connects larynx to pharynx
- ***Larynx:*** an organ that divides the upper and lower airways and contains the vocal cords



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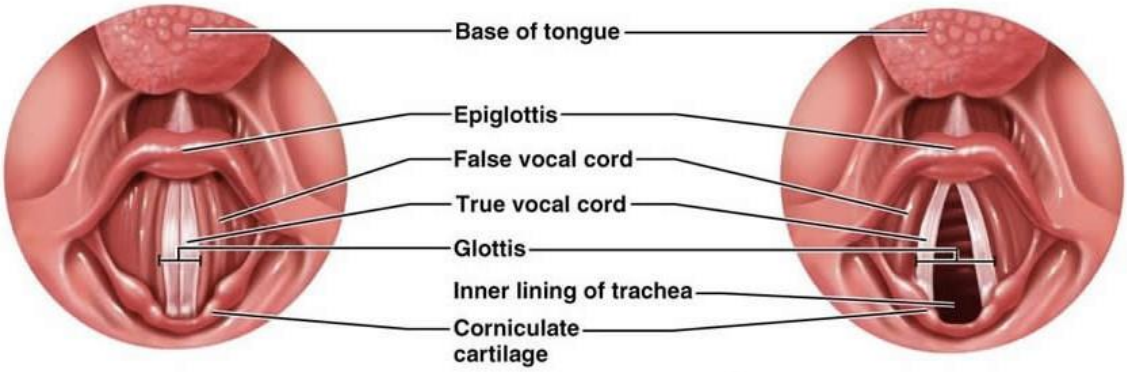
Separating the upper and lower airways in the larynx are the **vocal cords** (see below):

- The opening between the vocal cords is known as the "**glottis**."
- *Helpful Hint:* This is the view an anesthesiologist sees while performing laryngoscopy. An endotracheal tube is placed between the vocal cords.



http://classes.midlandstech.edu/carterp/Courses/bio211/chap22/chap22.htm

Lower Airway Anatomy

The lower airway consists of all structures that lie below the vocal cords (aka sub-glottic) and includes what is commonly referred to as "the lungs": the trachea, bronchi, bronchioles, alveoli and capillaries. The role of the lower airway is to warm and moisten air as well as conduct it to the alveoli, where gas exchange occurs.

- ***Trachea:*** aka windpipe; divides into bronchi
- ***Bronchi:*** a right and left bronchus divides into bronchioles
- ***Bronchioles:*** numerous branch on each side divides into alveoli
- ***Alveoli:*** consists of millions of thin, grape-like structures that exchange oxygen and carbon dioxide with surrounding capillaries
- ***Capillaries:*** the smallest blood vessels; allow oxygen to diffuse from alveoli into blood

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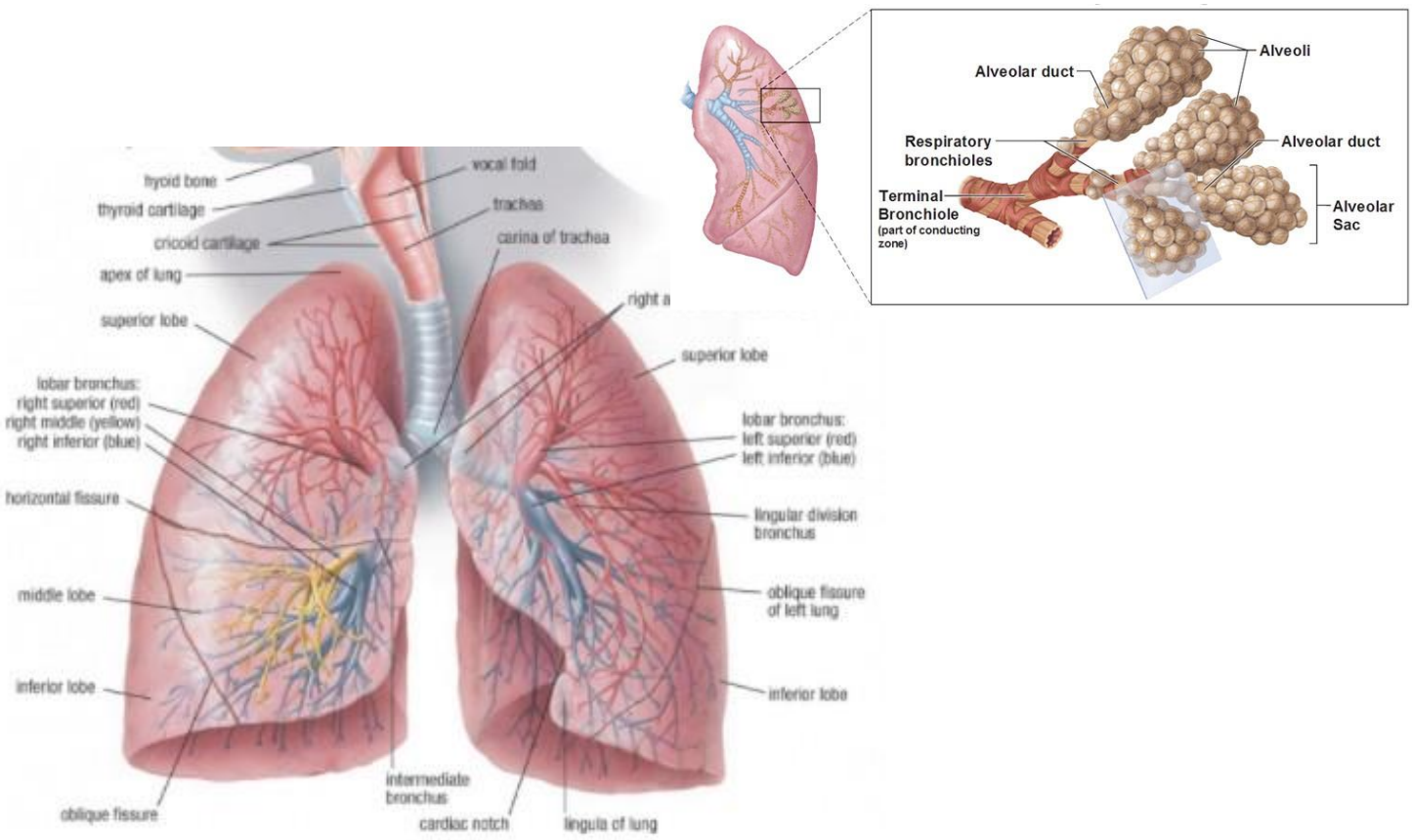

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Physiology of the respiratory system

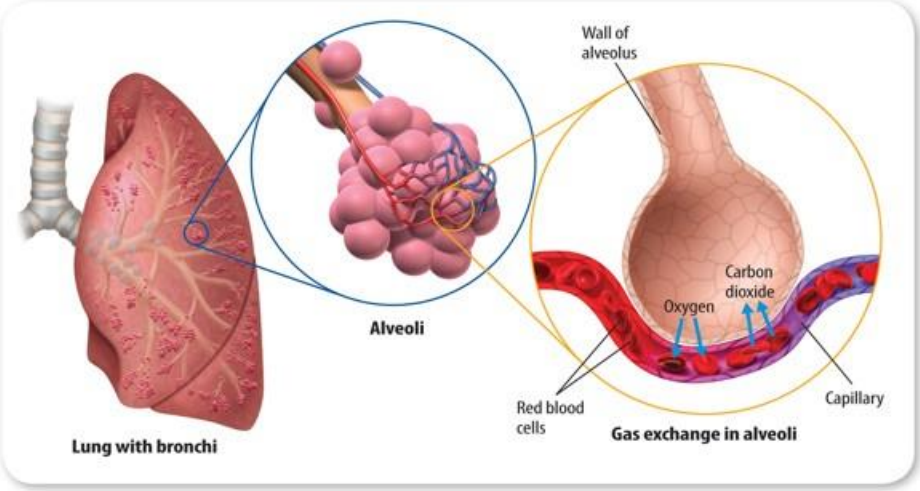
Gas Exchange

Gas exchange is a physiologic process by which oxygen and carbon dioxide are transferred in opposite directions within the lower airways across the alveolar-capillary membrane. It is important to understand that every cell in the body uses oxygen to create energy and perform metabolic tasks. During this cellular process, carbon dioxide is formed as a waste product that must be eliminated. The ultimate goal of breathing is to supply O_2 to the blood and body's tissues as well as remove CO_2 waste from the blood gathered from the tissues. The bronchioles and the alveolar ducts are responsible for 10% of the gas exchange. The alveoli are the major contributors and are responsible for 90% of gas exchange in the lungs.

The blood acts as a transport system for O_2 and CO_2 between the lungs and tissues, with the tissue cells extracting O_2 from the blood and releasing CO_2 as a waste by-product into the blood. The alveolar-capillary membrane is very thin and is permeable to O2 and CO2 (allows

them to pass freely through it). Due to the permeability, gas exchange occurs by simple diffusion of O2 and CO2 into and out of the lungs down a concentration gradient (moving from an area of high concentration to low concentration).

As long as the alveolus is open (inflated, not collapsed) and the capillary is not blocked, oxygen will travel down its concentration gradient (from the lungs into the blood) and carbon dioxide will do the same (except this time it is traveling from the blood into the lungs). This allows the lungs to supply the blood stream with oxygen for delivery to the body's tissues and allows for extraction of CO2 waste from the blood whereby it can be exhaled into the atmosphere and removed.



Pulmonary Ventilation and the Mechanics of Breathing

The term "breathing" refers to a mechanical process by which coordinated muscular efforts result in the movement of air from the atmosphere into and out of the lungs. The mechanics of breathing involves coordinated contraction and relaxation of muscles in the thorax (see figure below) creating volume changes in the thoracic cavity, resulting in pressure changes. Based on those pressure changes, air will flow from the outside atmosphere via the nose/mouth through the respiratory tract and vice versa into and out of the lungs.

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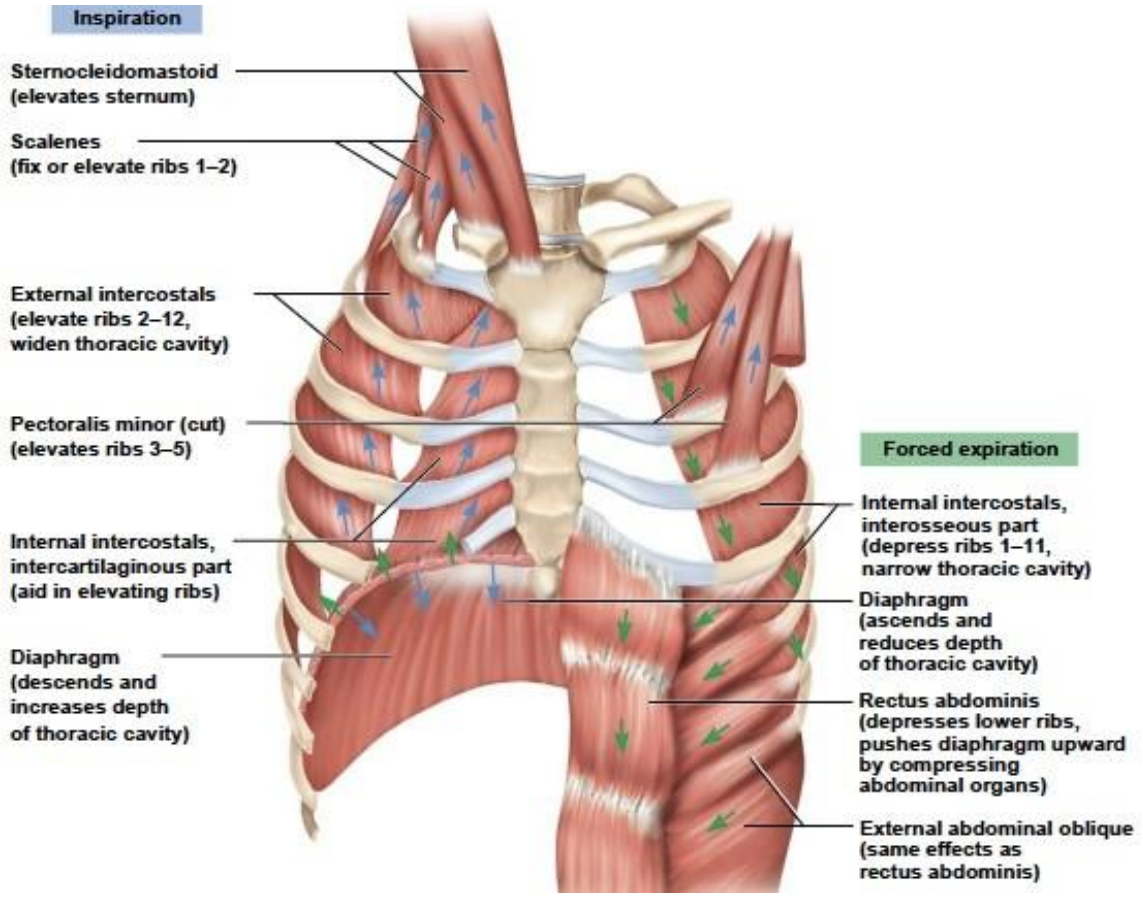

Ventilation and Breathing: a lesson in Physics

One of the basic principles in Physics is the relationship between pressure and volume within a container, also known as Boyle's Law. It is not necessary to know or remember Boyle's name, but it is important to understand the relationship between pressure and volume to understand breathing/ventilation in the awake patient breathing on their own (spontaneously) or in the anesthetized patient who is not breathing spontaneously.

To put it simply, if there are two containers, and each contains the same number of molecules, then the relationship between the pressure and the volume of those containers is equivalent. As the volume of the container gets smaller, the pressure increases, and as the volume of the container increases, the pressure decreases.

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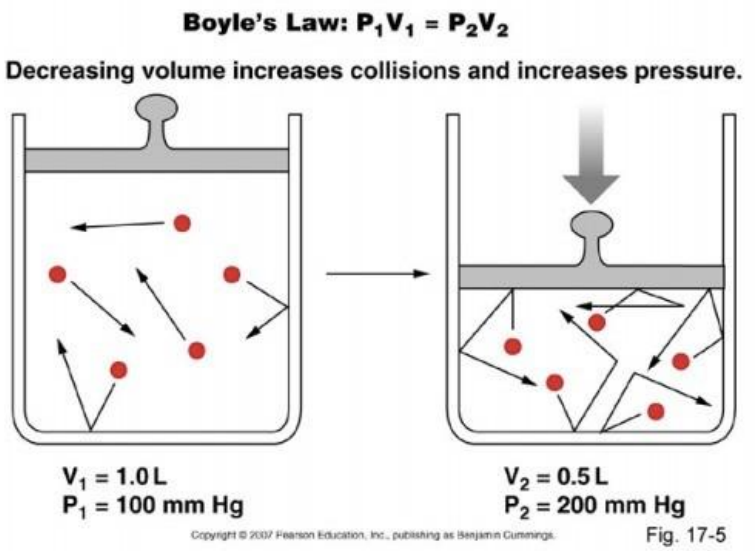
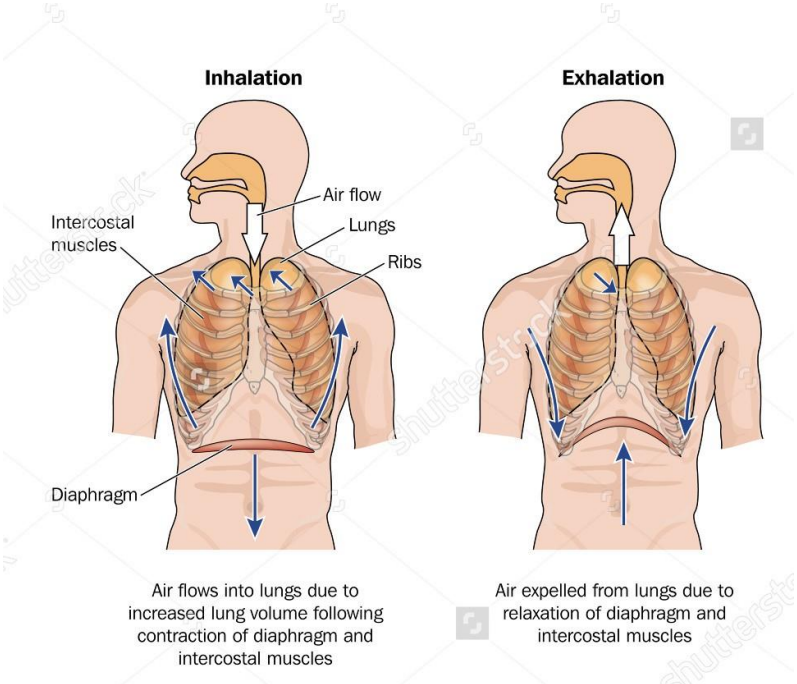


Spontaneous ventilation occurs when a patient is able to breathe on their own and occurs by pulling air into the lungs. It is also referred to as **Negative Pressure Ventilation** and is divided into two phases, inhalation and exhalation.

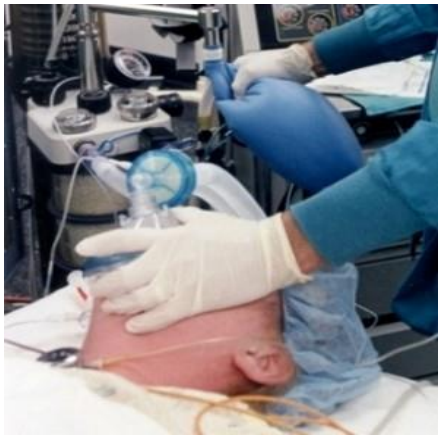
During **inhalation**, the inspiratory muscles contract (predominantly the diaphragm and external intercostal muscles, see above image), and the volume of the chest (thoracic cavity) increases. Due to Boyle's Law, as the volume of the thoracic cavity increases, the pressure decreases and becomes negative. The negative pressure of the thoracic cavity creates a "pulling" or vacuum-like effect on the respiratory tract which draws air into the lungs from the outside atmosphere via the nose or mouth, hence the term *negative pressure ventilation*.



Exhalation, conversely, is largely a passive process. When the muscles of inspiration relax, the elastic lungs and thoracic cavity recoil inward, in an attempt to return to its original shape and size, much like a balloon will deflate and return to its original shape and size. This recoil causes a decrease in the volume of the thoracic cavity, resulting in an increased pressure. The resultant increased pressure drives air out of the lungs via the respiratory tract and into the atmosphere. If desired, there are several muscles of expiration that may be used to create a forceful exhalation, such as the abdominal muscles and some intercostal muscles. These muscles act by decreasing the volume of the thoracic cavity, thereby increasing the pressure and pushing air out of the lungs. We use these muscles to blow forcefully and to cough/sneeze.

Positive Pressure Ventilation (PPV), refers to the process by which clinicians or ventilators may "breathe" *for* patients when they are unable to do so themselves, or *support* breathing in patients for whom spontaneous ventilation is inadequate. Both of these states may be encountered in a patient under the effects of anesthesia. This practice requires forcing air into the lungs through the respiratory tract via the generation of external positive pressure.

> **Remember*: this process actively "pushes" air into the lungs and differs from spontaneous ventilation whereby patient muscle contractions of the thoracic cavity creates a *negative* pressure which "pulls" air in.
>
> Example: Bag-Mask Ventilation, aka manual ventilation. The mask is placed over the patient's face and sealed tightly, preventing air from escaping. The continuous flow of air from the ventilator combined with the clinician squeezing the reservoir bag generates a positive pressure, which drives air into the lungs via the respiratory tract. Exhalation, much like in spontaneous ventilation, occurs passively due to the natural recoil of the lung and thoracic cavity when the positive pressure in the mask is released, allowing air to escape the lungs.



Respiratory Cycle:

A single respiratory cycle consists of both inhalation and exhalation. The total amount of air moved in one cycle is known as the *Tidal Volume (Vt).* The average tidal volume for an adult is approximately 450-700ml.

Respiratory rate:

The respiratory rate (RR) is the number of respiratory cycles per minute, commonly referred to as a frequency. For example, if a patient inhales and exhales 12 times in one minute, then their respiratory rate is 12. The average respiratory rate for adults is 10-14 breaths per minute.

Minute ventilation:

Minute ventilation (MV) is the amount of air moved into and out of the lungs per minute during ventilation. In order to calculate minute ventilation, simply multiply the volume of air in each respiratory cycle (tidal volume) by the number of breaths per minute (respiratory rate).

$$MV = Vt \times RR$$

If the average tidal volume is 500 mL/breath and the respiratory rate is 10 breaths per minute, the minute ventilation is 5000mL.

$$500mL \times 10/min = 5000\ mL/min$$

Work of breathing:

The work of breathing refers to the level of energy expenditure required during spontaneous breathing to adequately maintain proper oxygenation and ventilation in the patient. It is commonly measured as a percentage of total body oxygen consumption and typically accounts for 5% of the total amount of oxygen the body consumes. Remember that all cells consume oxygen in the process by which they create energy, so the amount of work a cell (or organ) is performing is directly correlated to the amount of oxygen consumed. In morbidly obese patients or critically ill patients, the effort of breathing may be increased 4-10 times that of a healthy person; therefore, breathing alone may consume up to 20-50% of their body's total oxygen consumption.

Lung Volumes

The term *Lung Volumes* refers to a group of volume measurements collected via spirometry that are used to extrapolate information for specific functions in respiratory physiology. The term "volume" henceforth is a direct measure of the amount of air for that function, while the term "capacity" is the sum of two or more volumes.

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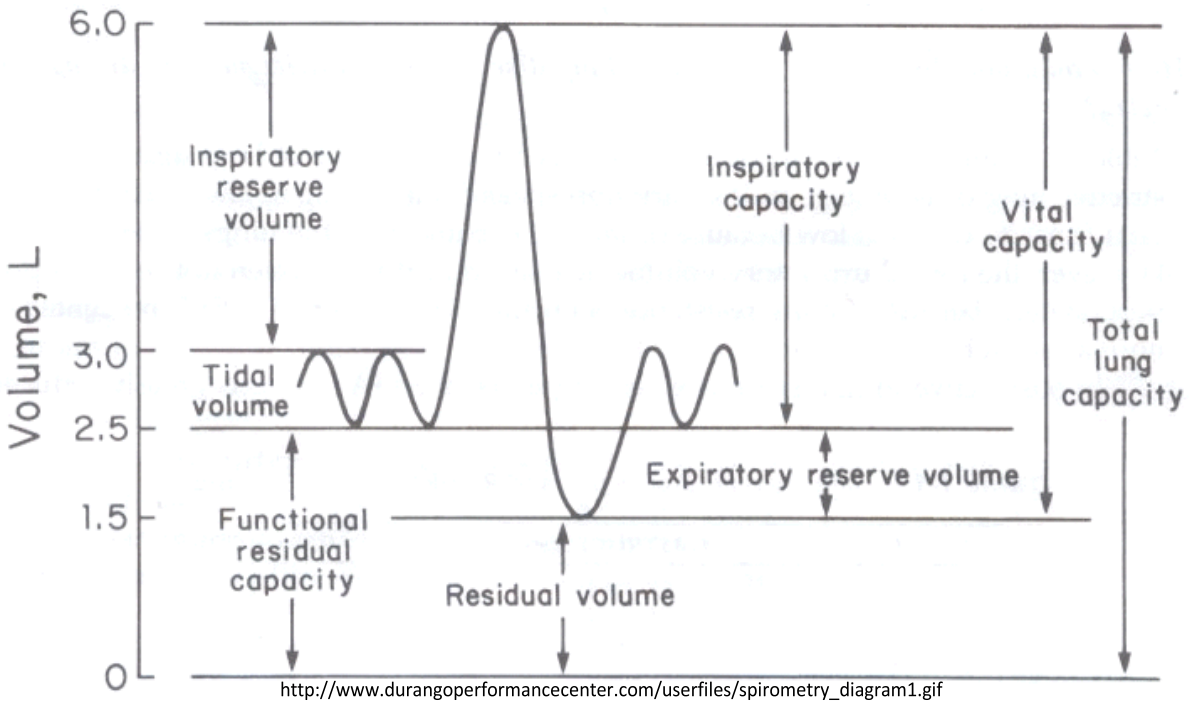

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- **Tidal Volume (TV) –** volume of air that moves into and out of the lungs with each respiratory cycle (approximately 500 mL under normal conditions).
- **Inspiratory Reserve Volume (IRV) –** volume of air that can be inspired forcibly with maximal inspiratory effort beyond the tidal volume (approximately 2100–3200 mL).
- **Expiratory Reserve Volume (ERV)** – volume of air that can be forcibly evacuated from the lungs with maximal expiratory effort beyond a tidal volume expiration (approximately 1000–1200 mL).
- **Residual Volume (RV)** – volume of air remaining in the lungs after maximal forced expiration (approximately 1200 mL).
- **Total Lung Capacity (TLC)** – sum of all lung volumes, aka total amount of air in lungs after maximal inspiratory effort (approximately 6000 mL).
- **Functional Residual Capacity (FRC)**** – amount of air remaining in the lungs after a passive tidal volume expiration. It is the sum of the Residual Volume and Expiratory Reserve Volume (RV + ERV).

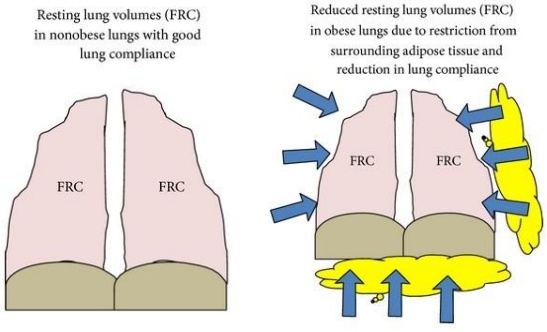
**FRC represents the volume of air present in the lungs at the end of passive expiration and is critically important to airway management and anesthesia. When large doses of anesthetic medications are given to a patient to induce general anesthesia, it is very likely that the patient

will stop breathing spontaneously. This is referred to as ***apnea***, defined as the temporary cessation of breathing. During this time, oxygen continues to be taken away from the lungs by the blood and tissues but fails to be replenished because the act of breathing has been halted. In this scenario, the patient continues to utilize the oxygen remaining in the lungs until it is exhausted, after which a lack of oxygen may contribute to extreme morbidity in the patient leading to tissue and organ injury, even brain damage and death. FRC is the volume remaining in the lungs during apnea and represents the volume of oxygen-containing air available for use until the patient resumes spontaneous ventilation or the clinician initiates mechanical ventilation. If one of these does not occur, the patient will eventually exhaust the stores of oxygen in the FRC, causing oxygen desaturation, hypoxemia and patient harm.

- During anesthesia and standard airway management procedures, there is always the risk that apnea may occur, either expected or not. To maximize the amount of oxygen in the FRC if this were to happen, supplemental oxygen is always given to patients receiving anesthesia. The concentration of oxygen in the normal air we breathe is 21%, which can be increased up to 100% using supplemental oxygen. During the process of pre-oxygenation prior to giving anesthetics, the air in the lungs is replaced with 100% oxygen, also referred to as *de-nitrogenation*. This multiplies the quantity of oxygen left remaining in the FRC almost 5 times during periods of apnea, thereby providing the patient with more oxygen reserves, ultimately prolonging the time to oxygen desaturation during apnea.

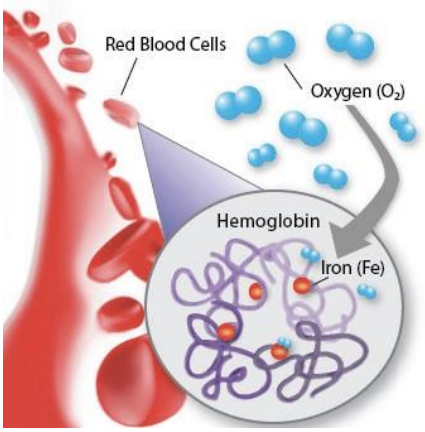
- There are many disease processes and clinical scenarios that cause a reduction in lung volumes, particularly the FRC. With a reduction in FRC, the amount of oxygen reserves available for use to the patient during periods of apnea is reduced, therefore the time to oxygen desaturation is also reduced. This poses great risk to the patient and must be taken into consideration by the clinician giving anesthetics.

    - Example: the FRC may be severely reduced in a morbidly obese patient secondary to the compression of lung tissue. The added abdominal and thoracic weight from obesity puts external pressure on the lungs and causes compression, decreasing the volume within (see figure below).



Hemoglobin and Oxygen Saturation

Hemoglobin (Hb) is a protein found in red blood cells and is the primary vehicle (about 98.5%) for carrying oxygen from the lungs to the body's tissues and returning carbon dioxide from the tissues back to the lungs. Oxygen is also carried dissolved in the blood's plasma, but to a *much lesser* degree. Hemoglobin is made up of four protein molecules, each containing an important iron-containing compound termed *heme*, which holds oxygen molecules for transport throughout the blood. Each hemoglobin molecule can bind up to four oxygen molecules and this combination of hemoglobin and oxygen is rapid and reversible, meaning the hemoglobin can easily load and unload oxygen molecules. The iron contained in heme is also responsible for the red color of blood.



When all four heme groups have attached to an oxygen molecule, the hemoglobin is fully saturated. If one, two, or three heme groups are bound to oxygen, the hemoglobin is partially saturated. The hemoglobin-oxygen combination is called *oxyhemoglobin*. The binding strength of the iron to the oxygen depends on the level of hemoglobin saturation. Once the first oxygen molecule attaches to the iron, the hemoglobin itself changes shape. As a result, it can more easily pick up the subsequent two oxygen molecules, and uptake of the fourth oxygen molecule is even easier. Similarly, as the hemoglobin releases each oxygen molecule, the strength of the bond between the iron and remaining oxygen molecules grows progressively weaker.

In general, hemoglobin saturation varies depending on the needs of the body at the time. Factors including temperature, blood pH, and partial pressures of oxygen and carbon dioxide can all influence the rate at which hemoglobin binds or releases oxygen molecules. These factors work together to maintain sufficient delivery of oxygen to and carbon dioxide removal from the tissues of the body. Since heat, declining blood pH, and rising levels of carbon dioxide are all by-products of active tissues hard at work in the body, these factors ensure that oxygen is unloaded where it is needed most.

When referring to the body as a whole, expressed as a percentage, the *oxygen saturation* is a measure of the oxygen-carrying capacity in the arterial blood. The amount of oxygen bound to

hemoglobin at any time is related to the amount of oxygen present in the surrounding tissue at the time (referred to henceforth as the *partial pressure* of oxygen). In the lungs, the partial pressure (amount of oxygen) is typically very high, therefore oxygen binds readily to hemoglobin as it passes by the alveolar-capillary interface. As the blood circulates throughout the body, the partial pressure of oxygen (amount of oxygen) is decreased because tissues are constantly consuming oxygen, which leads to further release of oxygen molecules from hemoglobin for tissue use.

Oxygen saturation levels may be measured in two main ways: including taking blood from an artery (called an arterial blood gas test) or using a pulse oximeter. An arterial blood gas test is considered more accurate; however, it is more invasive.

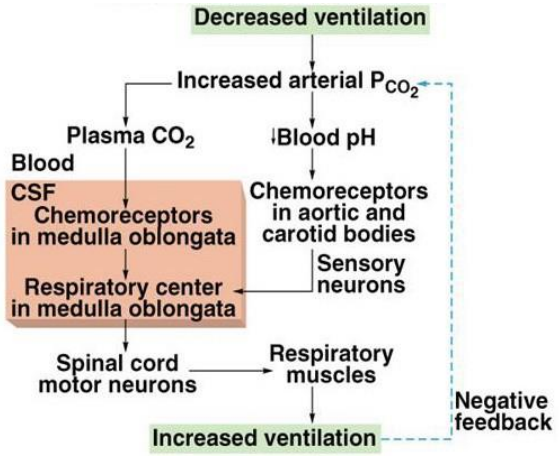
Normal oxygen saturation levels vary slightly depending on age and activity, but a saturation of 95-100% is considered normal and saturation less than 90% is considered abnormal. Remember that the cells of our organs require a continuous supply of oxygen to function properly and if oxygen saturation becomes too low (known as **hypoxemia**), various life-threatening problems can develop. Various conditions can cause a low oxygen saturation level in the blood. With breathing disorders, such as asthma or pneumonia, patients may not be able to get enough oxygen into their lungs. Other conditions, such as drug overdose or head injuries, can impair effective breathing, leading to a decreased amount of oxygen reaching the patient's lungs.

Carbon dioxide is also transported in the blood from the tissues to the lungs; however, it is somewhat different. It is carried by three mechanisms: dissolved in the plasma (7-8%), chemically bound to hemoglobin (carbamino hemoglobin 20%), and primarily as the bicarbonate ion in plasma (HCO3- 70%). CO_2 enters the red blood cell in the plasma where it is converted into HCO_3- via the enzyme carbonic anhydrase. When it reaches the lungs, HCO_3- re-enters the red blood cell and is converted back to CO_2 and diffuses across the alveolar membrane for removal during exhalation.

Control of Ventilation

Control of ventilation refers to the mechanisms involved to control spontaneous breathing. Under most conditions, the amount of carbon dioxide in the arterial blood (partial pressure of CO_2, aka PCO_2) or the amount of oxygen in the arterial blood (PO_2) controls the rate of ventilation. The sensors that measure PCO_2 in the blood are located in the medulla oblongata within the brainstem, whereas sensors for PO_2 lie in small organs situated near the aorta and carotid arteries. Information from these sensors is conveyed via nerves to the respiratory center in the reticular formation in the brain stem, which is responsible for alternately activating inspiratory and expiratory centers controlling coordinated muscle movements of inspiration and expiration.

Ventilatory rate and minute ventilation is tightly controlled by blood levels of carbon dioxide, and secondarily by blood levels of oxygen, as is determined by metabolic rate, carbon dioxide waste production, and oxygen utilization. The sensors in the brainstem immediately sense changes in CO_2 levels and change the rate and depth of breathing accordingly to maintain homeostasis. These mechanisms are involuntary, although we all know that control of ventilation can be affected voluntarily as well, by higher brain conditions such as emotional state, temperature, or free will. Keep in mind, however, that chemoreceptor reflexes are capable of overriding this voluntary control.



Anatomy and Physiology in the Sleep State

Regulation of respiration differs between sleep and wakefulness. Minute ventilation usually falls with the onset of sleep in response to both decreased metabolism and decreased chemosensitivity to CO_2 and O_2. Overall, the brain is less-responsive to changes in PCO2 and PO2, reducing respiratory responses to hypoxemia and hypercarbia. Despite the reduction in responsiveness, moderate to extreme changes in PCO_2 and PO_2 (hypercarbia or hypoxemia) may trigger arousals from sleep, resulting in a return to the more tightly regulated ventilatory control associated with wakefulness. The threshold for arousal in response to hypercarbia and hypoxemia differ among patients and among different sleep stages.

In addition to the changes in controller responses during sleep, there exist other significant sleep-related functional variation. The upper airway muscles are the most dramatically affected during sleep. The muscles surrounding the upper airway function to maintain patency and prevent collapse of the upper airway during inspiration. Reduced muscle activity along with redundant soft-tissue-related airway narrowing during sleep are cause for an increased risk for upper airway collapse and obstruction during sleep, leading to ineffective ventilation, ultimately resulting in hypercarbia and hypoxemia.

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Obstructive Sleep Apnea

In people with sleep-disordered breathing, ventilation pauses often occur during sleep due to the airways either completely or partially collapsing. Obstructive Sleep Apnea (OSA, where no ventilation occurs) and hypopneas (reduced ventilation due to partial airway obstruction) lead to intermittent increases in blood carbon dioxide levels and reductions in blood oxygen levels. The resultant hypercarbia and/or hypoxemia brings the patient to a more awake stage of sleep and can wake them up from deep sleep. Symptoms include snorting, snoring, gasping, and choking during sleep.

OSA is diagnosed by performing a sleep study whereby constant monitoring of physiologic parameters during sleep elucidate periods of apnea. It is characterized by repetitive partial or complete collapse of the upper airway (sometimes hundreds of times during the night in severe cases) that are accompanied by a $\geq$ 50% decrease in breathing amplitude, increased respiratory effort with arousal from a deeper to a lighter stage of sleep and/or an oxygen desaturation of $\geq$ 4%. The diagnosis includes $\geq$ 5 apneas plus hypopneas per hour of sleep. In the short-term, effects of OSA are characterized by excessive daytime tiredness not explained by other factors. In the long-term, these constant interruptions in ventilation associated with hypercarbia and hypoxemia lead to increased activation of the sympathetic nervous system, inflammation, and hormonal changes. As a result, patients with OSA have increased risk of hypertension, coronary artery disease, abnormal heart rhythms, stroke, diabetes, obesity, mood disorders (depression), and death.

The preferred treatment endorsed by the American Academy of Sleep Medicine for people with OSA is the use of positive airway pressure devices (CPAP) to aid breathing by preventing upper airway collapse during sleep and reducing apnea/hypopnea.

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Monitors of the respiratory system

The respiratory system is designed to deliver oxygen to the body and remove CO_2 from the body. There are three common monitors used in today's practice, which help clinicians determine how effectively their patient is delivering oxygen and removing CO_2:

1. Pulse oximetry
2. Capnography
3. Arterial blood gas

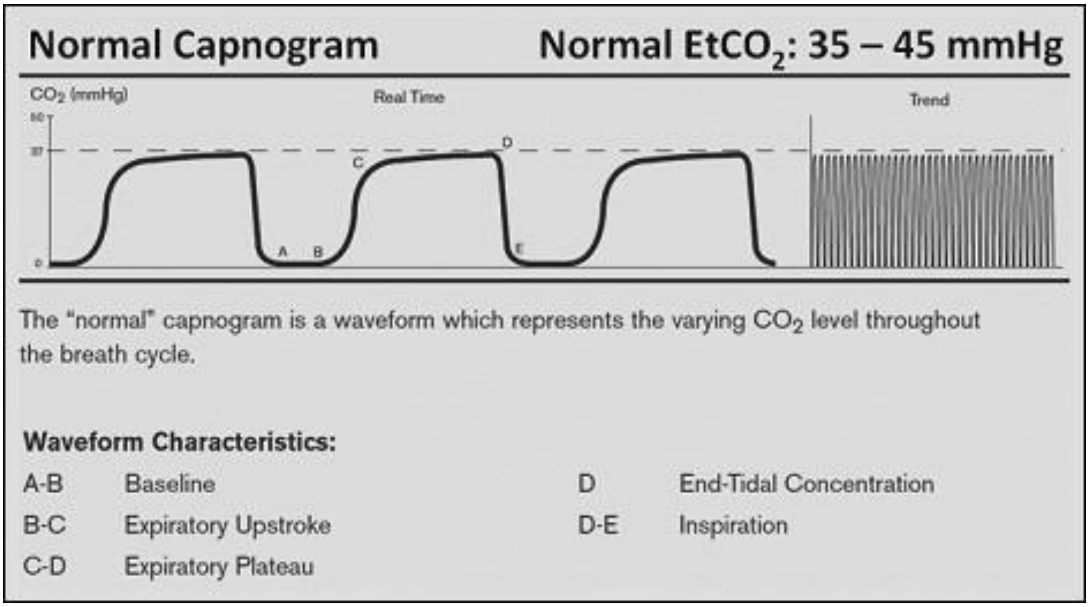
Oxygenation monitors measure the amount of oxygen in blood and Ventilation monitors measure the amount of CO_2.

The most common monitor for oxygenation is **pulse oximetry**. Pulse oximetry is a noninvasive method for monitoring a person's oxygen saturation (SO_2), the amount of oxygen bound to hemoglobin. It is a safe, convenient, noninvasive, inexpensive method for measuring how well a patient is being oxygenated.

The blood-oxygen monitor displays the percentage of blood that is saturated with oxygen. More specifically, it measures what percentage of hemoglobin molecules are loaded with oxygen molecules. The normal range for patients is from 95 to 100%. Less than 90% is considered life-threatening oxygen desaturation.

Although pulse oximetry is used to monitor oxygenation, it cannot measure ventilation. Therefore, it is not a complete measure of respiratory sufficiency. For example, a patient's oxygen saturation may read 100% because supplemental oxygen is driving oxygen into the blood. However, that same patient may not be breathing well, causing their CO_2 levels to be dangerously high.
The most common monitor for ventilation is **capnography**. Capnography refers to the measurement and display of CO_2, including end-tidal CO_2 (ie: CO_2 that is expired), inspired CO_2, and the CO_2 waveform, which is referred to as the *capnogram*. If end-tidal CO_2 ($EtCO_2$) is positive, it means that the CO_2 produced by the body and dissolved in the blood effectively

diffused into the alveoli and was exhaled. Therefore, end-tidal CO_2 is mainly used to determine whether or not a patient is breathing.



Capnometry refers to the measurement and display of CO_2 in numeric form. The normal value for end-tidal CO_2 is between 35-45mmHg. End-tidal CO_2 measurements higher than 45mmHg usually signify that the patient is not breathing effectively (ie: hypoventilating) and needs assistance. For example, deep sedation causes hypoventilation via decreased minute ventilation. Placing the patient on pressure support ventilation or bag-masking the patient may assist in ventilation and decrease the end-tidal CO_2 down toward normal values.

Although capnography effectively measures the elimination of CO_2 and ventilation, it cannot measure how well a patient is being oxygenated (ie: oxygen saturation). For example, a patient with a collapsed lung may have a normal end-tidal CO_2 value of 40mmHg, however, because only one lung is receiving oxygen, their oxygen saturation may be severely reduced.

The most common monitor for both oxygenation and ventilation is the **arterial blood gas (ABG).** An ABG test is the most commonly used method to measure both oxygenation and ventilation. It is an invasive procedure that involves taking blood from an artery and measuring the amounts of oxygen and carbon dioxide dissolved in the blood. An ABG test involves puncturing an artery with a thin needle and syringe and drawing a small volume of blood. The test is mainly used to determine the partial pressure of carbon dioxide (PCO_2) and oxygen (PO_2) as well as the oxygen saturation (ie: the percentage of oxygen bound to hemoglobin).

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FIO_2	0.3	
pH	7.43	
pCO_2	65	mmHg
pO_2	67	mmHg
Bicarbonate	43	mmol/L
Base excess	15	
O_2 saturation	92	%
Lactate	2.7	mmol/L

Chapter 2: Pathophysiology of Respiratory Compromise

Respiratory pathophysiology during anesthesia

Sedation scale

Sedation drugs are given to patients undergoing surgical procedures in order to suppress their Central Nervous System (CNS), which controls the brainstem and musculature. This helps to reduce anxiety about the procedure (ie: anxiolysis), relax muscles, decrease levels of consciousness (ie: hypnosis) and reflexes, and impair memory (ie: amnesia). Sedation drugs induce a state of relaxation by inhibiting signals to the brainstem and muscle. It is important to note that the level of sedation is a continuum that consists of minimal sedation, moderate sedation, deep sedation, and general anesthesia, where high levels of sedation can cause patients to be completely unconscious and stop breathing. Below are formal definitions of the different levels of sedation:

- **Minimal Sedation (Anxiolysis)** is a drug-induced state of mild relaxation and comfort during which patients respond normally to verbal commands. Typically, airway reflexes and breathing are unaffected.

- **Moderate Sedation ("Conscious Sedation")** is a drug-induced depression of consciousness during which patients have *purposeful* response to verbal commands, either alone or accompanied by light tactile stimulation. Again, airway reflexes and breathing are typically unaffected.

- **Deep Sedation** is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain breathing may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate.

- **General Anesthesia** is a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain breathing is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug induced depression of neuromuscular function.

Because sedation is a continuum, it is not always possible to predict how an individual patient will respond. Hence, practitioners intending to produce a given level of sedation should be able to rescue patients whose level of sedation becomes deeper than initially intended. Listed below is the ASA's Continuum of Depth of Sedation chart, which summarizes the definitions of the different levels of sedation.

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CONTINUUM OF DEPTH OF SEDATION:
DEFINITION OF GENERAL ANESTHESIA AND LEVELS OF SEDATION/ANALGESIA*

Committee of Origin: Quality Management and Departmental Administration

(Approved by the ASA House of Delegates on October 13, 1999, and last amended on October 15, 2014)

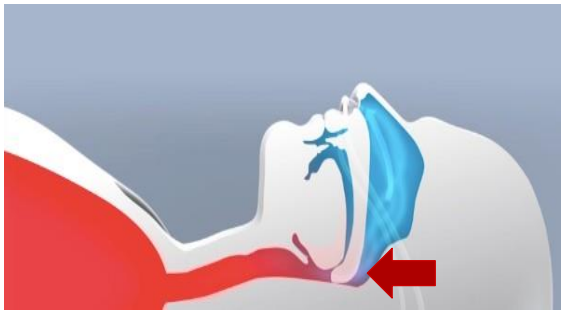
	Minimal Sedation Anxiolysis	*Moderate Sedation/ Analgesia ("Conscious Sedation")*	*Deep Sedation/ Analgesia*	*General Anesthesia*
Responsiveness	Normal response to verbal stimulation	Purposeful** response to verbal or tactile stimulation	Purposeful** response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous Ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular Function	Unaffected	Usually maintained	Usually maintained	May be impaired

Pathophysiology: Upper Airway Obstruction & Hypoventilation

Sedation drugs induce a state of relaxation by inhibiting signals to the brainstem and muscle. Relaxation of the brainstem directly depresses the respiratory center, which inhibits the drive to breathe, also known as *hypoventilation*. Relaxation of airway soft tissues results in collapse of the soft tissue into the airway causing a mechanical obstruction, referred to as an *Upper Airway Obstruction (UAO)*.

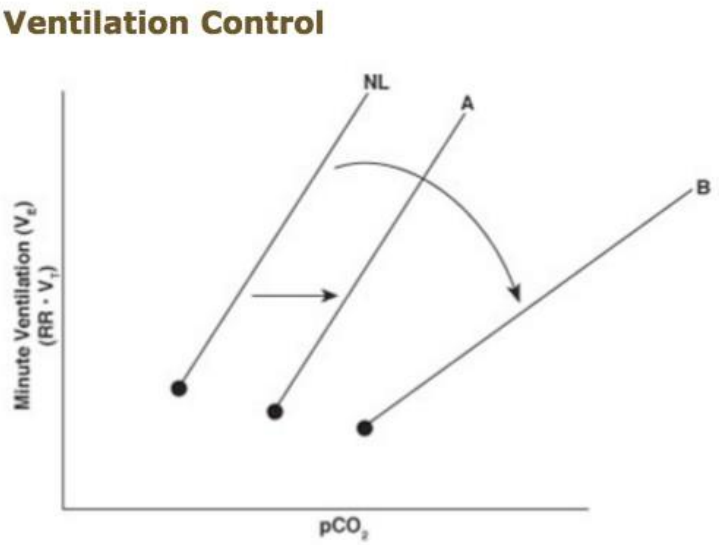
Upper Airway Obstruction

Sedation drugs inhibit signals to activate the muscles of the soft palate, which causes them to relax, leading to a mechanical obstruction of the upper airway. The obstruction prevents air and oxygen from entering the trachea while attempting to breathe.



Hypoventilation

The ability to breathe effectively is controlled by the central respiratory center, which is mainly located in the brainstem (Medulla Oblongata). The primary factor that drives us to breathe is changes in carbon dioxide (CO_2). Changes in carbon dioxide concentration are among the most important determinants of respiratory drive from the respiratory center. Under normal conditions, your minute ventilation typically increases linearly with rises in PCO_2 (ie: levels of CO_2 within your blood). The normal response to increases in carbon dioxide is noted by the line designated NL in the CO_2 ventilation response curve shown below.

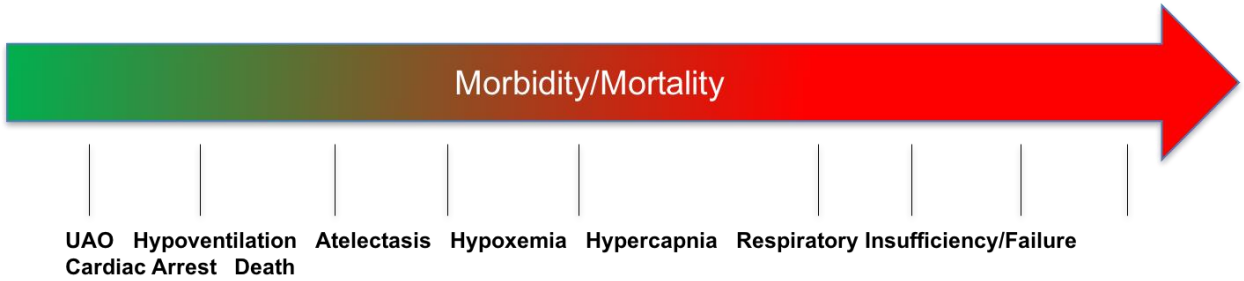


In general, sedative drugs depress the central respiratory center and decrease the drive to breathe even in response to increasing levels of carbon dioxide. This results in inadequate gas exchange, leading to hypoventilation. Line A represents a patient under minimal sedation. The brain is still responsive to changes in pCO_2 but has accepted a higher baseline level. Line B portrays deeper levels of sedation, where the slope of the CO_2 ventilation response curve decreases as well as shifts to the right. This shows that sedation is decreasing the brainstem's responsiveness to breathe. A decreased slope indicates less of an increase in minute

ventilation for any given rise in carbon dioxide, a situation that may lead to severe hypercapnia, hypoxemia, or apnea.
Another major drive to breathe is low blood oxygen (O_2) levels. Although, low blood oxygen levels are not as powerful a driver to breathe as CO_2 levels, it is still a contributor. However, sedation medications also decrease the respiratory centers drive to breathe in response to low blood oxygen levels.

Complications associated with UAO and hypoventilation

Hypoventilation and upper airway obstruction are the two mechanisms that result in a life-threatening complication known as Respiratory Compromise (RC). Respiratory Compromise is a continuum of respiratory complications that consists of minor complications such as oxygen desaturation to life-threatening complications such as respiratory failure. The diagram below shows the Respiratory Compromise continuum:



When a patient's airway is obstructed during sedation, oxygen cannot diffuse into the blood, which may result in oxygen desaturation. Simultaneously, sedation drugs depress the respiratory drive resulting in hypoventilation. Ineffective respiratory mechanics can lead to shallow breathing, resulting in collapse of some lung segments, called *atelectasis*. This further results in low blood oxygen levels, since the blood passing through those alveoli have no air to exchange gas with. Furthermore, since the patient's respiratory center is depressed, they cannot respond to the low blood oxygen levels. Altogether, this constellation of respiratory changes during sedation can result in their blood oxygen levels falling to life-threatening levels, which can lead to respiratory failure, cardiac arrest, anoxic brain damage, and potentially death.

All sedative drugs depress the Central Nervous System (CNS) in a dose-dependent manner, which means the higher the dose, the more the CNS is depressed. Typically, this is accompanied by a reduction in CO_2 responsiveness in the respiratory center resulting in respiratory depression and loss of airway control resulting in upper airway obstruction. UAO and respiratory depression are the most common serious adverse effects associated with sedative drug administration. The greater the degree of sedation, the greater the degree of respiratory depression. Respiratory depression increases when combining sedative drugs or when using large doses of a single drug.

Clinical example of Upper Airway Obstruction and Hypoventilation

An obese patient is to undergo an upper endoscopy (ie: EGD). Supplemental oxygen is placed on the patient via nasal cannula and the patient is induced with Propofol. Under sedation, the tongue quickly collapses into the soft palate and obstructs the airway. If not recognized immediately, the patient may begin to desaturate and approaches Respiratory Compromise if interventions are not quickly employed. In this clinical scenario, effective ventilation was not possible secondary to upper airway obstruction. Pre-oxygenation was performed with nasal cannula but was inadequate to maintain oxygenation in this obese patient. The clinician at this point would use positive pressure ventilation to relieve the upper airway obstruction and assist breathing for the patient.

Question: if a sedated patient is receiving supplemental oxygen and has a patent airway, can they still suffer Respiratory Compromise?

Yes. Despite breathing a higher percentage of oxygen with supplemental oxygen, a low minute ventilation may still result in respiratory compromise. Since sedation also causes hypoventilation, the ideal solution would provide supplemental oxygen, relieve upper airway obstruction, *and* support ventilation in sedated patients. This can be done by continuously supporting the patient's breaths, by using intermittent recruitment maneuvers, or by doing both.

Recruitment breaths: very large vital capacity breaths that are delivered to patients who are at high risk for atelectasis in order to recruit and re-expand alveoli that may have collapsed. This is a well-documented technique to help prevent and treat Respiratory Compromise and maximize gas exchange.

Respiratory Compromise

Since Respiratory Compromise is a continuum it is important to know what the incidence is of each individual complication. For high-risk patients, which is hereby defined as a patient with either Morbid Obesity, Obstructive Sleep Apnea, heart failure, or lung failure, the incidence of respiratory compromise following general anesthesia is:

Respiratory Complications	Incidence
Hypoxemia	25-40%
Atelectasis	20%
Pneumonia	2.4%
Respiratory insufficiency	6.5%
Respiratory failure	5.1%

Risk factors for respiratory compromise

Respiratory Compromise is a result of either low blood oxygen levels and/or high carbon dioxide levels. Therefore, patients who start with low oxygen levels (ie: low oxygen reserve) or

high CO_2 levels are at a higher risk. The six most significant risk factors for Respiratory Compromise include:

1. Morbid Obesity
2. Obstructive Sleep Apnea (OSA)
3. Congestive Heart Failure (CHF) with Pulmonary Edema
4. Chronic Obstructive Pulmonary Disease (COPD)
5. Pneumonia
6. Patients that are difficult to ventilate and intubate.

Morbid obesity

There are three reasons why morbidly obese patients are at a higher risk for Respiratory Compromise. First, they have a significantly larger amount of pharyngeal soft tissue, which increases their risk for Upper Airway Obstruction under sedation. Second, their larger amount of abdominal tissue compresses their lungs, resulting in decreased FRC, increased atelectasis, and a reduction in oxygen delivery. Third, the oxygen consumption in obese patients may be up to 5 – 10x higher than average. Therefore, not only do they have less oxygen entering their blood, but their oxygen requirements are also much higher.

Obstructive Sleep Apnea

The first reason for increased risk in this population is the tendency towards upper airway obstruction. A patient who obstructs during normal sleep will certainly obstruct during anesthesia, leading to increased risk for hypoxemia and hypercarbia. Second, over time chronic hypercarbia and hypoxemia leads to a reduced responsiveness of the normal respiratory center in the brain. This leads to chronically elevated pCO_2 levels at baseline, and a further reduced responsiveness under anesthesia. Therefore, even small amounts of sedative medications can cause significant depression in respiratory drive.

Congestive Heart Failure/pulmonary edema

Congestive Heart Failure (CHF) is associated with two processes that significantly decrease the oxygen reserves and therefore increase risk for RC. First, the heart is unable to pump sufficiently to maintain blood flow to meet the body's demands, which means that the amount of oxygen being delivered to the patient is less than what the body is consuming (ie: oxygen delivery < oxygen demand). Second, the heart as a pump may be so weak that it cannot effectively pump the blood out, leading to a backup of blood in the pulmonary system (like traffic). This causes blood to back up into the lungs (ie: alveoli) resulting in fluid to be built up (ie: pulmonary edema). As fluid fills the alveoli, there is no opportunity for air or oxygen to enter, and therefore no opportunity for gas exchange in those lung segments.

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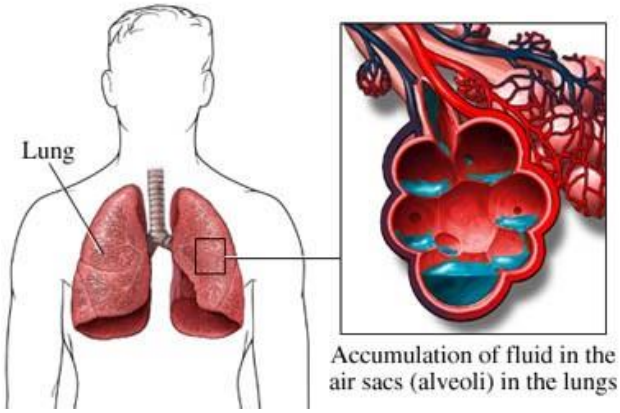
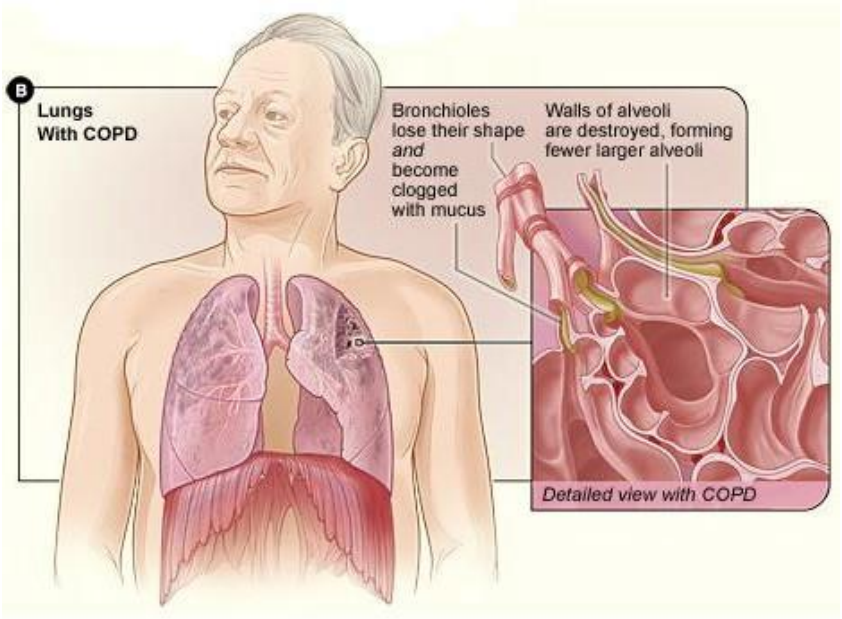


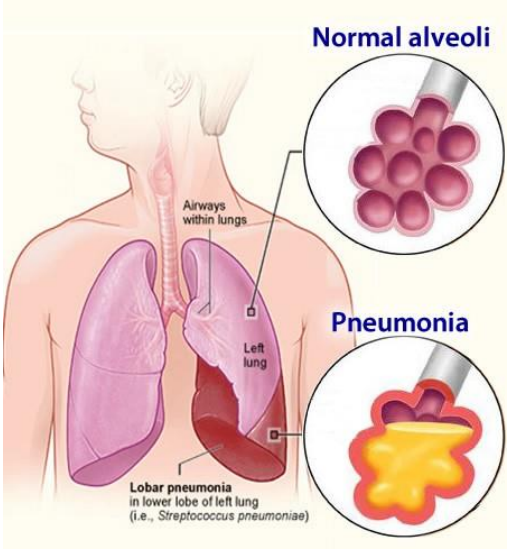
COPD

COPD is a type of obstructive lung disease in which poor airflow and inability to breathe out fully (air trapping) exist. Two processes cause poor airflow. The first process is the breakdown of lung tissue, which prevents a significant amount of oxygen from being able to diffuse from the alveoli into the blood. For example, normal lungs have several million alveoli, which can be filled and allow oxygen to diffuse into the blood. In COPD, more than half of their alveoli are destroyed, which leaves them only able to fill half as many alveoli with oxygen for diffusion. The second process is due to inflammation of the small airways, which blocks the small airways and prevents the patient from exhaling CO_2. Since a significant amount of CO_2 remains in the lungs, it prevents oxygen from entering. Therefore, patients with COPD have significantly less oxygen in their lungs because they have less alveoli and CO_2 taking its place. These patients are a very high risk for oxygen desaturation once sedation is induced because they have significantly less oxygen stored in their alveoli.



Pneumonia

Pneumonia is any inflammatory process that affects the alveoli. The most common causes of pneumonia are viral and bacterial infections. Inflammation of the alveoli causes fluid to build up. Similar to pulmonary edema, the fluid within the alveoli prevents oxygen from entering and participating in gas exchange, resulting in increased risk for hypoxemia and hypercarbia.



Known difficult intubation or ventilation

The most dangerous situation in all of airway management is "can't intubate, can't ventilate," where the clinician attempts both mask ventilation and endotracheal intubation, and fails at both. Since the patient is not receiving oxygen or eliminating CO_2, they are at risk of desaturation resulting in cardiac arrest and eventually death. To avoid this life-threatening situation, clinicians are trained to predict which patients will be difficult to ventilate and/or intubate by identifying risk factors. There are five risk factors associated with difficult mask ventilation and numerous risk factors associated with difficult intubation. If a patient is at risk for either difficult mask ventilation or difficult intubation it is vital to provide the patient with a continuous source of oxygen in order to maintain their oxygen saturation levels and prevent life-threatening Respiratory Compromise. Some clinicians limit sedation medications in these patients, or avoid them altogether.

Difficult mask ventilation

Difficult ventilation has been defined as the inability of a trained anesthetist to maintain the oxygen saturation > 90% using a facemask for ventilation and 100% inspired oxygen. Below is a list of risk factors associated with difficult ventilation.

- Body mass index (BMI) > 26
- Age > 55
- Edentulous (ie: no teeth)

- Beards
- History of snoring

Patients that are obese (BMI > 26) or have a history of snoring are difficult to ventilate because they have an abundance of soft tissue surrounding their airway that collapses into the airway once they are sedated. The more soft tissue that collapses into the airway, the more pressure that will be required to stent it open. Patients that are over 55 years old are also more likely to have obstruction under anesthesia secondary to decreased muscular control of the upper airway and depressed airway reflexes associated with age. Having no teeth (edentulous) is a risk factor for difficult ventilation because the oral cavity collapses when closed, forcing the tongue posterior into the soft palate. In addition, it is more difficult to create a mask seal around the mouth, making leaks more common.

Finally, patients that have beards are at risk for difficult ventilation because the facial hair creates an uneven surface, over which it is difficult to create a proper air-tight seal. This causes gas to leak out around the mask, which prevents positive pressure from being generated.

Difficult intubation

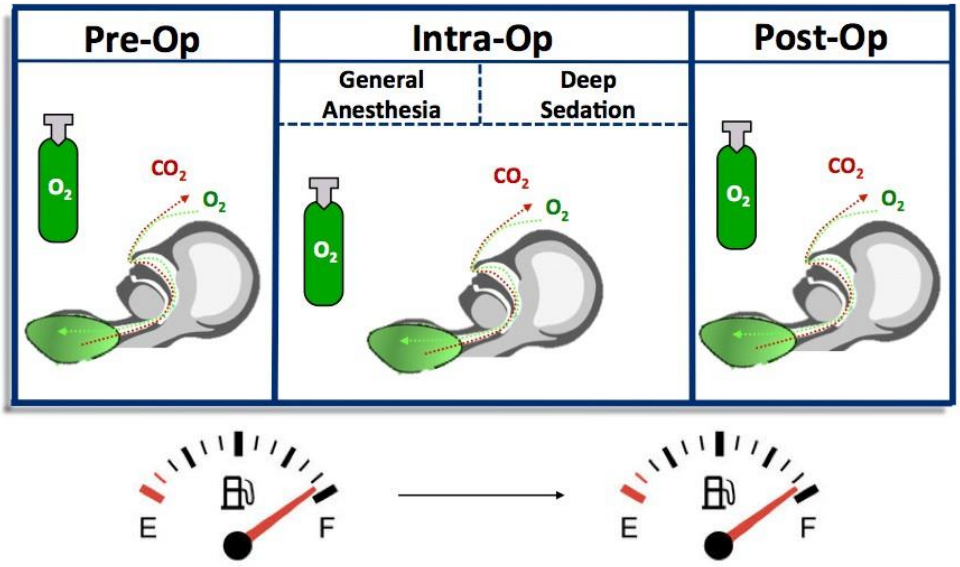
Difficult intubation has been defined by the need for more than three intubation attempts or attempts at intubation that last > 10 min. Below is a list of some of the risk factors associated with difficult intubation:

- History of difficult intubation
- Small mouth opening
- Increased Mallampatti class
- Glottic view grade > III
- Thyromental distance < 6cm
- Limited neck mobility

If a patient has one or more of these risk factors, there is a higher probability that the intubation will be difficult and the risk of life-threatening Respiratory Compromise will be high.

Perioperative management for the different levels of anesthesia

The peri-operative period consists of the pre-operative, intra-operative, and post-operative periods. If you think of the patient's lungs as a fuel tank, then the objective is for the lungs to be completely filled with O_2 and eliminated of CO_2 (see image below).



Although the ultimate goal is to maintain oxygenation and ventilation throughout the perioperative period, regardless of the level of sedation, there are a few differences in objectives between deep sedation and general anesthesia.

In order to achieve the goal of maintaining oxygenation and ventilation throughout the peri-operative period during a ***deep sedation procedure***, the following objectives must be addressed:

- Provide a source of continuous passive oxygenation throughout the procedure
- Maintain airway patency without an advanced airway
- Monitor ventilation
- Support ventilation with or without PPV when necessary

However, in order to maintain oxygenation and ventilation throughout the peri-operative period during a ***general anesthesia procedure***, the following objectives must be addressed:

- Pre-oxygenate with 100% O_2 to prolong apnea time to desaturation
- Place an advanced airway to maintain airway patency
- Support ventilation with PPV

Current clinical solutions and their limitations

Passive oxygenating devices

A passive oxygenating device is any device that provides a continuous flow of oxygen. Oxygen delivery here depends on the patient breathing spontaneously. These devices are not capable of producing positive pressure and delivering ventilatory support. Examples of oxygen devices

are the nasal cannula, supplemental oxygen mask, and the POM mask.

Disadvantages: It is very important to understand that the major disadvantage to an oxygenation device is that they are open to the atmosphere and therefore cannot create a seal (ie: closed system). When a system is open to the atmosphere it entrains air, therefore lowering the amount of oxygen concentration delivered and it creates leaks, which prevents pressure from being generated. Without positive pressure, the device is inadequate to treat upper airway obstruction or mask ventilation.

Nasal cannula with capnography is a passive oxygenation device that can deliver an FiO_2 between 30-40% and measure $EtCO_2$ at flows < 5lpm. It cannot be used as a pre-oxygenation device as it cannot completely eliminate nitrogen from the alveoli and it cannot be used to relieve upper airway obstruction or to maintain ventilation since it cannot generate positive pressure.

Supplemental oxygen mask is a passive oxygenation device that can deliver an FiO_2 between 50-70%. It cannot measure $EtCO_2$ and cannot be used as a pre-oxygenation device or be used as a positive pressure device to relieve upper airway obstruction or maintain ventilation.

POM mask is a passive oxygenation device that can deliver an FiO_2 between 50-92% and measure $EtCO_2$ at flows < 5 L/min. It has a fenestration in the middle of the mask, which allows

an endoscope to be passed. However, both the fenestration and the mask cannot seal. Therefore, it cannot be used to relieve upper airway obstruction or to maintain ventilation since it cannot generate positive pressure.

High flow nasal cannula

It is an open system, oxygenation device, that can provide up to 70L/min of oxygen to the patient via nasal cannula. Despite being an open system, at such high flows it is able to provide a mild continuous positive airway pressure (CPAP) between 2-5cmH_2O. Contrastly, because of the extremely high flows, it cannot measure EtCO_2 as the CO_2 is washed out (ie: diluted). Limited to only 2-5 cmH2O, it cannot provide pressures high enough to reliably relieve upper airway obstruction or perform positive pressure ventilation. Other disadvantages include:

- Requirement of capital equipment (HFNC heat and humidifier) costing ~ $10,000 plus the additional cost of the disposable nasal cannula per case.
- It lacks the ability to provide positive pressure ventilation, which requires an anesthesia mask to always be readily available.
- Minimal efficacy on obese patients.
- Not indicated to be a rescue ventilation device.

Non-invasive ventilation (NIV) Devices

When spontaneous ventilation is inadequate or absent secondary to anesthetic medications, the lungs must be actively ventilated using positive pressure. The goal is to inflate the lungs and allow for deflation, whereby gas exchange will allow for the delivery of oxygen to the blood and the removal of carbon dioxide. A ventilation device is any device that creates a

closed- sealed system, which allows positive pressure to be generated within the airway, stent open an obstruction and actively inflate the lungs. Examples of ventilation devices are the SuperNO_2VA™, an anesthesia mask, and a CPAP mask.

Nasal CPAP mask

The market leaders are Philips Respironics and Resmed. Conventional CPAP was designed to be used at home by patients with obstructive sleep apnea. A moderately tight-fitting mask seals around the nose and/or mouth and an external CPAP machine regulates a high flow of air into the mask. This flow of air is capable of delivering a positive pressure adequate to relieve upper airway obstruction. The mask itself is designed to have expiratory leaks and vents, in order to protect the patient from over-pressurization in an un-monitored setting. Additionally, very high flow rates are required (60-100 L/min) to overcome these leaks, but overall the amount of pressure able to be generated is severely limited. Attempting to use a nasal CPAP mask in the OR has several disadvantages such as:

- Cannot be used as a resuscitation mask for positive pressure ventilation because it cannot support pressures >20cmH2O, which are frequently needed in anesthetized and paralyzed patients.
- Have expiratory leaks, which prevents the use of high pressures and the use of anesthetic gases, since they will leak into the atmosphere.
- Requires very high flows, which inhibits the ability to measure $EtCO_2$ due to washout.
- Requires an additional piece of capital equipment because standard anesthesia equipment and ventilators cannot support such high flow rates.
- Disrupts the workflow having to call respiratory therapy and set it up.

NOVA technique utilizing a pediatric anesthesia facemask and head strap

The pediatric mask has several significant disadvantages such as:

- Tedious set up that requires multiple products and negatively impacts workflow.
- Partially obstructs the glottic view during intubation.
- High risk of eye injury (ie: corneal abrasion) from the headstrap.
- Cannot provide high flow nasal oxygen therapy (only provides low flow nasal oxygen therapy <15L/min), which is essential for high-risk patients such as the morbidly obese and critically ill.

Tse-PAP assembly utilizing a pediatric anesthesia facemask, head strap, and nasal cannula capnography

Tse-PAP is very similar to the NOVA technique set up. The only difference is the addition of a nasal cannula that can measure $EtCO_2$. The Tse-PAP assembly has the same disadvantages as above with the NOVA technique.

Anesthesia Full Facemask

The standard anesthesia full facemask has been used for over a century in anesthesia and remains the gold standard for pre-oxygenation and mask ventilation for patients requiring PPV. It covers the nose and the mouth and connects to standard anesthesia equipment. A detailed description of full facemask ventilation is covered in Chapter 3. By covering the mouth, it cannot be used during intra-oral procedures (e.g. EGD, TEE, laryngoscopy, bronchoscopy, etc.) severely limiting its use during sedation and general anesthesia.

Competitive advantage	Nasal cannula	High Flow nasal cannula	Anesthesia mask	Nasal CPAP	SuperNO$_2$VA™
Oral access	●	●	○	●	●
Passive oxygenation	●	●	●	●	●
High flow O_2	○	●	○	○	●
Rescue ventilation/RC	○	○	●	○	●
Additional capital equipment	Not required	●	Not required	●	Not required

Chapter 3: Intro to Nasal Ventilation & the SuperNO_2VA™

The ideal solution

In today's practice, there is an increasing frequency of deeper levels of sedation without the use of an advanced airway resulting in Respiratory Compromise secondary to upper airway obstruction and hypoventilation. Deep sedation has become a preferred method because it renders the patient amnestic, allows for physical relaxation during highly stimulating procedures, and improves turn-around time. Also, there has been an increase in the number of procedures that require access to the patient's mouth, such as upper endoscopy, transesophogeal echocardiography, and laryngoscopy, which significantly increases the risk of RC due to the inability to apply positive pressure ventilation as the current devices cover both the nose and mouth.

In order to address Respiratory Compromise the ideal solution needs to address the following objectives. Understanding these principles is vital for successful communication with clinicians and comparison to other already marketed devices.

1. Effectively pre-oxygenate
2. Relieve Upper Airway Obstruction
3. Maintain effective ventilation in unconscious patients
4. Provide access to oral cavity during intra-oral procedures

1. For effective pre-oxygenation and denitrogenation to occur, the solution must deliver a high percentage of FiO_2. The entire lung volume and FRC must be exchanged with oxygen to maximize the stores of oxygen the patient has access to during apnea, prolong the time to oxygen desaturation and thus reduce RC. All patients who receive **anesthesia incur the risk of apnea, and current recommendations are to maximize oxygen stores during anesthesia to best prepare for apnea, even if it is not expected.**[1]
2. It must relieve upper airway obstruction since many patients who receive anesthesia develop upper airway obstruction, due to relaxation of the muscles of the airway and decreased central (brain) control of ventilation. Current methods employed during sedation, such as apneic oxygenation and passive supplemental oxygenation, are not capable of generating positive pressure and thus are not equipped to relieve UAO, thereby rendering them effectively useless in these situations.
3. As apnea is common and at times unpredictable during anesthesia, the device must be capable of maintaining effective ventilation in unconscious patients. Ideally, a single device should be preventative, therapeutic, and capable of rescue.
4. It must provide sufficient access to the oral cavity for intra-oral procedures, yet still be able to provide rescue ventilation when needed. What good is a respiratory device if it cannot be used during the procedures where respiratory compromise is most common (e.g. EGD, TEE, laryngoscopy, etc.)?

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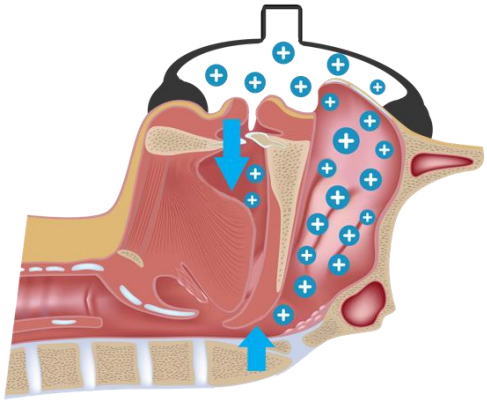
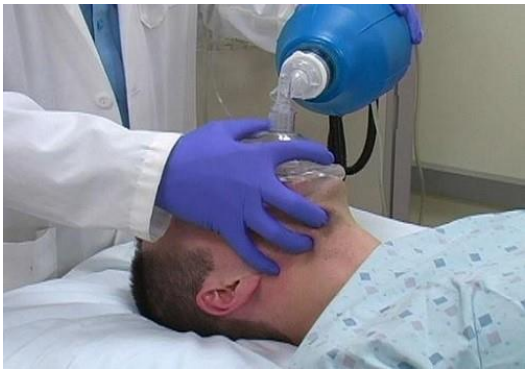
Introduction to nasal ventilation

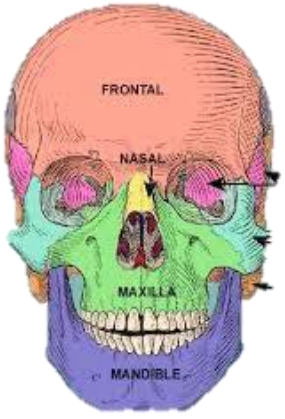
Full facemask ventilation

Historically, the only type of mask ventilation technique performed was full facemask ventilation, where a ventilation mask is place over the patient's nose and mouth. A positive pressure is delivered to both the oral and nasal cavities and air/oxygen is driven into the airway and lungs. By understanding the anatomy of the upper airway (refer to image below), we can see that a positive pressure within the mouth may force the tongue posterior towards the pharynx, which would be counterproductive. A posteriorly-displaced tongue may contribute to upper airway obstruction by narrowing the posterior pharynx.



In addition, traditional mask ventilation training with a full facemask teaches the clinician to lift the patient's face to the ventilation mask in order to create a seal. The reason for this is because the lower mandible is mobile. Therefore, if one pushes the facemask into the face, the mandible moves downward and the jaw is forced toward the chest, contributing to upper airway obstruction by collapsing the soft tissue around the airway. For these reasons, the technique is to lift the mandible into the facemask and squeeze together to form a seal. This is a difficult skill to master; it is the first airway skill we learn in anesthesia and is the reason many non-anesthesia trained personnel are poor at mask ventilation.





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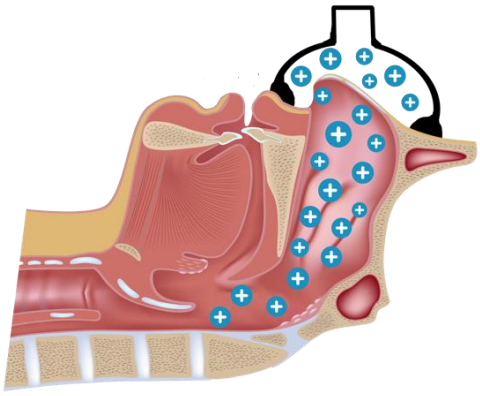
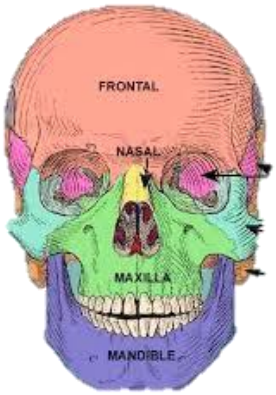
Nasal mask ventilation

Nasal mask ventilation is a technique gaining recognition only recently, which provides positive pressure solely through the nose. Rather than having the pressures delivered to the oral cavity and the tongue forced back into airway, pressure is only delivered to the nasal cavity. By pressurizing behind the soft palate and tongue, we effectively create a pneumatic stent, which forces the tongue forward, maximizes the cross area of the pharynx, decreases resistance, and relieves the obstruction.



The technique for nasal ventilation is different from that with a full facemask. All of the structures surrounding the nose are rigid and immobile; the objective is to push down firmly into those structures. Downward pressure of the mask into the face does not cause a displacement of the mandible and does not disrupt head position. To “close” the system and create positive pressure, the provider must occlude the oral cavity in order to prevent an air-leak out of the mouth. This can be done manually by simply holding the mouth closed or the clinician may press firmly against the soft tissue below the jaw (ie: the tongue in the submental space) toward the head pushing the tongue against the hard palate, occluding the oral cavity internally. By occluding the oral cavity fully, positive pressure is generated in the nasal cavity, the soft palate and tongue are displaced forward/anterior, and air/oxygen may be driven into the trachea and lungs.



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Clinical benefits of nasal ventilation

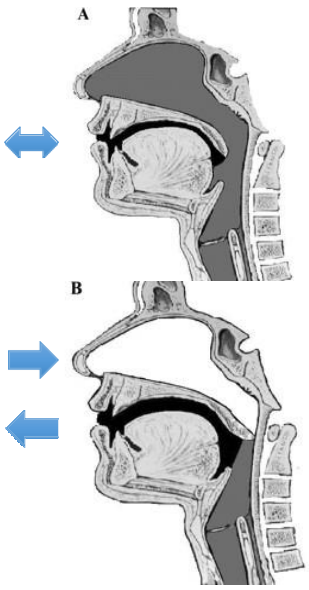
Nasal ventilation has several benefits over full facemask ventilation. First, it can provide 100% FiO_2 during procedures that require access to the oral cavity.[1] Another advantage is it reduces anatomical dead space by up to 50%, which improves effective alveolar ventilation.[2] This occurs because only the nasal cavity is used to deliver air/oxygen to the lungs and not the oral cavity. In addition, it allows the ability to apply positive pressure ventilation (ie: nasal CPAP and nasal ventilation) during intra-oral procedures. Nasal CPAP has been shown to be effective at relieving upper airway obstruction due to obstructive sleep apnea and secondary to sedation medications.[3,4]

In several studies, nasal mask ventilation in apneic patients was studied head to head against full facemask ventilation and was found to be superior in unconscious, anesthetized patients.[5] For example, effective nasal mask ventilation was achieved using lower peak airway pressures than full facemask for the same delivered tidal volumes. It has also been shown to have a higher rate of relieving upper airway obstruction and higher rates of successful ventilation.

Improved Anatomic Dead Space

Anatomic dead space refers to the total volume of the conducting airways. It is an important percentage of the tidal volume but does not participate in gas exchange. For normal spontaneous ventilation, anatomic dead space includes the volume of the nose, mouth, pharynx, trachea, and bronchi/bronchioles. We breathe this volume as part of our tidal volume, but is exhaled unchanged. In contrast, during exhalation air from the lungs that has already participated in gas exchange (deoxygenated with high CO_2 content) is exhaled into the dead space and never released into the atmosphere. Thus, upon inspiration we re-breathe a certain volume of air that is depleted in oxygen, and rich in carbon dioxide. Alveolar ventilation, the volume of air per minute reaching the alveoli that can participate in gas exchange, can be calculated in the same way as minute ventilation, subtracting the dead space volume from the tidal volume.

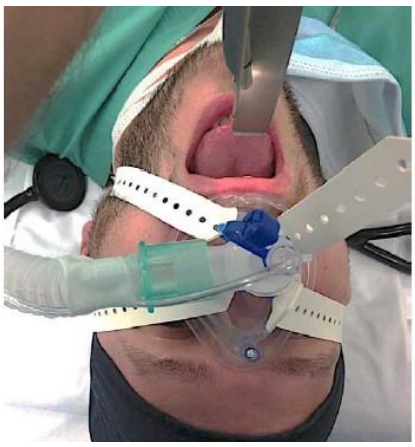
$$AV = (TV – DS) \times RR$$



By minimizing the percentage of tidal volume that is comprised by anatomical deadspace, we can improve alveolar ventilation and gas exchange without changing tidal volume or respiratory rate. With normal breathing, we inhale/exhale with our nose and mouth, creating bi-directional flow in both the nasal and oral cavities. With a pressurized nasal device, such as the SuperNO_2VA™ device, air/oxygen is inhaled via the nasal cavity only, therefore the volume of the oral cavity is removed from normal tidal volume. This decreases the anatomical dead space by up to 50%, improving alveolar ventilation and allowing for improved gas exchange.

NOVA Technique

An additional advantage of providing continuous nasal oxygenation and ventilation is the Nasal Oxygenation and Ventilation of the Airway (NOVA) technique, described by Drs. Michael Pedro and Steven Cataldo. It utilizes continuous nasal oxygenation and positive pressure ventilation during procedures that involve the oral cavity and was described as a technique designed to reduce life-threatening desaturation during rescue intubation.[6]



Introduction to apneic oxygenation and ventilation

Apneic oxygenation

Apneic oxygenation refers to the delivery of a high FiO_2 to the oropharynx and hypopharynx that is passively entrained into the lungs during apnea, extending the time to desaturation. It has been successfully described to prolong the safe apneic period during laryngoscopy and intubation by employing a nasal cannula at 15 L/min. Its efficacy is limited in patients with poor oxygen reserves or high oxygen demands (morbid obesity, OSA, and critical illness).

Apneic oxygenation relies on the discrepancy between the rate at which oxygen is normally removed from the alveoli compared with that at which CO_2 is typically delivered to them. This occurs due to differences in the volume of oxygen consumed versus the volume of CO_2 produced. During apnea it is estimated that CO_2 enters the alveoli at a rate of only ~10 ml/min while O_2 is removed at a rate of ~250 ml/min. This 240 ml/min net removal of gas volume from the lungs during periods of apnea results in a reduction in pressure. Essentially, this net movement of gas from the lungs into the blood causes a vacuum-like effect, which is transmitted to the upper airway, entraining air into the lungs. If nasal oxygenation via nasal cannula is being provided and the upper airway is patent, the air that is entrained from the hypopharynx will have a high oxygen content, therefore delivering additional oxygen to the lungs for delivery during apnea. This process of apneic air movement continues during periods of apnea as it does not depend upon the generation of positive pressure in the upper airway nor on respiratory effort by the patient.

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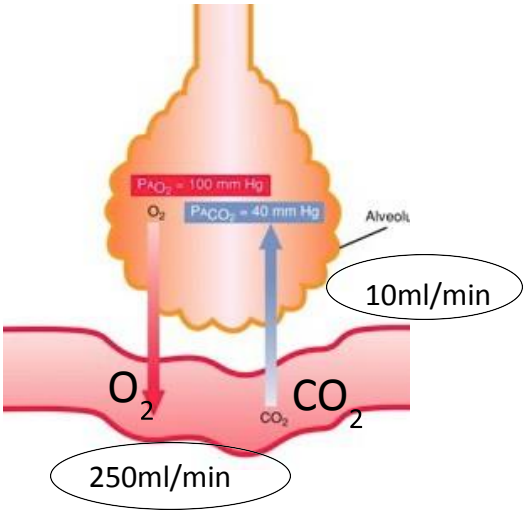

250ml/min O_2 out of lung

\- 10ml/min CO_2 into lung

= 240ml/min of gas removed from lung, which creates a vacuum effect, causing gas to be pulled into lungs

Effective apneic oxygenation requires:

- Oxygen source: to provide a continuous supply of oxygen to fill the hypopharynx. This is commonly described using a nasal cannula.

- Patent Airway: to permit the oxygen provided by the nasal oxygen source to move from the nasopharynx into the hypopharynx, allowing it to be entrained into the lungs in response to the negative pressure developed during apnea. The SuperNO_2VA and its ability to stent open the airway helps achieve this. A nasopharyngeal airway may also be useful to assist nasal oxygen passing from the nasopharynx into the hypopharynx.

Apneic Ventilation

Apneic ventilation takes this principle one step further by enabling the removal of carbon dioxide during apnea. Apneic oxygenation passively entrains oxygen into the lungs but does not allow for the escape of carbon dioxide from the lungs. This new term, apneic ventilation, refers to the delivery of high-flow oxygen directly into the trachea. By using higher flows of oxygen, the velocity of the delivered gas is high and the pattern of flow becomes turbulent rather than laminar. The high flow of nasal oxygen is not only able to forcefully enter the trachea on its own (rather than secondary to passive entrainment), but the turbulent flow allows for continuous flushing of the gas that is in the lungs, effectively removing carbon dioxide (see image).

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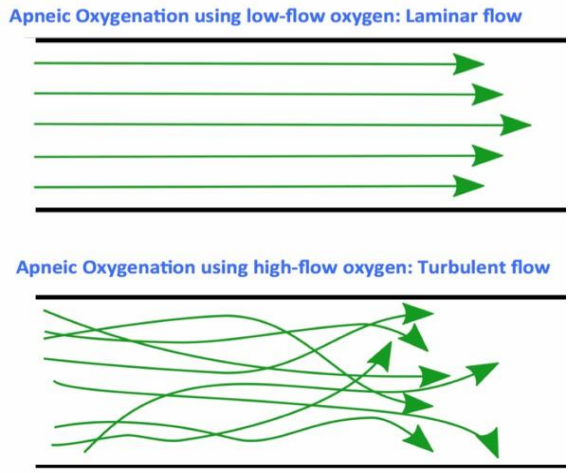

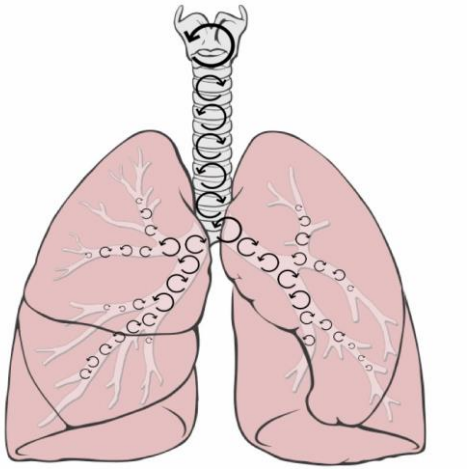

For apneic ventilation to succeed, the nasal oxygen delivery device must be able to provide very high flows of oxygen (> 15 L/min) to the oropharynx and hypopharynx. This process was described using a high flow nasal cannula (OptiFlow THRIVE, reference: Patel). It is important to note that there are three clinical scenarios that limit the efficacy of apneic ventilation (THRIVE) and should be understood:

1. **BMI > 30kg/m^2:** THRIVE studies have demonstrated only marginal benefits using THRIVE in obese patients. This is likely secondary to the limited FRC (reserve) in this population in addition to the increased oxygen consumption associated with obesity. The amount of oxygen delivered via THRIVE is not inadequate to account for the oxygen consumption. In addition, the lack of a seal around the nose allows only minimal positive pressure to be generated (ie: < 5 cmH_2O), which is insufficient to prevent or treat atelectasis in the obese patient due to increased abdominal tissue resulting in compression atelectasis.
2. **Upper airway obstruction:** If an UAO exists, the oxygen delivered via the nose will not reach the hypopharynx. UAO typically requires >7 cmH_2O in order to stent open the obstruction, which high flow nasal cannula does not achieve.
3. **Oxygen desaturation:** Despite high flow nasal cannula oxygenation, if a patient begins to experience oxygen desaturation, rescue ventilation is required. High flow nasal cannula, due to the lack of proper seal and positive pressure generation, is incapable of rescue ventilation and must be aborted to allow for a device that can be used for mask ventilation.

Prior to the SuperNO_2VA™ device, the only devices capable of high flow nasal oxygen delivery were high flow nasal cannula, nasal CPAP, and the use of the pediatric facemask configuration (NOVA,
Tse-PAP). Both HFNC and CPAP require capital equipment not readily available in procedural areas. The NOVA technique and Tse-PAP with a pediatric anesthesia mask placed over the nose can achieve high flow, but its cumbersome set up and hindrance of view during laryngoscopy have severely limited its adoption.

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Features and benefits of the SuperNO$_2$VA Platform

Clinical benefits of the SuperNO$_2$VA™ device

The SuperNO$_2$VA™ device is a nasal anesthesia mask designed to maintain upper airway patency and support ventilation in patients who are sedated with anesthetic medications. It was designed to utilize all of the benefits of nasal ventilation both inside and outside the OR. When placed over the patient's nose it creates a complete seal and when combined with an oxygen source and pressure relief valve, a positive airway pressure is generated allowing a clinician to maintain airway patency and provide ventilatory support. The SuperNO$_2$VA™ device has the following clinical benefits:

1. Delivers oxygen with positive airway pressure so can be used for pre-oxygenation
2. Reduces anatomical dead space, which improves effective alveolar ventilation
3. Creates a pneumatic stent in the posterior pharynx, which relieves upper airway obstruction
4. Provides access to the oral cavity while still allowing for rescue ventilation in apneic patients
5. Able to deliver >30 L/min during open airway procedures
6. Connects to standard anesthesia equipment readily available in procedural areas

Features of the SuperNO$_2$VA™ device

Nasal chamber

The nasal chamber has five unique features, which help it differentiate it from all other products on the market:

- A nasal bridge membrane
- The location of the anesthesia port
- A supplemental oxygen port
- Duckbill valve attachment sites
- Velcro strap anchor

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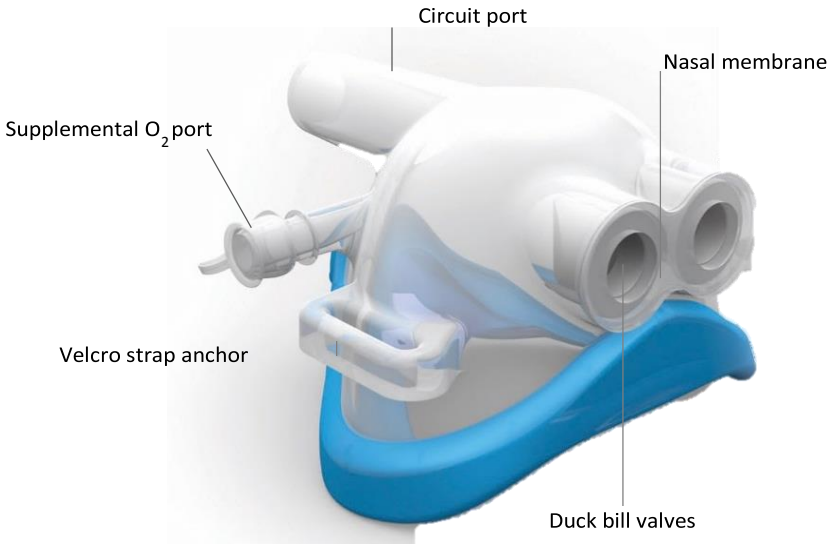

Nasal bridge membrane
The nasal bridge membrane is specifically designed to help maximize the SuperNO_2VA's seal for all demographics. It is elastic enough to adjust for different size noses (ie: nasal bridges), yet tight enough to prevent leakage of gas around it.

Circuit port
The circuit port is a standard 15mm OD port that allows connection to all standard anesthesia equipment, including: anesthesia circuit, hyperinflation bag, and bag-valve-mask resuscitator. It is located above the nasal bridge and is directed cephalad (toward the head) instead of anterior (out of the mask). Its location directs the circuit out of the way of the oral cavity, allowing for an unobstructed access and view of the mouth and airway during laryngoscopy.

The supplemental oxygen port
It is located to the right of the anesthesia port and has two essential functions. First, it can provide additional oxygen flow by connecting to a second oxygen source, allowing for high flow oxygen delivery. This is important during periods of apnea as it helps create a high flow oxygen system allowing for apneic ventilation. Second, it can be used as a supplemental oxygen mask for transport and in recovery.

Velcro head strap
One end of the Velcro strap comes pre-attached on the right side of the nasal chamber, while the other end comes detached but can quickly attach to the SuperNO_2VA^{TM}. The Velcro strap was designed to have both rigidity and flexibility, which allows the ability to create either a tight seal for non-invasive positive pressure ventilation, or a loose fit for patient transport outside of the operating room. There are also multiple attachment sites on the head strap to allow for easy tightening and loosening.

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Oral chamber accessory (discontinued)

The original SuperNO_2VA™ device came with an oral chamber accessory, which connected to the nasal chamber via the two (2) duckbill valves. Upon connection, the one-way valves are stented open, allowing for bi-directional flow. This allows the SuperNO_2VA™ to be used for full facemask ventilation. However, the market feedback received since the commercial launch of the SuperNO_2VA™ device was that the oral chamber is not needed. As of July 2016, the oral chamber has been discontinued and the nasal only SuperNO_2VA™ device is currently being sold.

Clinical benefits of the SuperNO_2VA™ Satellite Set

The SuperNO_2VA™ satellite kit contains the nasal only SuperNO_2VA™ device along with a hyperinflation bag.

A hyperinflation bag is a semi-closed Mapleson circuit that connects to a supplemental O_2 source (ie: wall oxygen or external oxygen tank). As a Mapleson circuit, it contains oxygen tubing connected to a circuit which has a reservoir bag and adjustable pressure limiting (APL) valve. Its built in resistance mechanism is via an adjustable pressure limit (APL) valve, which when closed can generate positive pressure when coupled with high oxygen flows. The positive pressure that is generated can be used with the SuperNO_2VA™ device to achieve a nasal positive airway pressure, capable of maintaining upper airway patency and providing mask ventilation. The following are major benefits of the hyperinflation bag:

1. Delivers an FiO_2 of 100% which can be used for pre-oxygenation anywhere a supplemental oxygen source exists (e.g. all patient areas).
2. Provides a simple, effective, and completely disposable positive airway pressure solution where an anesthesia machine or ventilator is absent.
3. Improves workflow by relieving upper airway obstruction throughout the peri-operative period, as it can be used in multiple settings and in transport.
4. Can be used as a rescue ventilation device.
5. Can be used for apneic ventilation during difficult or rescue intubations.

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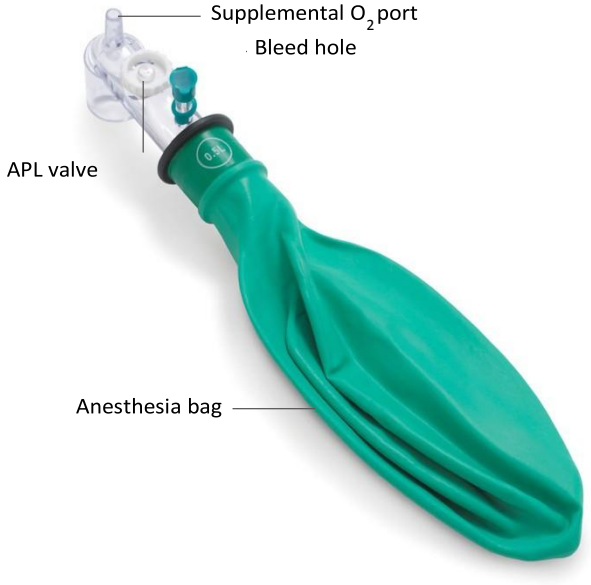
Features of the SuperNO$_2$VA™ Satellite Set

Hyperinflation bag

It consists of the following components:

1. Supplemental oxygen port
2. Anesthesia reservoir bag
3. Adjustable pressure limit (APL) valve
4. Bleed hole



Supplemental oxygen port

The supplemental oxygen port connects to one end of oxygen tubing and the opposite end of the tubing connects to a continuous source of supplemental oxygen.

Reservoir bag

The reservoir bag, as its name implies, allows for a reservoir of gas to be held. This is important because during inspiration, the actual flow of air may exceed that set by the oxygen source, thus allowing for uninhibited inhalation in the setting of a sealed nasal mask. Second, in the setting of manual ventilation, provides a volume of air/oxygen that the clinician may squeeze to drive into the lungs for assistance or rescue.

Adjustable pressure limit valve

The adjustable pressure limit valve allows the clinician to adjust the amount of pressure that is being delivered.

Bleed hole

The bleed hole is a pin-sized hole located between the oxygen port and the APL valve. Its function is to allow a small amount of gas to leak (ie: intentional leak) out of the hyperinflation bag, limiting maximal pressure to approximately 25cmH_2O. This is very important as it helps to prevent over-pressurization, which can cause barotrauma (ie: lung injury).

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Clinical benefits of the SuperNO$_2$VA™ Et (End-Tidal) Device *(AWAITING 510(k) APPROVAL)*

The SuperNO$_2$VA™ Et device is a nasal anesthesia mask designed to support ventilation in patients who are sedated with anesthetic medications and monitor ventilation via simultaneous sampling of end-tidal carbon dioxide ($EtCO_2$) from the nose and mouth. It was designed to utilize all of the benefits of the original SuperNO$_2$VA™ device, yet add the function of continuously monitoring $EtCO_2$. The SuperNO$_2$VA™ Et device will have the ability to provide nasal positive pressure ventilation, while simultaneously sample $EtCO_2$ from the nose and mouth.

Features of the SuperNO$_2$VA™ Et

The SuperNO$_2$VA™ Et device has the basic features of the SuperNO$_2$VA™ device (ie: circuit port location, supplemental O_2 port, Velcro head strap anchor, and the nasal membrane) along with 4 additional unique features, which include:

1. $EtCO_2$ Luer lock and tunneled chamber
2. Nasal $EtCO_2$ sample site
3. Oral $EtCO_2$ sample site
4. Oral hood

$EtCO_2$ Luer Lock and tunneled chamber

The $EtCO_2$ Luer lock is used to connect to an $EtCO_2$ sample line in order to measure $EtCO_2$. The unique tunneled chamber allows for sampling to occur immediately outside of the nares for best nasal sampling and simultaneous sampling from under the oral hood. It samples $EtCO_2$ from both the nose and mouth in order to measure $EtCO_2$ exhaled from either the nose or the mouth.

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Nasal $EtCO_2$ sample site

The nasal $EtCO_2$ sample site is a small hole located within the tunneled chamber located immediately adjacent to the nares. This hole allows for gas sampling from the nasal chamber into the tunneled chamber. From the tunneled chamber the exhaled CO_2 is then suctioned through the $EtCO_2$ sample line and measured via a capnography monitor.

Oral $EtCO_2$ sample site

The oral $EtCO_2$ sample site is a small hole located just outside of the nasal chamber, under the oral hood. This hole allows sampling of gas from the mouth to be brought into the tunneled chamber. From the tunneled chamber the exhaled CO_2 is then suctioned through the $EtCO_2$ sample line and measured via a capnography monitor.

Oral hood

The oral hood, made of the same TPE as the seal, hovers over the patient's mouth and collects exhaled gas from the mouth in order for it to be suctioned into the oral $EtCO_2$ sample site. It's TPE consistency is soft and malleable allowing it to retract over the nasal chamber during oral procedures, such as laryngoscopy, in order to not obstruct the oral cavity

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Chapter 4: Clinical Application of the SuperNO$_2$VA™

Basic Principles:

The SuperNO$_2$VA™ device, simply put, is designed to generate a nasal positive pressure, depending on the equipment used, that is capable of maintaining upper airway patency, deliver PEEP and ventilatory support, and provide rescue nasal ventilation. In order to generate a positive pressure, it is important to begin with this basic definition in Physics outlined by Ohm's Law (you don't need to remember Ohm, here, just the equation):

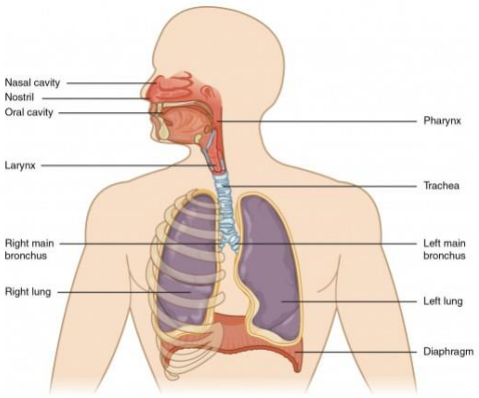
Pressure = Flow x Resistance

> *To dig deeper, the total pressure achieved within the* SuperNO$_2$VA™ *device is a product of the quantity of gas introduced to the system (via an anesthesia circuit and/or supplemental ports) and the resistance to gas escaping the mask and respiratory system.*

By knowing and understanding this principle, we can elucidate how to properly and effectively use the SuperNO$_2$VA™ device to achieve our desired goals. Let us define now the parameters that we will manipulate.

System/Container

The term *pressure* in this equation is referring to the measured pressure within a specific container or system. For the purposes of this discussion, the container/system we are henceforth referring to includes the SuperNO$_2$VA™ device, reservoir bag, and entire respiratory system, including the upper and lower airways.



Flow

Defined as "a steady stream of something," *flow* here refers to the introduction of external gas into the *system* defined above. Simply placing a SuperNO$_2$VA™ device on someone's face surely wouldn't generate a positive pressure, but introducing air or oxygen (via an anesthesia machine, ventilator, wall oxygen, etc.) to the SuperNO$_2$VA™ device once placed on the patient could. Henceforth, the *flow*, will be controlled and manipulated via the external device connected to the SuperNO$_2$VA™ device anesthesia machine or hyperinflation bag).

Resistance

Resistance is the measure of opposition to flow. Imagine a hose running water. The *container* is the hose itself and the *flow* is the quantity of water passing through the hose over time. The pressure within the hose is likely very low because water can freely escape the hose, unopposed. Now imagine closing off the tip of the hose with your finger. Essentially you are opposing the flow of water out of the hose by increasing the *resistance* to flow. The more you occlude the hose, the "stronger" the pressure within the hose becomes and is something I'm sure you can imagine feeling. This is how we achieve greater distances with hose water, by pressurizing the container and the water within it through the use of increased resistance. The *resistance* for us here will revolve around minimizing or eliminating air leaking from around the SuperNO_2VA™ device or from the mouth.

Returning to the project at hand, to create a positive pressure within the system (the SuperNO_2VA™ device, reservoir bag, and respiratory system), we must introduce a flow of gas (air or oxygen, with or without anesthetic gases), and we must maintain a high resistance (minimize gas leak from around the mask or from the mouth). These principles are vital to understanding how to operate the SuperNO_2VA™ device and in-service clinicians during sales calls. They will continue to come up throughout this chapter on SuperNO_2VA™ device application.

How to set up the SuperNO_2VA™ device utilizing an anesthesia machine

Introduction to the anesthesia machine

The anesthesia machine is a ventilator used for many different functions:

1. Deliver oxygen or air to a patient
2. Deliver anesthetic gases to a patient
3. Manually/Mechanically ventilate patients
4. Collect information and output data via monitors

There are two modes used for ventilation:

1. Manual mode: Using the anesthesia circuit, reservoir bag, and adjustable pressure-limiting (APL) valve (described below), the patient can breathe spontaneously with or without positive pressure and the clinician can deliver manual breaths via mask ventilation.
2. Mechanical/Ventilator mode: By circumventing the reservoir bag and APL valve, and using a system of bellows, the anesthesia machine can be set to deliver breaths to the patient mechanically, acting as a ventilator, with parameters set by the clinician.

The Anesthesia Machine-Circle System

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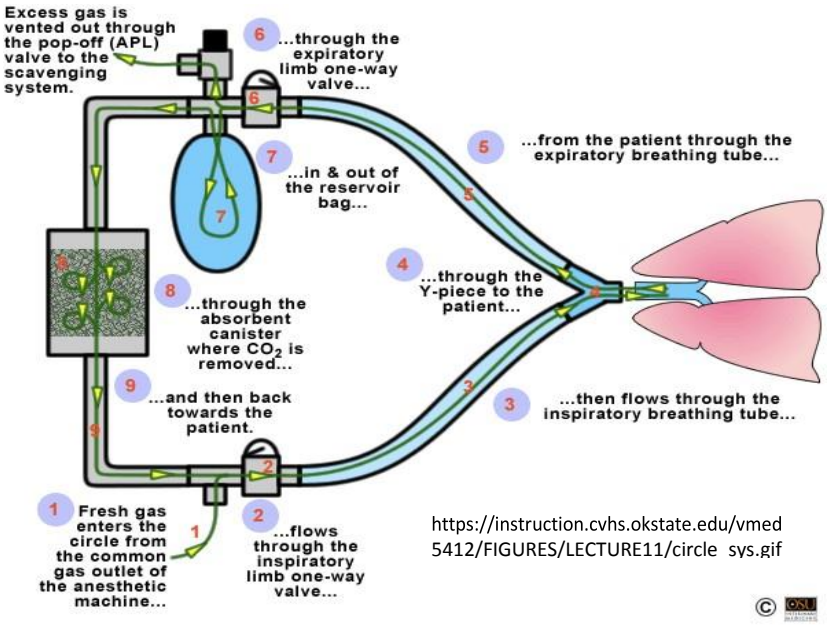
The flow of oxygen, air, and/or nitrous oxide (N_2O) is controlled by adjusting the individual flow meters on the machine (1). Total flow entering the system is a summation of all three of these flows. Flow will be measured in liters per minute (L/min).

Oxygen, air, and nitrous oxide is then sent to the patient via the *inspiratory limb* of the anesthesia circuit (2,3), which connects directly to the SuperNO_2VA™ device via the Y-piece (4). The anesthesia machine is designed to prevent a patient from re-breathing exhaled carbon dioxide, so there are one-way valves on the inspiratory limb (2) to prevent retrograde flow during exhalation. This forces the exhaled breath to travel down the expiratory limb (5) where it reaches a second one- way valve tha prevents the exhaled breath from returning to the patient during inhalation (6).

At this point everything exhaled enters the reservoir bag (7). Keep in mind that the flow of fresh gases delivered by the anesthesia machine is typically greater than that needed for the patient, there will always be overflow, which gathers in the reservoir bag. Attached to the reservoir bag is an adjustable pressure-limiting (APL) valve (aka "pop- off" valve), which can be set anywhere from 0-70 cmH_2O pressure.

When the pressure within the reservoir bag exceeds the pressure set by the APL valve, excess gas is vented to a scavenging system and removed from the circuit. Otherwise, gas will continue through the circuit through an absorbent canister where carbon dioxide is removed (8) and then back to the inspiratory limb towards the patient for re-use (9).



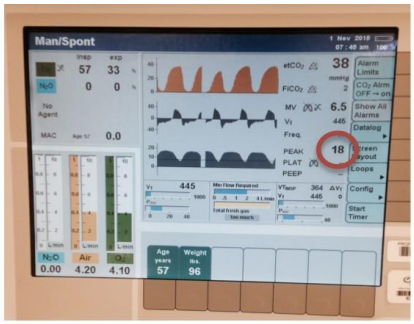
https://instruction.cvhs.okstate.edu/vmed5412/FIGURES/LECTURE11/circle_sys.gif

Providing oxygen to the patient

In order to provide oxygen to the patient, the clinician connects the y-piece of the anesthesia circuit to the SuperNO$_2$VA™ device. The circuit may be connected to the SuperNO2VA either prior to or after placement on the patient; however, ensure that there is sufficient oxygen flow present prior to connection. *Placement of the SuperNO$_2$VA™ and connection to the anesthesia circuit without fresh gas flow may cause difficulties in patient breathing and may cause asphyxiation or hypoxemia.* For supplemental oxygen only, turn on oxygen fresh gas flow and keep the APL valve set to zero (aka "open") which will provide zero resistance to patient exhalation.

Manual Ventilation

Manual mode refers to when the anesthesia machine is not set to deliver ventilator-driven breaths to the patient. The clinician can control the amount of pressure delivered to the patient by adjusting the fresh gas flow entering the circuit and adjusting the resistance to flow via the APL valve. By manipulating these settings, the clinician may provide supplemental oxygen to a patient breathing spontaneously, give positive pressure to alleviate upper airway obstruction and/or give ventilatory support to a patient breathing spontaneously, or deliver manual breaths to a patient who is apneic.



Positive Airway Pressure (PAP) and Continuous Positive Airway Pressure (CPAP)

(Note: The SuperNO$_2$VA is contraindicated for use with a CPAP device.)

The goal is to create a PAP within the SuperNO$_2$VA™ device which, when applied to the patient, will deliver the positive pressure to the airways to maintain upper airway patency and provide ventilatory support to the lower airway to improve gas exchange. Continuous PAP, or CPAP, refers to a PAP throughout the entire respiratory cycle, including inhalation and exhalation. The pressure in the circuit (and therefore the SuperNO$_2$VA™ device and airway) is set by the APL valve and can be monitored at any given time via the pressure waveform monitor on the anesthesia machine. Distention and pressurization of the reservoir bag is also an indication of PAP in the system and can be monitored by the clinician.

Setting up the SuperNO$_2$VA™ *for nasal PAP during deep sedation using the anesthesia machine*

1. Ensure the anesthesia machine is turned on and set to manual mode. Set high fresh gas flows on the anesthesia machine (> 10L/min). *Remember, the higher the flow, the greater the pressure that can be achieved (Pressure = **flow** x resistance), this can be adjusted later.*
2. Place SuperNO$_2$VA™ securely over the patient's nose. A tight seal around the nose will ensure that no air can escape around the SuperNO$_2$VA™, therefore minimizing leak

and maximizing resistance in the system. *The greater the resistance, the greater the pressure that can be achieved (Pressure = flow x **resistance**).*

3. Connect anesthesia circuit to the SuperNO$_2$VA™ device.
4. Set the APL valve to the desired pressure. The majority of patients require 6-10 cmH2O to maintain airway patency, however, keep in mind that some patients may require much more (e.g. morbidly obese, OSA). *This may need to be adjusted throughout the procedure.*
5. Adjust the SuperNO$_2$VA™ device, fresh gas flow, and APL valve as necessary to achieve desired airway pressures.

Monitoring and Troubleshooting

Reservoir bag and Pressure Waveform Monitoring During operation of the SuperNO$_2$VA™ device, if the system is under pressure, you should be able to appreciate distention of the reservoir bag, as the bag is a part of the circuit. The pressure within the reservoir bag is equivalent to the pressure in the SuperNO$_2$VA™ device and airway. During spontaneous breathing, the patient will breathe via negative pressure ventilation. The negative pressure will arise during inhalation and you should visualize intermittent depressurization and deflation of the reservoir bag. Upon completion of the inhaled breath, the reservoir bag should quickly refill and re-pressurize because it is refilled from the fresh gas flow from the machine in addition to the exhaled breath from the patient.

This same information can be gathered from the pressure waveform on the monitor, showing a positive pressure (above zero) during the expiratory phase of the respiratory cycle with intermittent depressurization during the inspiratory phase.

The presence of this intermittent depressurization/deflation in the system is *confirmatory* of successful patient inhalation. If the patient were to be apneic or if there was a complete upper airway obstruction, it is impossible to pull volume from the circuit and no negative deflection would be observed.

Oral leak

Given an adequately tight seal around the SuperNO$_2$VA™ device, the most common location for a leak in the system is the mouth. A completely open mouth, much like the open end of the water hose, allows unopposed escape of air flow from the mouth, making it difficult to pressurize the system and airway. By minimizing the air leak from the oral cavity, you can maximize resistance in the system and maximize pressure and efficiency. Techniques to minimize oral leak will be discussed in detail later.

Snoring

Snoring refers to the sound that is made when turbulent air flow is traveling through a partially obstructed upper airway. By its definition, it is a sign of *partial* upper airway obstruction. Complete upper airway obstruction would not cause a snoring sound because there would be zero flow through the airway secondary to complete obstruction (there would also be no deflation/depressurization of the system appreciated in the reservoir bag or pressure waveform). In the setting of continued snoring with the SuperNO_2VA™ device employed, the clinician should increase the amount of pressure within the system to better relieve the obstruction and maintain upper airway patency.

> If the pressure reading on the monitor equals that which is set on the APL valve, simply increase the setting on the APL valve to deliver higher pressures.
>
> If the pressure reading does not match what is set with the APL valve, there is likely a lack of adequate flow into the system or there is too large a leak from the system (and hence a lack of adequate resistance). Be sure to maximize flow and resistance by following the steps of the initial set up (high flows, tight seal, closed APL valve, minimized oral leak).

Adding ventilatory support (recruitment breaths)

Under anesthesia, patients tend to hypoventilate, secondary to both upper airway obstruction and from decreased input from the brainstem. To maintain normal levels of oxygen and carbon dioxide in the blood, particularly in higher risk patients, it is important to support the breathing efforts throughout the case. This is achieved by allowing for PAP/CPAP as described above. In addition, it is helpful to intermittently support one or more large tidal volume breaths in order to re-expand any lung segments that have atelectasis. This will improve all lung volumes and allow for improved gas exchange. With one hand on the reservoir bag, the clinician will appreciate the patient's breathing by feeling the intermittent depressurization and repressurization. During inhalation, by manually squeezing the reservoir bag, an increased tidal volume can be delivered to the patient, allowing for lung expansion and recruitment of atelectatic lung tissue. The clinician will perform this maneuver as he/she sees fit clinically.

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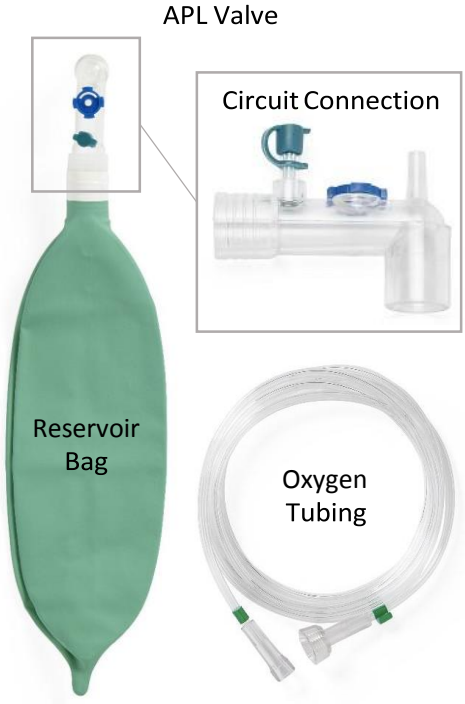
Rescue ventilation

(Note: The SuperNO₂VA is contraindicated for the treatment of sleep apnea)

The SuperNO₂VA™ device is a device designed for mask ventilation and as such, is safe to use for ventilation of patients who are apneic. It is common to have periods of apnea when using a deep sedation technique, albeit undesirable. This is the main reason why non-anesthesia providers are not permitted to give deep sedation, for fear the provider is not trained sufficiently enough in rescue techniques. In these situations, the anesthesia provider must be comfortable rescuing the patient via mask ventilation. For rescue mask ventilation, a closed system must be achieved that eliminates all leaks. A tight seal can be achieved around the SuperNO₂VA™ device by using the headstraps or using downward pressure on the SuperNO₂VA™ device itself to seal it to the face. The oral cavity must be occluded as well, by manually closing the mouth and using submental pressure when necessary to seal the tongue against the palate. With successful sealing of both the SuperNO₂VA™ device and the oral cavity, breaths can be given manually by squeezing the reservoir bag. With no leak, all volumes squeezed from the bag should be transmitted to the airway and lungs, then returned to the bag upon exhalation. PAP can be adjusted through the APL valve on the anesthesia machine.

How to set up the SuperNO₂VA™ device without an anesthesia machine

Proper use of the SuperNO₂VA™ device does not require an expensive ventilator or anesthesia machine. In fact, the parts of the anesthesia machine described above include a fresh gas source, a reservoir bag, an APL valve, and a connection to the SuperNO₂VA™ device. These can all be found on a standard hyperinflation bag. Overall, the use of the SuperNO₂VA™ device in this configuration is no different from use with an anesthesia machine, however it doesn't have pressure waveform monitoring. Providing oxygen, PAP, recruitment maneuvers, and rescue ventilation are performed in the same manner as with the anesthesia machine.



Rebreathing CO2

One major difference between this set up and the anesthesia machine is the absence of an expiratory limb with CO2 absorbent. The anesthesia machine is designed with these so that the air used for ventilation can be re-used (with CO2 removed) in order to conserve anesthetic gases. With a hyperinflation bag, fresh gas and the exhaled breath are both gathered in the reservoir bag. If the flow of fresh gas is low, the patient is at risk for rebreathing their own de- oxygenated breath and rebreathing CO_2 which may lead to hypercarbia and hypoxemia. For these reasons, it is important that the gas within the reservoir bag is constantly being renewed. To achieve this, the manufacturers of many hyperinflation bags recommend setting the fresh gas flow to 1.5 to 3 times the patient's minute ventilation. At these higher flows, sufficient washout of the exhaled CO_2 is achieved and prevents re-breathing.

APL valve

The APL valve on the hyperinflation bag functions differently from the anesthesia machine's. On the anesthesia machine, the APL valve is a spring-loaded one-way valve that releases gas into the scavenging system when the pressure in the reservoir bag exceeds that set manually on the APL valve. For the hyperinflation bag, the "APL valve" is a simple dial that allows for leak from the system. By opening the valve to the atmosphere, it will vent excess gas from the reservoir bag. This can be adjusted by turning the dial, making the opening smaller or larger. The larger the opening, the less resistance permitted in the bag and the less pressure able to be delivered. With the patient awake, we recommend starting with the APL valve set to the "open" position to allow for easy ventilation without positive pressure. As anesthetic medications are given to the patient and deeper levels of sedation are achieved, closure of this valve will allow the clinician to titrate the total positive pressure in the system, and hence the airway and lungs. This can and should be changed as needed by the clinician and may need to be adjusted frequently throughout the procedure.

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SuperNO$_2$VA™ device application for different clinical scenarios

There are three broad categories in which the SuperNO$_2$VA™ device is designed to be used. These clinical categories serve as umbrella classifications under which nearly any clinical scenario can be placed into. Understanding how to best manage the SuperNO$_2$VA™ device for each of these three will allow you to master the teaching and training and tailor your in-service efforts to what the clinician would like to use it for. The three categories include: procedures not involving the oral cavity, intra-oral procedures without a sterile field, and open-airway procedures.

SuperNO$_2$VA™ device for procedures not involving the oral cavity

Most fall into this category and include all procedures that do not involve the oral cavity or involve a sterile field that prevents access to the face and neck. To describe it another way, any case where the patient's mouth can be closed and the anesthesia provider continues to have access to manipulating the mouth, head, and neck without disrupting the surgeon/proceduralist. All non-facial procedures are included here.

Keeping in mind that in order to generate and maintain a positive pressure within the SuperNO$_2$VA™ and airway, one must minimize any leak exiting from the mouth. In these cases, where the anesthesia provider can freely and easily access the head/neck of the patient, and given an adequately tight seal around the SuperNO$_2$VA™ device, there are three techniques that are helpful in minimizing oral leak. Remember, that complete resolution of the mouth leak is not always necessary, as long as the volume entering the system via the fresh gas flow is greater than the volume leaked from the mouth:

1. Head Position: although counter-intuitive at first, a neutral or slightly flexed head position is advantageous during nasal positive pressure generation. Upon flexing the head slightly, the tongue is forced into the hard palate, occluding the oral cavity and allowing for positive pressure to build behind the soft palate and oropharynx. We recommend only 10-15 degrees of flexion, as extreme flexion may drive the tongue posterior and occlude the oropharynx, causing complete upper airway obstruction.

2. Manual closure of the mouth: simple manual closure of the lips and mouth will minimize oral leak.

3. Sub-mental pressure: at times closure of the mouth is not enough to prevent pressurized air from exiting the lips. In these scenarios, forceful lip closure may prevent air leak from the mouth. Another technique is to occlude the oral cavity internally by manipulating the tongue to your advantage. With your fingers, feel for the soft tissue in the sub-mental space (underside of the jaw). This is the base of the tongue. Using firm pressure, drive the tongue upwards toward the top of the head (cephalad) into the hard palate. The tongue, now driven against the hard palate, will seal the oral cavity. When applying this technique, it is best to remain as anterior as possible with your fingers and the direction of the force. If the tongue is manually forced posterior, one could cause a complete upper airway obstruction by driving the tongue into the oropharynx. It is helpful to advise the clinician that the force with their fingers should be aimed at the backside of the top teeth so as to aim anteriorly, and sometimes helpful to use a claw-like finger grip to drive the tongue into the palate and teeth.

Although the goal is to provide nasal positive pressure with a hands-free technique, some patients may require continued or intermittent manual assistance to minimize oral leak and

maintain positive pressure throughout the procedure. The patients in our focus populations (morbidly obese, obstructive sleep apnea) tend to have obstructive pathologies, which occlude the oral cavity without these manual techniques, but this should never be promised and the above techniques should be described and demonstrated prior to starting the case.

SuperNO$_2$VA™ device for intra-oral procedures without a sterile field

This subset of procedures includes all cases where a surgeon/proceduralist is occupying the oral cavity, but where external manipulation of the mouth, neck, and head do not interfere with a sterile field. The most common procedures in this group are upper endoscopy and trans-esophageal echocardiography (TEE). During these cases, it is common to use a bite block to protect both the patient's teeth and the expensive scope equipment. The mouth, therefore, is fixed in the open position, which coupled with the scope traveling through the mouth and oropharynx, contributes to oral leak.

It should be immediately obvious that external closure of the mouth is not possible during these procedures, therefore manual closure of the mouth and lips should not be recommended here. If head/neck manipulation does not interfere with the surgeon/proceduralist, we recommend maximizing the most efficient head position which again is in the neutral or slightly flexed position (10-15 degrees). As above, this forces the tongue cephalad towards the hard palate, despite the presence of the scope in the mouth.

The most effective technique for these cases is with sub-mental pressure. Despite the bite block and presence of the gastroscope or echo probe, firm sub-mental pressure can be applied to seal the oral cavity internally, much the same as mentioned previously. Remember, the pressure is to be applied to the soft tissue of the sub-mental space and should be directed cephalad and anterior, so as to occlude the oral cavity but not drive the tongue posterior into the oropharynx.

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SuperNO$_2$VA™ device for open-airway procedures

One of the most challenging clinical situations for anesthesia providers is the maintenance of oxygenation and ventilation for anesthetized patients during completely open-airway procedures. An open-airway, as described here, refers to any procedure without an advanced airway device where a surgeon or proceduralist needs direct access to the oral cavity, and closure of that oral cavity, whether externally or internally, is impossible or would disrupt the procedure. The most common examples include bronchoscopy, laryngoscopy, and surgeries of the tongue, mouth, and pharynx/larynx. Because the closure of the oral cavity is impossible, one would expect the leak from the oral cavity to be substantial. Due to the massive oral leak and inability to "close the system," a positive pressure cannot be generated.

For these procedures, the goal for oxygenation and ventilation is to maximize the flow and velocity of the oxygen/air entering the oropharynx and larynx. In achieving this, we may therefore maximize the clinical benefits of apneic oxygenation and apneic ventilation.

- Apneic oxygenation describes the passive entrainment of oxygen into the lungs from the oropharynx during periods of apnea secondary to an unequal volume of oxygen being removed from the lungs in comparison to the smaller volume of CO_2 being deposited.
- Apneic ventilation describes the use of high flow jets of oxygen to drive turbulent oxygen flow into the trachea. Turbulent oxygen flow allows for active delivery of oxygen to the lungs (not passive) and washout of CO_2 (hence, "ventilation").

To accomplish this, utilizing equipment readily available in the OR and non-OR clinical settings, the SuperNO$_2$VA™ device was designed with a supplemental oxygen port built directly into the device. In addition to the 15 L/min maximal oxygen fresh gas flow from the anesthesia circuit, an additional 15+ L/min can be delivered by attaching standard oxygen tubing to the supplemental O_2 port connected to any auxiliary oxygen source (wall oxygen, oxygen tank, or auxiliary oxygen from the anesthesia machine). At this point 30+ L/min can be achieved, which is transmitted via the nasopharynx directed towards the glottis. This extra flow is considerably valuable in maintaining oxygenation and jet-like ventilation during spontaneous respiration and periods of apnea. There are two recommended techniques here:

1. Simple flow without assistance: simply dial the fresh gas flow of oxygen through both the circuit connection and supplemental oxygen port to their maximal settings, respectively. For the grand majority of regulators, the maximal flow rate is approximately 15 L/min each, totaling 30+ L/min of constant flow.
    a. Using the APL valve, set the pressure relief to 10-20 cmH_2O. In most cases, the open system will not allow pressurization of the SuperNO_2VA™ device, however, in the setting of an upper airway obstruction or closure of the mouth, a closed system may be achieved, either purposefully or unintentionally. In a closed system, high flows within the SuperNO_2VA™ device coupled with a closed APL valve may lead to high pressures being generated quickly and the potential for patient harm. By setting the APL valve to 10-20 cmH_2O or partially open on the hyperinflation bag, harmful high pressures won't occur above the limit set by the valve.
    b. On the contrary, you wouldn't want the APL valve completely open either because in the setting of upper airway obstruction, mild to moderate positive pressure is necessary and advantageous to relieve that obstruction and deliver the oxygen flows to the pharynx and larynx. This represents a common limitation to other apneic oxygenation devices (nasal cannula, high flow nasal cannula, etc.).
    c. The use of the "oxygen flush" on the anesthesia machine can increase this flow even further, and may be used at the discretion of the anesthesia provider.

2. Delivering manual or mechanical breaths. By setting the ventilator to deliver breaths, or by simply squeezing the reservoir bag during the procedure, you can essentially drive oxygen through the SuperNO_2VA™ device at incredibly high flows. These higher flows (albeit intermittent, not constant as above) may prove more successful at delivering oxygen to the trachea and lungs. This science has not been fully elucidated but may prove more effective.
    a. Using the same maximal fresh gas flows as above from both sources, a second provider can squeeze the reservoir bag to deliver breaths through the SuperNO_2VA™ device.
    b. Alternatively, the ventilator can be set to deliver breaths mechanically. We recommend setting a tidal volume of 10 mL/kg at a rate of 15-20 per minute with a maximal pressure of 20-30 cmH_2O. When using mechanical ventilation, keep in mind that the fresh gas flow may be inadequate to maintain a full reservoir bag and/or bellows. The oxygen flush may be necessary to refill the bag/bellows intermittently during the procedure.

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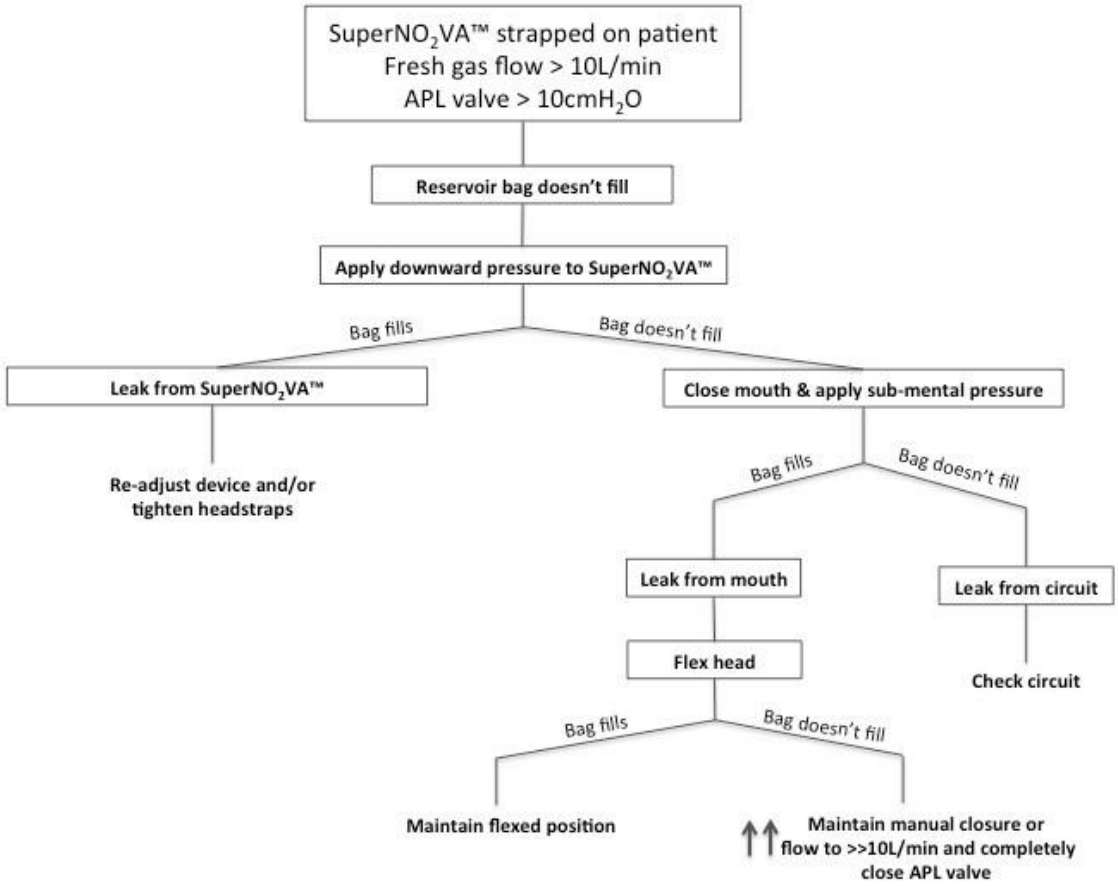
SuperNO2VA™ device troubleshooting intra-operatively

At this point in training, you should be able to understand how to troubleshoot using the SuperNO$_2$VA™ device intra-operatively for positive pressure generation. The following algorithm may prove helpful in navigating any difficulties you encounter.



Chapter 5: Most common objections and how to address them

Monitoring End-Tidal Carbon Dioxide

The SuperNO_2VA™ device does not have direct capnography monitoring capabilities, it is important to emphasize that there are other monitors that are useful to ensure successful ventilation. First, the reservoir bag should intermittently deflate or depressurize during inhalation as the patient pulls volume from the system into the lungs through negative pressure ventilation. The presence of intermittent depressurization is confirmatory of successful inhalation. Additionally, if there is continuous pressure waveform monitoring (as seen with all ventilators and anesthesia machines), you will appreciate intermittent depressurization on the monitor during successful inhalation. Once ventilation is confirmed, apply other useful techniques that maximize EtCO_2 capture.

Addressing Oral Leak

The objective is to first try and minimize leak out of the mouth. Given an adequately tight seal around the SuperNO_2VA™ device, the most common place for a leak in the system is via the mouth. There are several ways in which to minimize the leak from the mouth; keeping in mind that complete resolution of the mouth leak is not always necessary (as long as fresh gas flow volume is higher than the leak volume):

1. Head Position: Although counter-intuitive at first, a neutral or slightly flexed head position is advantageous during nasal positive pressure generation. When the head is slightly flexed, the tongue is forced into the hard palate, occluding the oral cavity and allowing for positive pressure to build behind the soft palate and oropharynx. We recommend only 10-15 degrees flexion, as extreme flexion may occlude the oropharynx and cause complete upper airway obstruction.
2. Manual Closure of the Mouth: Simple manual closure of the lips and mouth will minimize oral leak.
3. Sub-mental Pressure: In the event that simple lip closure results in continued leak from the mouth or if the external mouth cannot be closed (bite block in place, endoscope or probe in the mouth, etc.), the oral cavity can be occluded internally. With your fingers, feel for the soft tissue in the sub-mental space, the base of the tongue. Using firm pressure, drive the tongue cephalad and anterior towards the hard palate. The tongue, driven against the hard palate, will seal the oral cavity. This is useful despite presence of an endoscope, bronchoscope, or TEE probe because the tongue will seal around the scope and into the hard palate. Although the goal is to provide nasal positive pressure with a hands-free technique, some patients may require continued or intermittent manual assistance to minimize leak and maintain positive pressure throughout the procedure.

If it is impossible to prevent leak from the mouth it is important to note that the path of least resistance is not an all-or-none phenomenon, where all of the flow goes out of the mouth. The amount of flow going out of the mouth and into the lungs will be inversely proportional to the resistance. For example, if the diameter of the mouth opening is slightly larger than the diameter of the trachea (ie: there is less resistance in the mouth than the trachea), then there will be slightly more flow out of the mouth than into the trachea.

The way to compensate for the increase in flow out of the mouth is to add an additional 15L/min of flow by connecting the SuperNO_2VA™'s oxygen tubing to a supplemental oxygen source.

Risk of Gastric Insufflation

The SuperNO_2VA™ device is designed to provide positive airway pressure when used with equoment, so just like with other similar devices, a formal risk-benefit analysis should be performed for every patient prior to using any device capable of delivering positive pressure. The standard contra-indications to PPV/CPAP apply to the SuperNO_2VA™ device as well. When using the SuperNO_2VA™ device for sedation-related ventilatory support, most patients require only 5-10 cmH_2O to maintain airway patency and support ventilation. Pressures within this range are usually associated with minimal to mild gastric insufflation and in the setting of a patient who is NPO, poses a fairly small overall risk. Formal prospective studies evaluating gastric insufflation and aspiration risk, or PONV in sedation patients with use of positive pressure have not been performed.

Additionally, the SuperNO_2VA™ device uses a fixed-flow CPAP as opposed to variable flow CPAP. The risk of gastric insufflation is most present during inspiration, where airway pressures may exceed intra-gastric pressures, therefore overcoming the lower esophageal sphincter and driving air into the stomach. During exhalation, the intra-abdominal and intra-thoracic pressures are increased from chest and abdominal wall recoil, and gastric insufflation is unlikely because the intra-gastric pressures are high. With the SuperNO_2VA™ device (fixed flow CPAP), the airway pressure decreases during inspiration, the most dangerous time for gastric insufflation. This decreased airway pressure during this time may decrease the chances of gastric insufflation, although this needs formal study to prove. For conventional CPAP devices (variable flow), the depressurization caused by inspiration triggers the CPAP machine to deliver increased flows to maintain a constant level positive pressure. The positive airway pressure and added flows (at times >100 L/min) during the most likely time period for gastric insufflation is thought to be the major culprit.

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Using a Nasopharyngeal Airway (NPA) to Relieve Upper Airway Obstruction

Nasal pharyngeal airways relieve upper airway obstruction by providing a physical stent in the nasopharynx and behind the soft palate and tongue. However, the NPA addresses only one of the two major contributors to hypoxemia during sedation, upper airway obstruction, but does not address the second, central hypoventilation. This is why it is not uncommon to see a morbidly obese patient under sedation saturating 92% or worse despite the presence of a NPA and patent upper airway.

It's not just the upper airway obstruction that is the problem it's also the hypoventilation (ie: rapid shallow breathing) induced from sedation. Furthermore, hypoventilation may result in post-operative pulmonary complications such as Respiratory Compromise. The treatment for hypoventilation is not to just relieve upper airway obstruction but to also provide ventilatory support via positive lower airway pressure which helps improve tidal volume and provide PEEP to maintain alveolar patency and treat atelectasis. The SuperNO_2VA™ device is a mask that creates a seal when positioned over a patient's nose and mouth, or nose only, to direct anesthesia gas, air, and / or oxygen to the upper airway during the continuum of anesthesia care.

THRIVE and High Flow Nasal Cannula vs. SuperNO_2VA™

High flow nasal cannula provides continuous oxygenation at rates up to 70 L/min, which may result in low levels of continuous positive airway pressure (CPAP). However, because it cannot provide positive pressures >5 cmH_2O it is ineffective in the relief of upper airway obstruction or as a ventilation device to manually ventilate patients. With that said, there are three contraindications for high flow nasal cannula:

- Upper airway obstruction
- Obesity (BMI > 30)
- A patient that is desaturating

High flow nasal cannula is contraindicated for upper airway obstruction because it is a completely open system and therefore cannot generate high enough pressures required to stent open the airway. If an upper airway obstruction exists, no flow of oxygen will reach the oropharynx and larynx, and thus the device is considered obsolete.

For obese patients, positive airway pressures are important to counteract the increased intra-abdominal and intra-thoracic pressures present secondary to their added weight. In an open system, high flow nasal cannula cannot generate high enough pressures to overcome compression atelectasis caused from their abdominal contents shifting upwards.

Finally, it is contraindicated in patients that are desaturating because in the setting of hypoxemia, the treatment is to mask ventilate the patient via an anesthesia mask. High flow nasal cannula is not equipped to perform these rescue procedures and must therefore be aborted in the setting of arterial oxygen desaturation and replaced with an anesthesia mask.

The SuperNO_2VA™ device has the ability to provide continuous oxygenation at rates greater than 30 L/min as well as positive pressure ventilation up to pressures as high as 60 cmH_2O. It can provide >30 L/min of oxygen because it has two ports that each connect to an oxygen source, a supplemental oxygen port and an anesthesia circuit port. As a no-leak device, the SuperNO_2VA™ device can be used as an anesthesia mask and is suitable for mask ventilation when needed.

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Chapter 6: Sample Sales Pitches

Notes from Sales Roleplays

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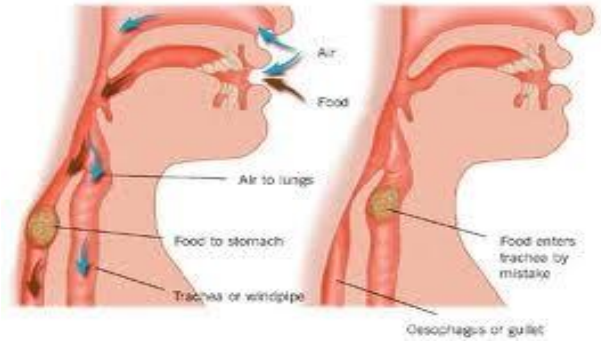
Appendix: Definitions

Apnea: temporary absence or cessation of breathing

Apnea time to desaturation: the length of time it takes for a patient's oxygen saturation level to fall below 90% from the time the patient stops breathing

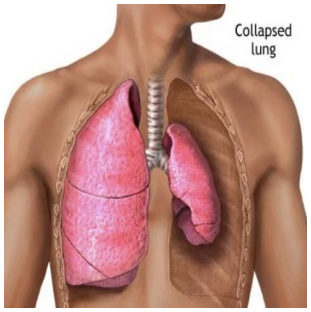
Aspiration: the drawing of a foreign substance, such as the gastric contents, into the respiratory tract during inhalation

Ex: The phrase "going down the wrong pipe", when a substance such as food enters the lungs



Atelectasis: the collapse or closure of lung units resulting in reduced or absent gas exchange

Ex: When obese patients lie flat on their back their abdominal contents push against the lower lungs causing alveoli in that region to close. A ramped position uses the force of gravity to enable the lungs to push against the abdominal contents and prevent them from closing.



Capnography: Capnography refers to the measurement and display of CO_2, including end-tidal CO_2 (ie: CO_2 that is expired), inspired CO_2, and the CO_2 waveform, which is referred to as the capnogram.

Capnometry: refers to the measurement and display of CO_2 in numeric form. The normal value for End-tidal CO_2 is between 35-45mmHg.

Continuous Positive Airway Pressure (CPAP): is a continuous pressure that is generated by connecting tubing to a continuous flow of gas along with an adjustable resistance. It is used to treat upper airway obstruction.

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(Pressure = Flow x Resistance)

Controlled ventilation: application of positive pressure into the airway to force gas into the lungs in the absence of spontaneous ventilatory efforts.

Ex: Bag-Mask Ventilation (BMV): the clinician places the mask over the patient's face and squeezes the bag to drive air into the lungs

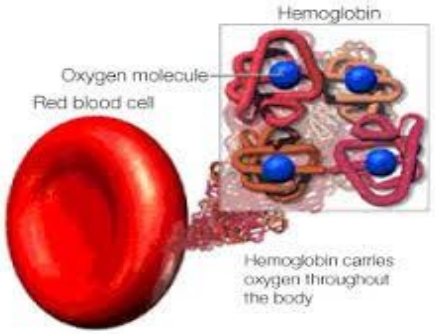
Dead space: is the volume of air which is inhaled that does not take part in the gas exchange, either because it (1) remains in the conducting airways, or (2) reaches alveoli that are not perfused or poorly perfused. In other words, not all the air in each breath is available for the exchange of oxygen and carbon dioxide.

Anatomical dead space is the volume of the conducting respiratory passages (150 ml). No gas exchange is possible in these spaces.

End-tidal CO_2 ($EtCO_2$): is the measurement of exhaled carbon dioxide, which is a sign that the patient is effectively breathing (ie: effective ventilation)

Functional residual capacity: represents the volume of air present in the lungs at the end of passive expiration.

Hemoglobin: a protein within red blood cells that binds to oxygen and carries oxygen to the tissues of the body.



Hypercapnia: is an increase in the amount of carbon dioxide within the blood, typically due to inefficient or inadequate breathing.

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Hyperinflation bag: A hyperinflation bag is a semi-closed oxygenation device that connects to a supplemental O_2 source (ie: wall oxygen) to utilize a continuous flow. It has a built in resistance mechanism via an adjustable pressure limit (APL) valve, which when closed allows for the generation of positive pressure.

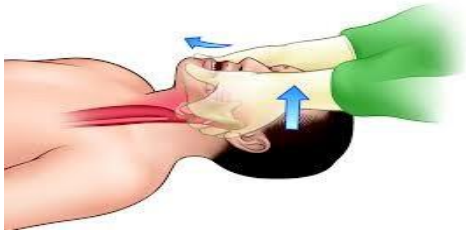
Hypoventilation: inadequate breathing or respiratory gas exchange to maintain a normal level of carbon dioxide in the blood, resulting in rising levels of carbon dioxide (hypercapnia).

Hypoxemia: an abnormally low level of oxygen in the blood

Intubation: the introduction of a tube into a hollow organ (as the trachea or intestine) to keep it open or restore its patency if obstructed

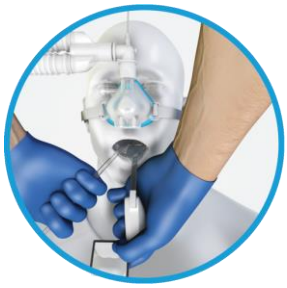
Jaw thrust: the provider uses their fingers to push the angle of the mandible upwards, while their thumbs push down on the chin to open the mouth

Ex: The anesthesiologist uses a jaw thrust to displace the mandible forward, causing the tongue to be pulled with it and preventing the tongue from occluding the entrance to the trachea.



Laryngoscopy: a medical procedure that is used to obtain a view of the vocal folds

Ex: a clinician will perform to laryngoscopy to facilitate the placement of a breathing tube



Minute ventilation (MV): is the amount of air moved into and out of the lungs per minute during ventilation. In order to calculate minute ventilation, simply multiply the volume of air in each respiratory cycle (tidal volume) by the number of breaths per minute (respiratory rate).

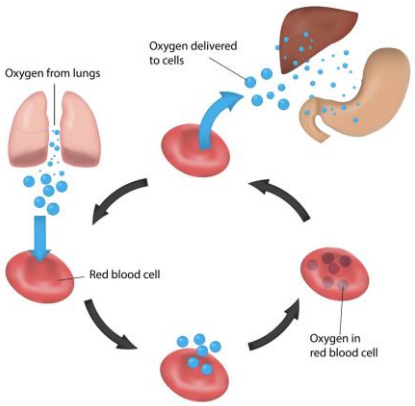
MV = Tidal Volume x Respiratory Rate

Non-invasive ventilation (NIV): refers to positive pressure ventilation that includes continuous positive airway pressure (CPAP) and controlled ventilation via a mechanical ventilator.

Oxygen desaturation: an abnormally low amount of oxygen in the blood, causing the fraction of oxygen bound to hemoglobin (SpO2) to be < 90%.

Oxygen Saturation: the fraction of hemoglobin in blood that is saturated with oxygen; Normal saturation (SpO2) is 95%-100%.

Oxygenation: is the process of how oxygen is taken into the lungs, passively diffused into the blood, and distributed throughout the body for the body to use.



Pressure: is the product of flow and resistance, where flow refers to the introduction of external gas into a container (ie: chest cavity) and resistance refers to the measure of opposition to flow.

Pressure = Flow x Resistance

Respiratory Compromise (RC): a spectrum of respiratory complications, such as upper airway obstruction, hypoventilation, oxygen desaturation, respiratory failure, and death, due to disease or sedation drugs.

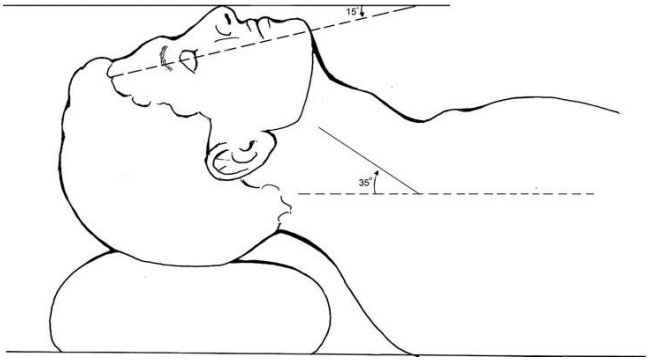
Respiratory failure: when the respiratory system fails to maintain either adequate oxygenation and/or ventilation resulting in severe hypoxemia necessitating mechanical ventilation in order to sustain life.

Respiratory rate: is the amount of breaths per minute (bpm). A normal respiratory rate is between 8-12 breaths per minute.

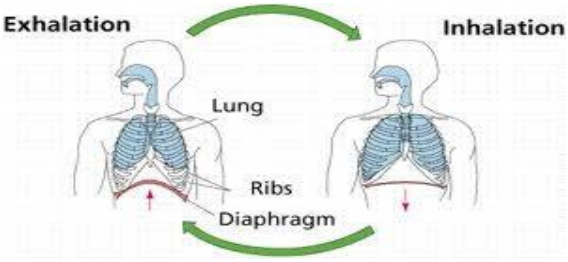
Sniffing position: traditionally been considered the optimal head position (35° neck flexion and 15° head

extension) for ventilation and direct laryngoscopy (DL). Also achieved by aligning the ear at the level of the sternum.

Ex: A clinician will place the patient in the sniffing position to help open up the patient's airway during controlled ventilation



Spontaneous ventilation: normal breathing in the awake state where the patient generates a negative pressure using muscular movements that moves air into and out of the lungs. Spontaneous ventilation may be assisted or unassisted



Tidal volume: the normal volume of air displaced between normal inhalation and exhalation. In a healthy, young human adult, tidal volume is approximately 500 mL per inspiration or 7 mL/kg of body mass.

Upper airway obstruction: a blockage of the upper airway, which can be in the trachea, larynx or pharynx.

Ex: Sedation drugs cause relaxation of the tongue, which results in it falling back into the airway causing upper airway obstruction, preventing spontaneous ventilation.

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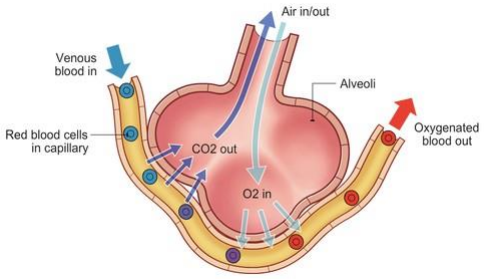
Ventilation: Ventilation is the removal of carbon dioxide from the blood, into the lungs, and out into the atmosphere. The ultimate goal for ventilation is to remove CO2 from the body.



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