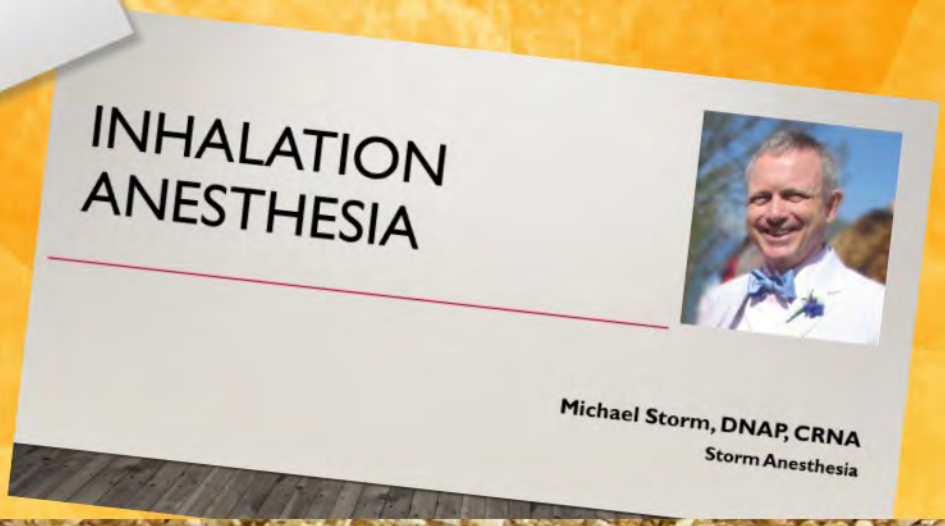
Anesthesia for Post Anesthesia Care Nurses

Video 2



Coleman, Harman, Koke, Sightler, Squires, Storm

STORM ANESTHESIA

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Introduction

Welcome to Storm Anesthesia's Anesthesia for Post Anesthesia Care nurses. This is a seven-video series on the basics on pharmacology and anesthesia techniques for the perianesthesia care nurse.

We, a group of five senior students from the University of South Carolina School of Medicine Nurse Anesthesia Program and one CRNA, have created this series in the hopes it will help the transition into the perianesthesia world. The series attempts to shine a bit of light on the techniques anesthesia uses during surgery, as well as explain the basics of the pharmacology behind our drug uses. This is by no means a series that will explain everything that happens during anesthesia, but our hope is that you, the perianesthesia nurse, will find our report a little less intimidating and a little more informative. After all, the better you understand the report, the better you can take care of the patient, and ultimately, this will increase the safety and satisfaction for both your patients and yourself.

The group consists of Alexandra Harman, BSN, RN; Braiden Sightler, BSN, RN; Jordan Coleman, BSN, RN, CCRN; Kelsey Squires, BSN, RN, CCRN; Victoria Koke, BSN, RN; and Michael Storm, DNAP, CRNA, CCRN.

The videos can be watched separately, but there are some references among the videos and the basics of the pharmacology along the way. Therefore, it may be beneficial to watch the series in order. Either way, have fun and don't forget to download the accompanying handouts. These handouts are the complete transcripts of the narrations and include all relevant pictures from the videos.

This video-series is sponsored by Storm Anesthesia and Palmetto Health Richland Anesthesia Department.

Enjoy and let's get started.

Michael Storm, DNAP, CRNA, CCRN
Editor
November 2017

Inhalation Anesthesia

Michael Storm, DNAP, CRNA, CCRN
Storm Anesthesia

This lecture is the second in the seven-lecture series for PACU nurses.

This second lecture is presented by Michael Storm, DNAP, CRNA and will cover inhalation anesthesia.

Objectives

The objectives of this lecture are to

- review keywords and definitions
- review basic concepts of anesthesia
- review pharmacokinetics and pharmacodynamics of inhaled anesthetics
- review potency of inhaled anesthetics
- review volatile agents, also known as inhaled anesthetic gases
- review effects of volatile anesthetics in PACU

At the end of the lecture you should have an enhanced understanding of

- goals of anesthesia
- what is Monitored Anesthesia Care (MAC) versus General Anesthesia (GA)
- stages of anesthesia
- induction and emergence
- volatile anesthetic agents
- important key points for the PACU nurse

Keywords and Definitions

Adjustable Pressure Relief (APR) Valve: Used for release of excessive gas on the circle system of an anesthesia machine.

Amnesia: A component of anesthesia in which the patient is unable to recall the events that occurred during the administration of the inhalational anesthetic.

Analgesia: A component of anesthesia in which the patient is unable to experience pain.

CO_2 Absorption Canisters: Located in a circle system on an anesthesia machine that clears rebreathed gas containing carbon dioxide by passing through a canister holding a chemical carbon dioxide absorbent

Delirium: An acute onset of variable and fluctuating changes in level of consciousness accompanied by a range of other mental symptoms.

Diffusion Hypoxia: Refers to the rapid exit of nitrous oxide and thus partial reduction of the percentage of oxygen that can be inhaled during the immediate emergence phase of recovery from anesthesia. Supplemental oxygen is recommended the first 5-10 minutes after emergence to avoid this phenomenon.

Effective Dose (ED): The dose of a drug necessary to produce a certain effect in a certain percentage of patients. For example, the ED50 is the term for when a drug produces a particular effect in 50% of patients.

Hypnosis: A component of anesthesia in which the patient becomes unconscious.

Inhalation Anesthesia: Anesthetic substances, in either volatile or gaseous form, that are inhaled via an anesthesia machine circuit.

Lacrimation: Tears from the lacrimal glands on the medial side of the tissue surrounding the eyes.

Minimal Alveolar Concentration (MAC): A measure of potency of inhalation anesthetic agents; occurs when the equilibrium end-tidal anesthetic concentrations, expressed as a fraction of 1 atm, prevent movement in response to surgical skin incision in 50% of human subjects.

Muscle Relaxation: A component of anesthesia in which the patient has reduced tension of the skeletal muscle.

Nociception: The perception of a painful or injurious stimulus

Scavenger System: Used to reduce exposure to escaping gases from the anesthesia machine; a waste gas suction tube (scavenger) is connected to the anesthesia machine, and the gases are then vented to the outside atmosphere via an operating room suction system.

Solubility Coefficient: The ratio of the concentration of an anesthetic in blood or other tissue to that in a gas phase when the two are in equilibrium.

Sympatholysis: A component of anesthesia in which the patient is blocked from having an autonomic response to nociceptive (painful) stimuli.

Vaporizer: A device on the anesthesia machine that converts liquid anesthetics into metered amounts of vapor that are added to the fresh gas mixture to produce a known concentration of the vaporized form of the inhalational anesthetic agent.

Goals for Anesthesia

The goals of anesthesia are to create a supportive environment for the surgeon, which include hypnosis or unconsciousness, analgesia, immobility or muscle relaxation, amnesia, and perhaps sympatholysis, which is attenuation of autonomic responses to noxious stimuli.

These goals may need to be obtained either fully or sometime just partially.

We do not always place patient under general anesthesia. Often less is more and monitored anesthesia care or MAC, which can be just light sedation, is all that is needed.

MAC vs. MAC vs. GA vs. GETA

General anesthesia or GA is when the patient is fully asleep and will not react to surgical stimulation. They will not have any awareness of what is going on around them and they will not feel any pain.

GETA or general endotracheal anesthesia is the same as GA, with the slight difference of using an endotracheal tube for intubation.

Monitored anesthesia care or MAC covers a wide range of sedation techniques from light sedation to general anesthesia.

The term MAC can easily be confused with the inhalation gas term minimum alveolar concentration, which is also called MAC. But they are not the same. Monitored anesthesia care is an anesthetic technique, whereas minimum alveolar concentration is a measure of anesthetic depth.

Minimum Alveolar Concentration - MAC

Minimum alveolar concentration is the amount of inhalational agent, or gas, that is necessary to ensure that 50% of patients will not move when the surgeon cuts the skin. This makes minimum alveolar concentration a measure of anesthetic depth.

There are several factors that may influence MAC.

Factors with Impact on MAC		
Decrease	**Increase**	**No Effect**
Advanced age (>40)	Young age (<1 year)	Anesthetic duration
Metabolic acidosis	Hyperthermia	K^+ levels
Hypoxia (<38 mmHg = SpO_2~70%)	Hypernatremia	Anesthetic metabolism
Hypotension	Chronic alcohol abuse	Thyroid conditions
Decreased CNS neurotransmitter levels	Increase in CNS neurotransmitter levels	Gender
Acute alcohol, marijuana		Species
Pregnancy		
Opioids, ketamine, diazepam, lithium		
Anemia		
Hypothermia		
Hyponatremia		

Anesthesia continuum

Anesthesia is not an on/off button. We don't have the ability to create full anesthetic depth instantly, but we see anesthesia as a continuum from awake and conscious to fully asleep and unconscious.

We tailor the level of anesthesia dependent of the need for the surgery and the individual surgeon preference, all considering what is safe for the individual patient.

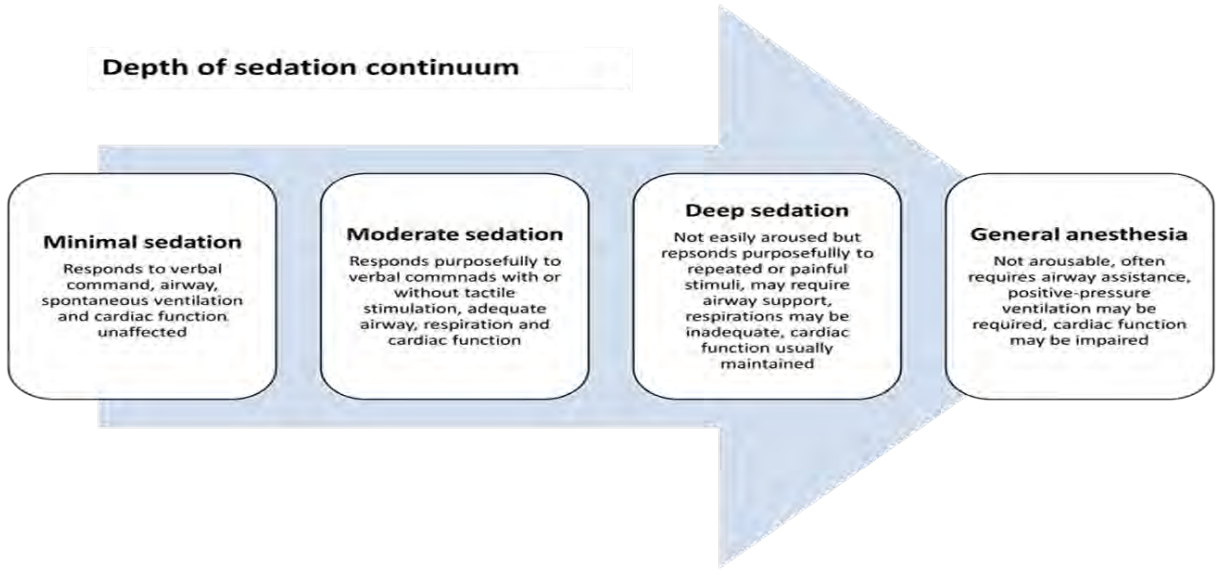
The depth of anesthesia goes from minimal sedation, where the patient can respond to verbal command, have patent airway with spontaneous breathing, and cardiac functions are unaffected.

A little deeper we call moderate sedation. Now the patient will respond purposefully to verbal commands with or without tactile stimulation, they have adequate airway, respiratory, and cardiac functions.

During deep sedation, the patient will not easily be aroused, but will respond purposefully to repeated or painful stimuli, they may require airway support, and respiration may be inadequate. Cardiac function is usually maintained.

During general anesthesia, the patient is not arousable even by painful stimuli. They often, but not always, require airway assistance by as little as chin-lift to possibly positive-pressure ventilation. Cardiac function may be impaired.

The difference from one level to the next is very fluid and can happen without notice, which is why it is not recommended that non-anesthesia personnel attempt moderate sedation. If the patient suddenly drifts deeper into deep sedation respiratory function could be impaired and beyond the capability of a non-anesthesia trained person.



Stages of Anesthesia

We categorize anesthesia in different stages from 1 through 4. The transit through the stages is facilitated by drugs or gasses administered by the anesthetist.

Stage I is also called the awake stage

During stage 1 the patient remains conscious but has no memory of what happens and will not experience pain. The pt can follow simple commands, but will not remember this. Most importantly protective reflexes (eg. gag reflex) are intact. This is the level the pt often experience going back to the room after versed and fentanyl administered in the holding area.

Physiologically we note

- Normal breathing
- Opens eyes to command
- Maintains protective reflexes (eg. gag reflex)
- May tolerate mild painful procedures, eg. IV start

Stage II is called stage of delirium

Stage 2 is the delirium stage, where the patient will have dreams. The patient experiences loss of eye lid reflex and the respiration becomes irregular, but still effective for the most part. Stage 2 passes quickly, but is very important to recognize, since in this stage the vocal cords are very susceptible to muscle spasm, thereby causing laryngospasm. It is important to recognize that the patient will experience stage 2 during both induction and emergence.

Physiologically we note

- Excitement
- Irregular respiration
- Risk of vomiting
- Risk of laryngospasm

Stage III or the stage of surgical anesthesia

This stage is divided into four planes, each representing a deeper anesthetic level than the previous. We look for certain physiologic parameters to identify these planes.

1st plane – regular respiration, normal pupils. Swallow - retching - vomiting reflexes disappear and come back in reverse order during emergence

2nd plane – regular respiration but shallow, cease muscle movements, laryngospasm reflex fully disappears

3rd plane – decreasing intercostal respiration, onset of diaphragmatic respiration

4th plane – irregular respiration, maximally dilated pupils, respiration is now diaphragmatic and eventually will stop

Most surgeries take place during stage 3. There is an absence of eyelash response, blink, and swallowing reflex. This is also the stage in which we intubate the pt.

Most general surgeries are performed in stage 3 – 3rd plane, where the surgeon have optimal conditions to perform the procedure.

A decreased depth of anesthesia can be useful, depending on how quickly we want the pt to wake up. Procedures may require wake up during the procedure to check neurological status.

Breathing is controlled either by the ventilator or by spontaneous breathing. If a ventilator is used, we keep the $PaCO_2$ low (around 30-35 mmHg or low normal) to suppress the pt's own drive to breathe. When breathing spontaneously the $PaCO_2$ is often elevated due to respiratory depression from narcotic and the volatile agent.

Stage IV or the overdose stage

Stage 4 is very deep anesthesia and not needed for surgery, mostly considered an overdose. You may see respiratory and CV collapse.

Despite this, stage 4 if often seen during pediatric inhalational induction and must then be addressed aggressively to avert disaster. Treatment with an anticholinergic, eg. administer atropine, and decrease in gas concentration as well as supporting blood pressure and ventilation may be necessary.

Predictors of Anesthetic Depth

There are several parameters we use to determine the depth of anesthesia apart from what we can read off our monitors.

Among the more sensitive are breathing and the eyes.

Breathing is the most sensitive parameter. Breathing is under autonomic control, which makes it so sensitive to anesthesia. When the patient uses diaphragmatic muscles without the intercostals we can reliable determine the patient is somewhere in stage 3 or surgical stage. In PACU this should not be the case, but hopefully a lighter level of anesthesia may be observed with a more regular breathing pattern, rhythm, and depth. These are signs to look for though, when emerging the patient from a ventilator in PACU.

Eye movement indicates light anesthesia and when the eyes become divergent, each eye looking outwards, the patient is in stage II. During induction, eventually the eyes become cross-eyed, which indicates stage III.

Lacrimation happens during light anesthesia and is a sign of pending emergence.

Another factor looked at is muscle tone and less tone equal deeper anesthesia.

No single parameter should be used alone, but rather taken as part of a whole in the assessment of the patient. Constant vigilance is the name of the game.

Pharmacokinetics of inhaled anesthetics

Anesthesia machine ⇨ Alveoli

The goal for the anesthetist is to maintain a constant and optimal concentration of gas in the brain. We do this by adjusting the delivery of gas from the vaporizer. We can only have a single gas vaporizer on at any given time, it is not possible to combine volatile agents.

Optimally the partial pressures in the alveoli, the arteries, and the brain equalizes. This takes time and does not happen immediately.

Consider the gas flow starting at the anesthesia machine the first stop is in the alveoli. When we perform an inhalational induction, when the patient breathes themselves to sleep, the following are important parameters.

Increasing the overall flow, called fresh gas flow, and the delivered gas concentration will speed up the onset of anesthesia by building up the partial pressure of gas in the alveoli faster.

A high minute ventilation by the patient will increase the speed of induction.

Interestingly, physics dictates that higher concentrations of delivered gas will cause a relatively even higher concentration of gas in the alveoli. This is called the concentration effect and will speed up induction.

The anesthesia machine uses non-absorbable parts, so gas does not adhere to machine parts.

Alveoli ⇨ Blood

Next step is to bring the gas into the blood system, the arterial blood.

Some physical properties of the gas determine how fast and how much of the gas is exchanged between the alveoli and the arteries.

Body Tissue Composition and Blood Flow

Tissue	Body mass (% of 70 kg adult)	Blood flow (% of CO)	Perfusion mL/minute/100 g	Time constant minutes
Vessel Rich Group (VRG) Lungs – liver – kidneys – brain - heart	10	75	75	2-3
Muscle Group (MG) Muscle, skin	50	19	3	33
Fat Group (FG) Fat, bone marrow	20	6	2-3	2500
Vessel Poor Group (VPG) Bone, tendons, ligament, teeth	20	<1	0	∞

The blood: gas coefficient, also called the Oswald solubility coefficient is important. The more soluble a gas is, the more will be absorbed in the blood. Isoflurane is more soluble than desflurane, so more isoflurane is absorbed in blood than for desflurane.

But, there is a slight hiccup with a high solubility; high solubility results in a slow induction. In other words, it takes longer for the patient to fall asleep. This is because the gas tends to stay in the blood and is not willing to pass on to the brain tissue, which is where we want it to go.

Also, the cardiac output is important. Low cardiac output results in fast induction and vice versa, high cardiac output results in slow induction. This is because the build-up of partial pressure in blood happens faster with low cardiac output.

Blood is distributed to the different tissues of the body at different speed and volume. Much more blood is going to the vessel rich group, lungs – liver – kidneys – brain – heart, than to the muscles and fat.

As can be seen in the chart using an average adult person of 70 kg, the vessel rich group receives 75% of the blood flow and this group is only 10% of the body mass. The VRG also receives a much faster blood flow than any other group. This group, therefore, receives a massive amount of blood compared to any other part of the body. The result is that the brain, being a part of the VRG, receiving a large amount of blood, can be anesthetized quickly.

We talk about a time constant regarding inhaled anesthetic, don't worry what it is, but the lower the time constant the faster a drug can influence a particular tissue. The time constant for the VRG is 2-3 minutes compared to 33 minutes for muscle and 2500 minutes for fat. Clearly, much lower for the VRG.

Eventually, during the anesthesia the other compartments will start to receive and fill up with inhaled gas. If the anesthetic is long enough the muscle group will saturate, but it is unlikely that the fat tissue will ever be saturated. The more these tissues saturate the longer it takes to emerge the patient, since these tissues will act as storage units and keep releasing gas long after the concentration in the brain has fallen to acceptable levels for wake up.

Interestingly, it requires a lower partial volatile gas pressure in the body to wake up than it does to go to sleep. Consequently, we must plan early for the emergence and try to minimize the gas concentration in the body to allow for a rapid wake up of the patient.

Despite this, the patient will still be giving off volatile gas when they arrive to PACU, which you can smell on the patient's breath. Be aware of this when you dose the patient with sedatives or narcotics.

Blood ⇨ Tissue/Organs

We just saw that a massive amount of blood goes to the brain. The brain is obviously the most important organ when we want the patient to go to sleep, induction.

The partial pressure of gas in the alveoli is the first to be maximized, then the blood concentration will start to increase, and finally the concentration in the brain will start to rise.

The goal, as stated earlier, is for these three areas to equalize: PA = Pa = Pbr.

The larger the gradient, the faster the gas will move across the membranes towards equilibration. Therefore, high fresh gas flow and high concentration of gas from the vaporizer is beneficiary for fast inhalational induction.

Why does the patient fall asleep?
(Induction)

We keep talking about induction. What happens, why the patient goes to sleep is still a mystery. We don't know why anesthetics work. We have theories, but no confirmation that any of these are correct.

One theory is the Meyer-Overton theory. This postulates that gas will dissolve in fatty tissue, of which there is plenty around brain cells. When these fatty tissues saturate they cause anesthesia.

Another theory is the receptor theory. This theory states that there are anesthesia receptors, eg. GABA or others, that bind the gas and cause anesthesia.

We do know that increased lipid solubility of a gas will increase the potency of that gas. The more lipid soluble a gas is, the more potent or "stronger" it is. Isoflurane, being the most lipid soluble, is the most potent of the volatile agents.

Waking Up the patient
(Emergence)

So how do we wake up the patient. We call this emergence.

Basically, we just turn off the gas and turn up the flow of oxygen.

Physiologically, this will create inverse partial pressure gradients, where the lowest partial pressure now is in the alveoli and the highest in the brain tissue.

When we no longer deliver gas to the lungs, but only oxygen, the partial pressure of gas will become zero in the alveoli. Now gas in the blood will move out of the blood and into the alveoli, for then to be "aired out" by the next breath.

This will cause the partial pressure of gas to fall in the blood and, hence, the gas to flow out of the brain and other tissues and into the blood. The blood will again return to the lungs and the gas will be "aired out" of the system.

Eventually, there will be no more gas left in the brain and the patient will emerge from anesthesia.

When emerging a patient from anesthesia, there are several factors that will influence how fast this will occur.

Increasing the flow of non-volatile agent, mostly oxygen, will speed up the removal of volatile agent.

Increased minute ventilation, respiratory rate multiplied with tidal volume, will also speed up the removal of volatile agent.

It is beneficial to have relative larger tidal volume and fewer breaths, since this will decrease the movement of deadspace. Deadspace is the part of the airway/lung that does not take part of the gas exchange.

We can also introduce nitrous oxide towards the end of surgery, since this can replace some of the volatile agent. The very low lipid solubility of nitrous oxide allows this gas to come off the tissues faster than volatile agent.

Talking about volatile agents, these have different lipid solubilities and it, therefore, makes a difference which volatile agent we use for our anesthetic. Volatile agent with a lower lipid solubility will come off faster and the patient will thus emerge faster. Desflurane has the lowest solubility, followed by sevoflurane and isoflurane in that order.

It is also important for the patient to have an adequate core temperature. Colder tissues have an increased ability to retain volatile agent, which will slow down emergence.

Anesthesia Machine

(The Inner Workings)

The anesthesia machine is what we use to deliver the volatile agent along with oxygen, air, and/or nitrous oxide to the patient. Most commonly used is what we call a semi-closed-circuit system.

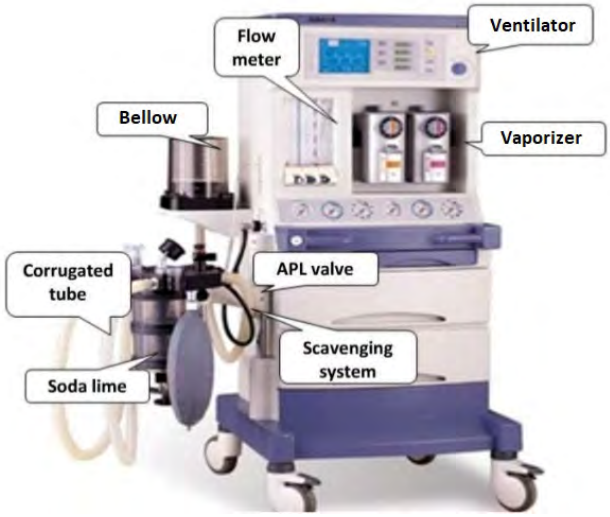
In its most basic form we can view the anesthesia machine as a device that delivers oxygen, air, or nitrous oxide via a flowmeter. This determines how much of each gas is delivered to the patient.

The vaporizer is where the volatile agent is stored. The device determines how much volatile agent is introduced into the flow of oxygen, air, or nitrous oxide.

The anesthesia machine has a ventilator built in, which we use to ventilate the patient during most of the case. This is much easier than to hand ventilate for extended periods of time!

The bellow is just a visual of the ventilator working. When the bellow moves up and down we can “see” the air going in and out of the lungs.

We deliver the gas mix (oxygen, air, nitrous oxide, and/or volatile agent) to the patient through a corrugated tube system called the "circuit".

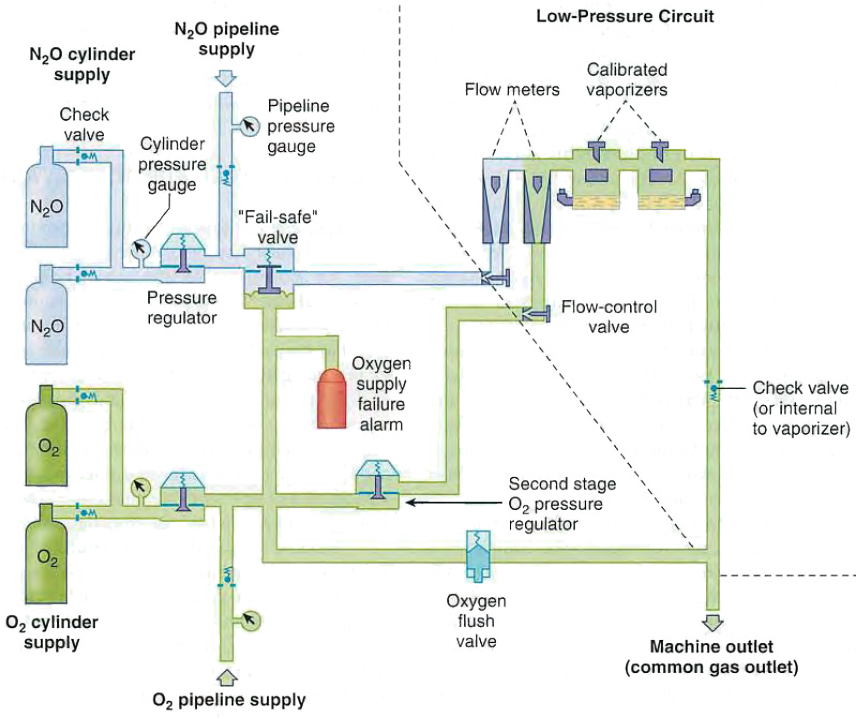


If we don't use the ventilator, we can adjust the resistance to flow by the adjustable pressure limiting or APL valve. This allows us to force air mix into the patient's lungs.

The soda lime canister removes the carbon dioxide (CO_2) from the flow mix. Remember, carbon dioxide, is a waste product of metabolism in the body.

Finally, excessive gas is removed from the system by the scavenging system, which is then removed from the OR. In the old days, these excessive gasses were vented into the OR!

This next picture shows in slightly more detail how the gasses flow through the anesthesia machine. The gasses come into the machine at different pressures.



The cylinders, seen to the left of the red arrow, are considered high pressure.

The gas passes through pressure regulators that decrease the high oxygen or air pressure (up to 2,000 psig) to intermediate pressure (around 45-50 psig).

The secondary pressure regulators decrease the gas pressure further to what is considered low pressure (around 15 psig).

The gasses, which can be any of the non-volatile agent gasses, oxygen, air, and nitrous oxide, can also come from the hospital wall supply. It is then delivered at intermediate pressure.

The gas now flows through the flowmeters, which allows us to regulate how much we deliver of each gas.

Next step is for the gas to flow through the vaporizer and collect the volatile agent we use to anesthetize the patient.

Finally, the gas mix is delivered to the patient via the corrugated circuit.

Inhalational Agents

The first anesthetic gas used to anesthetize a patient for surgery was diethyl ether (or just ether for short) in 1842. Nitrous oxide was initially a party gas, because it causes a light, giggly mood. It was introduced to anesthesia in 1845. Chloroform was introduced in 1847 and remained popular for many years, although, it is very toxic and the risk of death during anesthesia is at least 4-5 times higher than with ether.

Cyclopropane was introduced into anesthesia in 1934. All these anesthetic gasses were highly flammable and could easily explode in the OR.

Halothane, introduced into anesthesia in 1956, was the first modern anesthetic volatile agent.

Each of these gasses had significant side effects and toxicities. The therapeutic index was low for each of these gasses as well, which makes them harder to titrate.

The only good gas, although some will dispute this, was nitrous oxide. Nitrous oxide is not a volatile agent and it does have some side effects.

Modern Volatile Agents

Modern western anesthesia only uses three volatile agents: isoflurane, desflurane, and sevoflurane. Halothane is widely used in third world countries due to low cost.

Volatile agent means “easily able to change from liquid to gaseous form”.

Neither of these agents are flammable, nor explosive and each has a better therapeutic index than the old gasses.

The improvement of these volatile agents is due to a change in the molecular structure of the gas. It is now possible to add a halogen or fluoride atom into an ether gas and this will create these new gasses.

All these gasses are liquids at room temperature and sea-level atmospheric pressure. The anesthesia vaporizer unit will let this liquid vaporize and we can now introduce the volatile agent in gas form into the circuit that delivers gas to the patient. The vaporizer unit can deliver a precise concentration of volatile agent to the patient. The concentration of volatile agent delivered determines the depth of anesthesia of the patient.

Volatile Agents Effect on Organs

CNS – CV - GI

All volatile agents have similar effects on the human body systems.

The central nervous system will show a dose-dependent depression and decreased oxygen metabolism. There will be an increase in the blood flow, which will cause an increase in the intracranial pressure.

The cardio-vascular system may develop arrhythmias, ectopy, and increased heart rate. These symptoms are also dose-dependent.

In the gastro-intestinal system we will see a relaxation of the smooth muscles and a decrease in the muscle activity.

Respiratory – Renal/Hepatic - Uterine

All volatile agents cause a dose-dependent depression of the respiratory system, which causes the patient to breathe more shallow and slower. This will create a buildup of carbon dioxide in the system. The volatile agents also dull the ventilatory response to this increased carbon dioxide level. This should be of particular concern for the PACU nurse, since some of the volatile agent will still be coming off when the patient arrives to PACU.

On a positive note, all the volatile agents cause bronchodilation. Since sevoflurane is the least irritating of the three agents, it is often used for asthma patients.

The blood flow will decrease to the liver and kidneys due to a decrease in the cardiac output. This may lead to a decreased glomerular filtration rate and oliguria, decreased urinary output. The surgical insult will also cause an increase in antidiuretic output from the pituitary gland, which will affect the sodium balance and cause fluid retention. Overall, you may see a decrease in urinary output as well.

There will be a dose-dependent relaxation of the uterus, which may cause increased bleeding during C-sections. For a C-section we prefer regional anesthesia, ie. spinal or epidural.

Volatile Agents

(Toxicities and Side Effects)

Although the modern volatile agents are much safer than the old agents, there are still risks associated with the use of a volatile agent.

Most notable is the respiratory depression, which may even cause apnea, especially, when a volatile agent is used in conjunction with sedatives and analgesics.

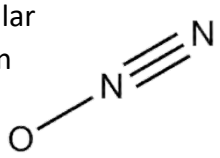
There is a dose-dependent cardiac depression, which leads to decreased cardiac output (bradycardia and decreased stroke volume).

Some patients will, when exposed to volatile agent or succinylcholine, have a vicious reaction with severe muscle contraction and a fast rise in body temperature. This is called malignant hyperthermia. Significant complications include rhabdomyolysis (break down of muscle cells) and hyperkalemia (high potassium level from muscle cell breakdown). The PACU nurse may note tea-colored urine from the rhabdomyolysis and ECG changes from the hyperkalemia. Other symptoms may be noted, like increased heart rate, increased respiratory rate caused by increased CO_2 production, mixed acidosis noted on ABGs, and masseter muscle rigidity (jaw muscle stiffness). The rise in body temperature is a late sign of malignant hyperthermia.

All the volatile agents cause vasodilation, which leads to a redistribution of the blood flow. There will be an increased blood flow to the periphery with an ensuing loss of heat. Additionally, volatile agents have a depressant effect on the hypothalamus, which will disrupt the temperature regulation. This may lead to hypothermia and possible shivering, or, if the patient is heated too much, hyperthermia. Shivering is a beneficial reaction by the body when getting cold. Unfortunately, many of our patients are elderly, with poor cardiac function, and shivering increases oxygen utilization, so this increased workload on the heart may be detrimental and should be avoided. Additionally, shivering is not pleasant for the patient. The operating rooms are notoriously cold, which makes it even harder to keep the patient comfortably warm during surgery.

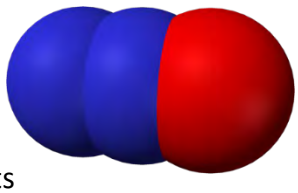
Nitrous Oxide

Nitrous oxide is an odorless, sweet smelling inorganic gas. It is a simple molecular structure, not flammable, but it will support combustion (fire) as well as oxygen will.



It does have analgesic effect and is often used at the dentist's office.

It has a very low lipid solubility, which makes for a fast onset/offset of anesthesia. It cannot be used as a sole agent, since its potency is very low.



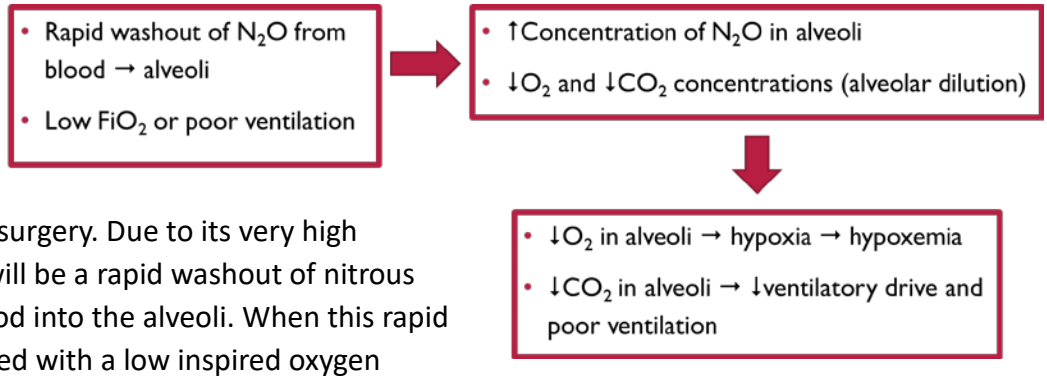
Nitrous oxide has a risk of causing post-operative nausea and vomiting – also called PONV. This is due to a pressure build-up in the inner ear.

Due to its high diffusibility it can fill closed spaces with excessive amounts of nitrous oxide. This can cause pneumocephalus (gas inside the brain), pneumothorax (gas inside the chest), gas in the middle ear (cause of PONV), and gas build-up in the intestines causing bowel obstruction.

Due to its very rapid diffusibility it can cause diffusion hypoxia after emergence.

Diffusion Hypoxia

Let me explain diffusion hypoxia, since this is a situation you may see in PACU, especially if you place the patient on room air to "get a baseline". Always check with the anesthesia provider if it is acceptable to not give supplemental oxygen.



Diffusion hypoxia can happen after emergence, when nitrous oxide has been used at the end of surgery. Due to its very high diffusibility there will be a rapid washout of nitrous oxide from the blood into the alveoli. When this rapid washout is combined with a low inspired oxygen concentration or poor ventilation (eg., narcotized) it creates an increased concentration of nitrous oxide in the alveoli, as well as a dilutional decrease in concentration of oxygen and carbon dioxide in the alveoli.

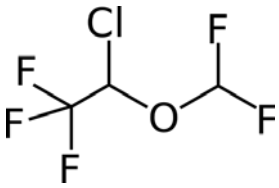
The combination of increased nitrous oxide and decreased oxygen concentrations in the alveoli will cause hypoxia and subsequent hypoxemia. The decreased carbon dioxide concentration will result in a decreased ventilatory drive, which in turn will cause poor ventilation and worsening of hypoxia. All in all, a detrimental situation for the patient.

The treatment is simple. Supply high inspired concentration of oxygen by leaving them on an oxygen mask for 10 minutes post extubation. After 10 minutes, the nitrous oxide has disappeared from the system.

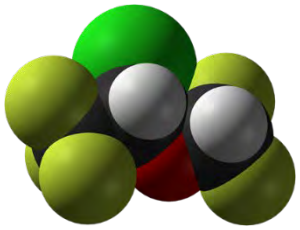
Key point, leave the patient on oxygen, when anesthesia brings the patient to PACU with oxygen, until safe per anesthesia.

Isoflurane

The oldest of the three modern volatile agents is isoflurane. Iso is a halogenated methyl ethyl ether, meaning it is an ether, like the old diethyl ether or just ether, although, with halogenated (chloride and fluoride) atoms attached to the ether bridge (the O in the drawing or the red ball).



Iso has a strong, pungent, and irritating odor and is, therefore, not to be used for inhalational induction. This could create breath holding and possible laryngospasm.



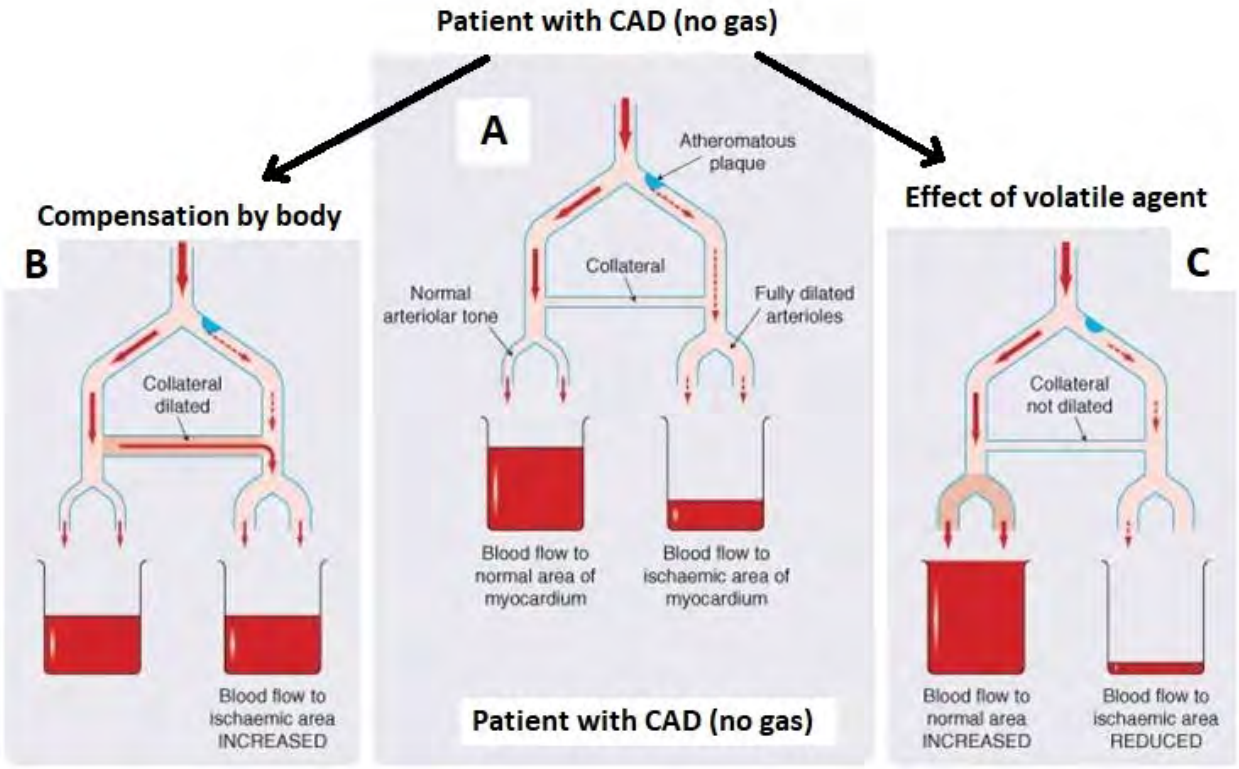
It is commonly used and is the cheapest of the modern volatile agents on the market.

One side effect of isoflurane is its tendency to sensitize the myocardial muscle. If we place a regional block, we should, therefore, add less than 5 mcg/kg of epinephrine to the local anesthetic.

Isoflurane may also cause coronary steal, although this is rare and mostly not considered an actual concern during anesthesia. Despite this, let me explain coronary steal.

Coronary Steal

When a blood vessel begins to narrow due to buildup of plaque, the blood flow through this vessel will decrease. This can cause ischemia.



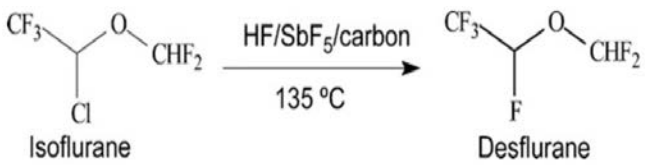
The body will try to compensate for this decreased flow by vasodilating this narrowed vessel and divert flow through collateral vessels. This is meant to increase the blood flow back towards normal. With increased plaque buildup, eventually the body will not be able to compensate for the narrowing effect of the plaque. At this point the narrow vessels cannot vasodilate any further; they are stuck.

Isoflurane is a known potent coronary vasodilator. Isoflurane induced coronary vasodilation will redistribute the blood flow away from these stuck vessels, since they cannot vasodilate any more than they already are. Therefore, this redistribution of blood flow will create an increase in flow to normal tissue and a reduced flow to ischemic tissue.

This is called coronary steal, since the vasodilation causes a steal effect away from the diseased tissue towards the normal tissue. This could be detrimental for the patient.

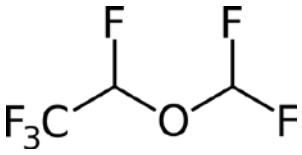
Desflurane

Desflurane is a fluorinated methyl ethyl ether. This volatile agent is derived from isoflurane by changing a single molecule;



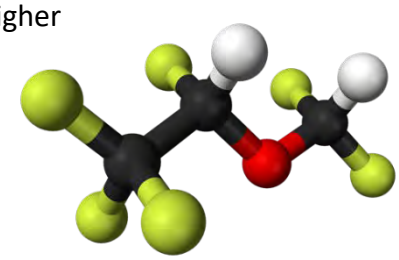
remove chloride from isoflurane and replace that with fluoride, creating desflurane.

Desflurane has a very pungent and irritating odor and should not be used for inhalational induction.



This volatile agent has a low lipid solubility, somewhat like nitrous oxide, which allows fast anesthesia onset and offset.

The low potency of desflurane means it must be used in much higher concentrations than the other volatile agents, which increases the cost.



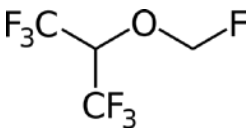
Like isoflurane, desflurane may sensitize the myocardial musculature and epinephrine should be limited to 7 mcg/kg if added to local anesthetic for a regional block.

Desflurane may also cause coronary steal, although, this is less likely than with isoflurane.

Sevoflurane

Sevoflurane is the newest of the commonly used volatile agents and sevoflurane is a fluorinated methyl isopropyl ether. Again, a molecular change to an ether molecule.

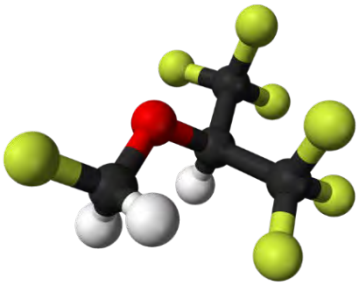
Sevo has a sweet-smelling odor, which makes it almost ideal for inhalational induction. It does not irritate the bronchial tree and is, therefore, good for patients with reactive airways, eg. asthma patients.



Sevo has a low lipid solubility that allows for a fast onset and offset of anesthesia, although, with longer cases (probably more than a couple of hours) emergence appears to be equal to isoflurane.

Sevo probably does not cause coronary steal.

But, sevo does sensitize the myocardial muscle to the same effect as isoflurane and, therefore, epinephrine should be limited to less than 5 mcg/kg when added to local anesthetic for regional blocks.



Although rare, sevo can cause seizure activity, especially with pediatric patients.

Clinical Advantages and Disadvantages

This chart summarizes the pros and cons for the different gasses we use in anesthesia. Despite many similarities there are situations where it will be beneficial to select a specific agent or gas.

A few things should be pointed out.

The only gas with analgesic properties is nitrous oxide. This gas is also nonpungent and is often used during inhalational induction. Nitrous oxide does not trigger malignant hyperthermia.

Biggest drawbacks with nitrous oxide are probably its tendency to enter closed spaces and cause nausea and vomiting.

Clinical advantages & Disadvantages		
Anesthetic	**Advantages**	**Disadvantages**
Nitrous Oxide	Analgesia Rapid uptake and elimination Little cardiac or respiratory depression Nonpungent Reduces MAC of more potent agents	Expansion of closed air spaces Requires high concentrations Amount of oxygen delivered is reduced Diffusion hypoxia Causes nausea and vomiting
Isoflurane	Moderate muscle relaxation Decreases cerebral metabolic rate Minimal biotransformation No significant systemic toxicity Inexpensive	Pungent odor Airway irritant Trigger malignant hyperthermia
Desflurane	Rapid uptake and elimination Stable molecules Minimal biotransformation No significant systemic toxicity Decreases cerebral metabolic rate	Airway irritant Expensive compared to other agents Needs special electrically heated vaporizer Rapid increases in inspired concentration can lead to reflex tachycardia and hypertension Trigger malignant hyperthermia
Sevoflurane	Rapid uptake and elimination Nonpungent Excellent for inhalation induction Cardiovascular effect broadly comparable to those of isoflurane	Reacts with soda lime More expensive than isoflurane Trigger malignant hyperthermia

Isoflurane is the oldest and the cheapest of the volatile agents. It is very reliable and easy to titrate to effect. Downsides are that it triggers malignant hyperthermia and has a pungent odor.

Desflurane has a very rapid uptake and elimination, and, therefore, fast onset and offset of anesthesia. Downsides are that it is very irritable to the lungs, so it is not good for inhalational induction and it also needs a special vaporizer that is heated and pressurized. Unfortunately, it is expensive and due to its low potency must be used in high concentrations. Like all volatile agents it can cause malignant hyperthermia.

Sevoflurane is excellent for inhalational induction, due to being nonpungent. It is almost as expensive as desflurane and may cause malignant hyperthermia.

Important Key Points for the PACU RN

To finish up this presentation of inhalational anesthetics I will cover a few key points special for the PACU nurse.

As always, vigilance is the key to recovering a patient after general anesthesia. It only takes a very short moment of not paying attention for a patient to suffer irreversible harm.

A basic understanding of the effects caused by the inhaled anesthetic agents will be most helpful during recovery. It is much easier to anticipate what can happen with a good understanding of the pharmacology of these gasses.

Monitor vital signs closely. All volatile agents have both cardiac and respiratory depressant effects. Preoperative vital signs are a good starting point when receiving the patient from the OR. Be observant of the $EtCO_2$ level in the obese and/or patients with obstructive sleep apnea.

Although the patient is awake when brought out from the OR, there is still volatile agent being released from the tissues. You can smell this on the patient's breath.

With a long anesthetic, more than 3-4 hours, volatile agents will be released for an extended period due to an excessive absorption into the muscle and fat tissues.

If nitrous oxide was used, you should allow for a minimum of 10 minutes with mask oxygen before attempting to have the patient on room air due to the risk of diffusion hypoxia.

Temperature regulation may be depressed by general anesthesia. You should observe for both hypo- and hyperthermia. Shivering is unpleasant and can significantly increase the oxygen consumption for the patient. Shivering is most often noted with pediatrics and if the core temperature is low; more than 2 degrees Celsius or 4-degree Fahrenheit below the patient's normal core temperature.

The most common drug treatment options are clonidine and meperidine, but don't forget about warm blankets or forced air-heating devices.

Surgery causes anti diuretic hormone (ADH) release. This is triggered by stress, pain, nausea, bleeding, various drugs given, and positive-pressure ventilation by anesthesia. ADH causes sodium and water retention, which will decrease urinary output. This can last 1-2 days post-operative and may warrant small doses of diuretic.

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