AIRWAY MANAGEMENT:
HYE ABOUT VENTILATION AND THE ANETHETIZED PATIENT
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“Life is not measured by the number of breaths we take but by the moments that take our breath away.”
Author unknown

An anesthetic event can be divided into the following components: premedication, **induction**, maintenance, and recovery. Each component is equally important and requires extreme vigilance by the anesthetist.

Most of the drugs used (anesthetics/analgesics) throughout an anesthetic event are respiratory depressants. Until we obtain the patient’s airway (intubation) we really have no control over our patient’s ventilatory status and well-being. The patient is at risk of becoming hypoxic (inadequate O2 at the tissue level); eventually leading to other possible complications – aspiration pneumonia, decreased cardiac output (CO), decreased perfusion, and potentially cardiac arrest.

Induction of an anesthetic event is defined as: drug administration to change the state of the patient from **AWAKE** to **ANESTHETIZED** and eventually securing the patient’s airway by placing a flexible rubber (endotracheal) down the trachea. Endotracheal intubation allows us to isolate the patient’s airway and support respiratory function during an anesthetic event.

Safe anesthetic management requires adequate support and function to the respiratory system. It is the responsibility of the anesthetist to assure appropriate airway management during an anesthetic event. The anesthetist should be familiar with respiratory terminology as well as the equipment available for the patient.

**Endotracheal Intubation**

Endotracheal intubation is the placement of an endotracheal (ET) tube positioned between the vocal cords and down the trachea. The main goal for ET placement is to protect the lungs from aspiration of any material that may be in the oral cavity. Other benefits include: delivery of oxygen (O2), eliminate carbon dioxide (CO2), administration of inhalant, provide positive ventilation (PPV) and minimize atelectasis. Prior to induction the anesthetist should prepare by having a few different size cuffed tubes available (one that is ideal fit, one smaller, one larger). The ET tube chosen should be a “snug fit” to reduce airway resistance and prevent over-inflation of the cuff. When inflating the cuff it is important to insure a good seal to avoid a leak without over inflating. An over-inflated cuff will put too much pressure on the trachea causing potential ischemia and tracheal damage.
Appropriate tube length (nares to thoracic inlet) is also important to prevent dislodging during a procedure (too short) and minimize dead space (too long). Always be sure to clean tubes thoroughly and use lubricant during placement.

**Preventive Measures to Avoid Complications during Intubation**
Intubation can cause irritation and damage to the larynx or trachea. Choose an ET tube that fits best allowing for a smooth placement.
Always tie the ET tube in place to prevent dislodging and premature extubation.
Be sure to clean the tubes thoroughly after use to avoid cross contamination.
To avoid obstruction watch for kinks in the tube or clogging of the tube with lubricating jelly, salvia, mucous, or blood.
Use elbow (right angle) adaptors if angling is necessary. Caution here with patients less than 6 kg; these connectors can not only increase mechanical dead space but also disturb laminar gas flow causing an increase in work for patient to breathe.
Use wire reinforced tubes for procedures that involve the head/neck to be manipulated to avoid occlusion of the ET tube.
When moving the patient it is advisable to disconnect the ET from breathing circuit to avoid tugging and pulling of trachea.
To avoid endobronchial (one lung ventilation) intubation or increased dead space (increase in CO₂) pre-measure the tube to the thoracic inlet.
If a stylet is necessary to accomplish intubation be sure the stylet does not protrude beyond the ET tube (proximal to the patient) – this may traumatize the laryngeal tissue. Visualization is “the best” way to confirm successful intubation.

**Airway Management – Terminology and Equipment**
In order for the anesthetist to “do right” by the anesthetized patient – specifically respiratory function – it is imperative he/she has a solid understanding of respiratory terminology and the equipment available for use in monitoring the patient’s respiratory function.

Dead space (DS) is defined as the volume of air that is inhaled that does not take part in gas exchange. It is referring to the portions of the respiratory tract that are ventilated but not perfused by pulmonary circulation. There are two different ways to define dead space – anatomic and physiologic.
Anatomic dead space is the total volume of the conducting airways from the nose or mouth down to the level of the terminal bronchioles; it includes the nose, mouth, nasopharynx, larynx, trachea, bronchus, bronchioles, and terminal bronchioles.
Physiologic dead space is the sum of anatomic dead space and alveolar dead space. Physiologic dead space gases do not participate in oxygen (O₂) and carbon dioxide (CO₂) exchange.
Alveolar dead space refers to the space in the alveolar occupied by air that does not participate in O₂-CO₂ exchange. Alveolar dead space is negligible in healthy individuals but can increase dramatically in patients with pulmonary disease due to ventilation-perfusion mismatch.
**Mechanical dead space** is related to the equipment. It is the dead space in the breathing circuit where both inhalation and exhalation passes through a common path. The portion of the ET tube that extends out of the trachea beyond the incisors to the breathing circuit, any added adaptors (CO2 adaptors, apnea alarm adaptors, etc) or elbow (right angle) connectors all make up mechanical dead space. Exhausted soda lime or malfunctioning unidirectional valves can also contribute to added mechanical dead space.

The anesthetist should be conscientious and attempt to keep mechanical dead space to a minimum. Excessive mechanical dead space can be detrimental and potentially fatal for the patient. As dead space volume increases (end-tidal CO2), effective alveolar ventilation decreases. Arterial CO2 levels increase. Increases in arterial CO2 can lead to: respiratory acidosis, sympathetic stimulation, cardiac arrhythmias (combo of sympathetic stimulation and hypoxemic effects), peripheral vasoconstriction (followed by peripheral vasodilation), central nervous system (CNS) effect and eventually narcosis, increased cerebral blood flow and intracranial pressure, tachypnea and increased work of breathing.

**Alveolar Ventilation** (VA) is the volume of air breathed in per minute that reaches the alveoli; the respiratory portions of the lungs where gas exchange occurs.  

\[ VA = (TV - DS) \times RR \]

**Tidal Volume** (TV) is the amount of air that moves in and out of the lungs in a single breath. (10-20mls/kg)

**Respiratory Minute Volume** (RMV) is the amount of air that moves in and out of the lungs in one minute.  

\[ TV \times RR = RMV \]

**Positive Pressure Ventilation** (PPV) is controlled mechanical ventilation. It is the process by which air is pushed into the airway via an ET tube; a delivery of oxygen and inhalant to the patient’s lungs and elimination of CO2. This can be achieved either by “bagging” (applying pressure to rebreathing bag) the patient or by use of a mechanical ventilator. Because the patient’s own ventilation efforts may be inadequate when anesthetized, it is the responsibility of the anesthetist to ensure the patient is ventilating appropriately.

**Hypoventilation** (also known as respiratory depression) (CO2 > 45mmHg) is inadequate ventilation to perform essential gas exchange. It is insufficient elimination of CO2 from the body and a reduction of oxygen delivery to the tissues. The concentration of CO2 in the blood stream rises in the circulating blood and produces a state known as hypercapnia (or hypercarbia). This is a common concern in the anesthetized patient as a result of insufficient (spontaneous) ventilation.
Causes of hypoventilation may include: anesthetic depth (too deep), excessive mechanical dead space, endobronchial intubation, pain, pulmonary disease (contusions, effusion, edema, pneumonia, etc) abdominal distention, diaphragmatic hernia, hypothermia, neurologic disease (myasthenia gravis), and surgical interference (surgeon, instruments resting on thorax).

Management options for hypoventilation: determine the cause (depth, pain, mechanical dead space, etc) in order to rectify the situation. Institute controlled ventilation (PPV) for the patient.

**Hyperventilation** (over breathing) \((\text{CO}_2 < 35\text{mmHg})\) is an excessive rate and depth of breathing resulting in decreased \(\text{CO}_2\) levels and increased \(\text{O}_2\) levels. Hyperventilation is an increase in alveolar ventilation. The concentration of \(\text{CO}_2\) in the blood stream falls and produces a state known as hypocapnia (or hypocarbia). Causes of hyperventilation may include: overzealous manual ventilation, anesthetic depth (too light), pain, inadequate \(\text{O}_2\) flow rate, hypoxia (a deficiency of \(\text{O}_2\) reaching the tissues), hypotension, hyperthermia (panting), upper airway disease/obstruction.

Management options for hyperventilation: determine the cause (depth, pain, \(\text{O}_2\) flows, PPV, etc) in order to rectify the situation. Use caution when ventilating for patients suspected of pulmonary trauma.

**Hypoxia vs Hypoxemia**
These terms have been used interchangeably.

**Hypoxia** – a less specific term - is the reduction of \(\text{O}_2\) supply at the tissue level; \(\text{O}_2\) in the lung, blood, and/or tissues is abnormally low (not measured by a laboratory value). Basically the patient is not getting enough oxygen.

\(\text{SaO}_2\) (hemoglobin saturation) < 95%

**Hypoxemia** refers specifically to low \(\text{O}_2\) content in the blood to meet metabolic requirement. It can be a result of reduced \(\text{PaO}_2\) (the partial pressure of oxygen dissolved in the blood), reduced \(\text{SaO}_2\), or low hemoglobin content. The patient does not have enough oxygen in the blood regardless of the etiology.

\(\text{PaO}_2 < 60\text{mmHg}; \text{SaO}_2 < 90\%\)

Causes of hypoxemia may include: low inspired \(\text{O}_2\) concentration (anesthetic machine malfunction), hypoventilation (CNS depression, mechanical impairment i.e. airway obstruction), diffusion impairment (interstitial pneumonia), ventilation/perfusion mismatch (V/Q mismatch) (poor perfusion, poor ventilation)

Management options for hypoxemia: pre-oxygenate prior to induction, proper ET placement, appropriate \(\text{O}_2\) flow rates (circle system vs. non-rebreathing system), PPV/IPPV in addition to an occasional “sigh” (an intermittent hyperinflation during PPV; a deep breath) for the patient (increase surface alveoli surface area), maintain adequate
hydration status (use of crystalloids, colloids, blood products), use of bronchodilators (terbutaline).

**Ventilation/perfusion mismatch**
Simply stated, when ventilation and blood flow (perfusion) are mismatched at the level of the alveoli. This results in inefficient gas exchange between the pulmonary blood and the lungs.
When the ratio between ventilation (V) and perfusion (Q) is equal to 1.0 maximum gas exchange occurs.
If the V/Q ratio is < 1.0 perfusion is occurring but ventilation is not i.e. atelectasis, endobronchial intubation; blood does not become fully oxygenated as it passes through the lungs.
If the V/Q ratio is > 1.0 ventilation is occurring but perfusion is not i.e. severe hypovolemia, hypotension, and dead space ventilation.
This is the most common cause of hypoxemia in the perianesthetic period.

**Atelectasis** is defined as the collapse of part or all of the lungs. This can happen when tidal volume (TV) is reduced and the alveoli (tiny air sacs) do not expand fully on inspiration; O$_2$ and CO$_2$ cannot be exchanged. Atelectasis can be a result of a patient in lateral or dorsal recumbency for a period of time where the alveoli are compressed by the weight of the mediastinal structures and/or overlying lung tissue, or the weight of abdominal contents pushing on the diaphragm. Patients with abdominal distention (ascites, GdV, dystocia) or large abdominal masses may be prone to atelectasis.
Management options for atelectasis: confirm proper ET tube placement, perform PPV/IPPV (may need to increase peak airway pressure), minimize recumbency time, and “sigh” for the patient occasionally (every 5-10 minutes) throughout the anesthetic event. Use caution when ventilating patients with long-term atelectasis (i.e. diaphragmatic hernia) to avoid re-expansion pulmonary edema; high inspiratory pressure can cause collapsed alveoli to rupture and “weep” and leak out into the bronchioles leading to edema.

**Respiratory Monitoring**
Normal breathing occurs as the brain responds to an increase in CO$_2$ in the blood. Normal blood (arterial) CO$_2$ is 35-45mmHg.
Anesthesia depresses the brain’s response to CO$_2$. It resets the level at which a breath will be taken, allowing the CO$_2$ to build up higher with increasing depth of anesthesia.
Assisted ventilation for the patient will bring CO$_2$ levels back down to normal or possibly below normal (if too much PPV).
It is the responsibility of the anesthetist to stay “connected” to the patient’s respiratory function. Successful intubation and assisted ventilation (PPV/IPPV) is the first step in maintaining a stable patient. During the maintenance period monitoring the patient physically and mechanically will help direct the anesthetist to make the appropriate adjustments in supporting the patient’s respiratory function.
Physical monitoring by the anesthetist involves the use of one’s senses (sight, sound, and touch). If monitoring devices begin to malfunction the anesthetist can always rely on his/her own senses. The anesthetist should be well practiced of watching the patient’s chest (during inspiration) and rebreathing bag movement. When manually ventilating (PPV/IPPV) the patient the anesthetist should pay close attention to how the compliance (flexibility) feels and if there is an increased resistance when delivering a breath. When applying PPV there should be minimal effort noted; a decrease in compliance will require a greater pressure for adequate lung inflation i.e. edema, pneumonia, fibrosis. An increase in resistance (increase work to adequately ventilate for the patient) can be the result of either upper or lower airway issues i.e. ET tube kinked, accumulation of secretions.

Incorporating the use of an esophageal stethoscope into your everyday use assures the anesthetist of both respiratory and cardiac function of the patient. This is a very valuable, inexpensive piece of equipment that can aide is saving a patient’s life in the event of other equipment failure.

Mechanical monitoring by the anesthetist involves the use of equipment that will aide in assessing the patient’s ventilation. A combination of devices, pulse oximetry (SpO₂) and capnometry (CO₂) can enhance anesthetic management (respiratory) in the patient.

Pulse oximetry (SpO₂ normal values >95%) involves the use of a device that measures the patients pulse rate and O₂ saturation (indirectly) of the blood; it provides information about both the respiratory and cardiovascular systems. The pulse oximeter uses red and infrared light to measure O₂ hemoglobin saturation. This monitor relies on the patient’s cardiac output (CO) to generate adequate signal. Hypotension, vasoconstriction, movement of the patient (shivering), improper probe placement, hypoxemia can all result in low readings.

This piece of equipment is reasonably inexpensive, portable, noninvasive, and easy to use. Because pulse oximetry just measures hemoglobin saturation it is not a complete measure of respiratory sufficiency.

Capnometry (normal arterial CO₂ = 35 – 45 mmHg) is a non-invasive monitoring technique that gives the anesthetist insight regarding ventilation in addition to circulation and metabolism. A capnometer is a true respiratory monitor. It is a valuable tool used to aide the anesthetist in recognition apnea (no breathing), hypoventilation/hyperventilation, ET tube obstruction, equipment malfunction. How well a patient is ventilating is dependant on the clearance of CO₂. A capnometer measures CO₂ in exhaled and inhaled gases and displays a waveform of the partial pressure of CO₂ throughout the breath cycle. End-tidal CO₂ (ET CO₂) monitoring is a non-invasive means of estimating the patient’s arterial CO₂ (PaO₂). The exact value of PaO₂ is obtained via blood gas analysis; this is not usually available in most practices due
to practicality/cost. A capnometer is also a very reliable tool in early detection of anesthetic complications such as cardiovascular collapse, cardiac arrest.

There are two types of capnometers. Mainstream capnometer – has a sensor that measures respired gases in the main stream of the anesthetic circuit. Sidestream capnometer – withdraws a continuous sample of respired gases from the side of the anesthetic circuit. Regardless of the type you choose for your practice make sure you purchase a capnometer that displays a waveform; valuable for troubleshooting equipment malfunction (dysfunctional uni-directional valves), anesthetic complications (airway obstruction) and patient safety (excessive dead space).

It is the opinion of the author that the capnometer is the most valuable tool an anesthetist can have in his/her repertoire of devices. Capnography can be a life-saving modality – changes in EtCO2 levels can be an early indicator of potential (fatal) complications (hypoventilation, airway obstruction, cardiac arrest, etc).

REFERENCES

Additional references available upon request