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Welcome

We would like to welcome you to the Canadian Dental Anaesthesia Assistant Certification (C-DAAC), an exciting and new educational program, the first of its kind ever offered in Canada. We want this experience to be nothing short of exceptional for you. This book has been prepared as a course home study manual which will provide you with the necessary tools to meet the challenges of working in a dental setting where general anaesthesia, procedural sedation and analgesia (PSA), and local anaesthesia are being delivered. In combination with the required two day hands on skills workshop you will be better prepared to be an integral part of a team delivering safe and effective anaesthetic and dental care to your patients.

Prerequisites

- The participant must hold a valid provincial designation as a dental assistant or hygienist and be in good standing with their governing body.
- The participant must hold a current BCLS healthcare provider CPR certification

Learning Objectives

Upon completion of this course the participant should be able to

- List the main divisions and components of the peripheral and central nervous system.
- Describe the basic anatomy and physiology of the respiratory system in relation to the field of dental surgery and anaesthesiology.
- Describe the basic anatomy and physiology of the cardiovascular system in relation to the field of dental surgery and anaesthesiology.
- Understand concepts of basic pharmacology.
- Understand the pharmacology of key drugs used in local anaesthesia, sedation, analgesia, and general anaesthesia.
- Understand the differences between the various levels of sedation, anaesthesia, nitrous oxide analgesia, and local anaesthesia.
- Understand appropriate monitoring and necessary chart keeping for safe delivery anaesthetic care.
- Understand the prevention, recognition and management of complications associated with office based sedation and anaesthesia.
- Understand the management of emergencies in the dental office and list and discuss the emergency drugs and equipment required for the prevention and management of emergency situations.
- To recognize, react to, and manage dental medical emergencies as a team member.
- To be able to manage or assist in the management of the compromised patient airway using basic and advanced airway techniques.
• Understand key principles of advanced cardiac life support.

Overview of Content

The Course is divided into the following modules

• Basic human anatomy and physiology review
• The anaesthetic spectrum of care
• Anaesthesia pharmacology and administrative techniques
• Preoperative/Postoperative care
• Anaesthesia equipment
• Anaesthetic emergencies

How to undertake the home study

Assignments

Each module has an accompanying online quiz that needs to be completed for grading. These quizzes are open book and are meant to help solidify key concepts for the student. As you read through each module you will notice words and sentences highlighted in **bold**. This is to aid you in the targeting of key information, words and concepts for study. This manual contains a lot of material which cannot be reasonably memorized. Areas of special importance for study are listed at the beginning of each module.

Disclaimer

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Module 1: Basic Human Anatomy and Physiology in Review

Key Areas for Study

- Understanding the key nerves used in nerve blocks for dentistry
- Understanding the autonomic nervous system and the sympathetic and parasympathetic nervous system components
- Flight or fight response
- The key adrenergic receptor types and example drugs that work through those sites.
- Vasovagal response
- Aerobic vs. anaerobic metabolism
- General structures of the upper airway and thoracic cage
- Structure and blood flow through the heart
- Cardiac concepts: automaticity, cardiac cycle, atrial kick, filling time, preload, afterload, shock,
- CO = SV x HR
- Acute Coronary Syndrome: MI and Angina
- MAP
- Basics of the electrical conduction system of heart
- Basics of ECG monitoring, the components of an ECG complex
- Main facial arteries

Part 1: The Nervous System

The nervous system is composed of two intercommunicating systems; the central and peripheral nervous systems. They work together receiving stimuli and coordinating a nervous system response. Nerves that carry information (impulses) towards the central nervous system (CNS) are referred to as sensory nerves while those that carry impulses away from the CNS are called motor nerves. Nerve cells themselves are called neurons. These types of cells are electrically excitable and transmit information by both electrical and chemical means.

The dendrites of a neuron bring impulses in from other neurons into the body (soma) of the cell. Impulses initiated/transferred are carried onwards down a long neural fiber called an axon. This part of a neuron can be up to a meter in length in humans. Axons are surrounded by an electrically insulating material called a myelin sheath whose main function is to speed up conduction. Impulses going down an axon terminate at axon terminals. A neural impulse is a wave of electrical depolarization. When traveling from the axon terminal to the dendrite of another neuron the impulse crosses a small gap called a synapse.
Central Nervous System

The CNS consists of the brain and the spinal column. The brain is composed of the cerebrum, the cerebellum, and the brain stem. The cerebrum is the largest part of the brain and sits above all other brain structures. There are two cerebral hemispheres which are covered with a cortical layer called the cerebral cortex. The cerebrum is the area of "higher thinking" such as learning, memory, emotions, self-control, hearing, and sight. The cerebellum plays an important role in motor function aiding in precision, coordination, and motor timing.

The brain stem is made up of the pons, the medulla oblongata, and the midbrain. The brain stem plays an important role in the control of cardiac, vasomotor and respiratory function. It controls pain sensitivity, alertness, awareness and consciousness. Lastly it plays a major role in neural conduction; all information relayed from the body to the cerebrum and cerebellum and vice versa, must traverse the brain stem. The spinal cord is composed of long tubular bundles of nervous tissue and support cells. It has three main functions: to conduct motor impulses down the cord, to conduct sensory information in the opposite direction, and as a center for coordinating certain autonomic reflexes.
Figure 1-2: The Brain
National Institute of Aging, Branch of the NIH, United States Department of Health and Human Services
The Peripheral Nervous System

The peripheral nervous system (PNS) is composed of sensory nerves that carry information from our environment to the CNS (afferent nerves) and motor nerves which carry the response from the CNS back out to the body (efferent nerves) to create such things as body movement. The main function of the PNS is to connect the CNS to the limbs and organs. The PNS is also divided into the somatic nervous system and the autonomic nervous system. The somatic nervous system is associated with the voluntary control of movement via skeletal muscles. The autonomic nervous system (ANS) controls body functions below the level of consciousness such as respiratory and cardiac regulation.

The cranial nerves (12 in total) originate from the base of the brain. These nerves are very important in oral and maxillofacial surgery. Some of these nerves are sensory nerves such as the olfactory nerve, the optic nerve and the auditory nerve. Some carry both sensory and motor fibers such as the trigeminal nerve (5th cranial nerve) which supplies sensation to the teeth and jaws and motor functions relating to mastication. Other cranial nerves carry only motor fibers such as the seventh cranial nerve (facial nerve).

Local anesthesia for oral surgeries primarily involves the trigeminal nerve. Anaesthesia of the inferior alveolar and lingual nerves occurs with an injection at the midpoint of the ascending ramus of the mandible. This anaesthetizes the mandibular teeth, the lip and the chin on the same side. With the lingual nerve half of the floor of the mouth and the tongue are anaesthetized. For the teeth of the maxilla a block injection can be done.
through the greater palatine foramen on the posterior palate to anaesthetize one half of the upper jaw. Individual teeth in the maxilla can be anaesthetized with an infiltration injection at the apex of the tooth.

Figure 1-4: The Trigeminal Nerve

The Autonomic Nervous System

The part of the PNS that controls visceral internal organs and unconscious functions is the ANS. The ANS can be divided into the sympathetic and parasympathetic nervous systems. These systems generally oppose/balance each other with the sympathetic side speeding things up and the parasympathetic side slowing things down.

Sympathetic System

The sympathetic nervous system is associated with the “fight or flight” response. It produces the optimal human response to having to fight for survival in a dire life.
threatening situation. If a person was being chased by a bear what responses would be optimal?

- Pupil dilation and ciliary muscles contraction for better close up vision
- Bronchial dilation and pulmonary blood vessel dilation for better gas exchange
- Increased rate and force of contraction of heart for increased cardiac output
- Constriction of skin and visceral organ vessels, dilation in skeletal muscles shunting blood to only where it is needed most
- Contraction of sphincters and inhibition of gut motility
- Stimulation of glycogen breakdown in liver and inhibition of bile production
- Contraction of spleen to place more blood into circulation
- Inhibition of urination

Medications that act on sympathetic (adrenergic) receptors are often used in emergency medicine and anaesthesiology. Drugs can be designed to block these receptors sites or to stimulate them. Several prominent classifications of receptor sites have been identified: Alpha, Beta-1 and Beta-2 receptors.

Alpha receptors are located throughout blood vessels in the body. Stimulation of alpha receptors leads to potent vasoconstriction. Epinephrine has strong alpha properties and is used in local anaesthetics to prolong the action of the anaesthetic or to decrease bleeding in a surgical field. Many sedatives, analgesics, and anaesthetics have a negative impact on vascular tone and heart rate, and hence blood pressure. The epinephrine from these injections can help counter those negative impacts.

Epinephrine also has strong Beta-1 and Beta-2 properties. Beta-1 action is associated with increasing cardiac output through increased heart rate and cardiac contractility. Epinephrine’s Beta-2 actions lead to bronchial dilation. Ventolin is a Beta-2 agonist (stimulant) and is the main drug used to treat an acute asthma attack. Before ventolin epinephrine was a main drug used for treating asthma.

A main category of cardiac medications are beta blockers. These drugs are used to rest the heart and decrease abnormal heart rhythms (arrhythmias) in patients having a heart attack. These drugs also have the unwanted side effect of attaching to some Beta-2 receptors. Blocking Beta-2 receptors causes bronchial constriction which can be dangerous in asthmatics so a contraindication to the use of beta blockers is a history of severe asthma.

**Parasympathetic System**

The parasympathetic nervous system is the system that slows down the body; it induces the body to be “at rest”. A main nerve that is parasympathetic is the 10th cranial nerve; the vagus nerve. The vagus nerve leaves the brain stem and innervates many organs before terminating in the rectum. Vagal stimulation leads to a decreasing heart rate “bradycardia”. A major cause of bradycardia and vasodilation is a vasovagal reaction. This often leads to syncopal episode for a patient.
Vasovagal reactions can be caused by a variety of stimuli such as:

- Rectal stimulation; vagus nerve innervates the rectum. A constipated patient on opioids bearing down in the bathroom may have a syncopal episode due to vagus nerve stimulation.
- Painful stimuli; many types
- Unpleasant stimuli; “the sight of blood, seeing a needle”
- Violent coughing
- Extreme emotions
- Pressure exerted on certain areas of the throat, sinuses, and eyes
- Cold stimuli to the face in children and infants

Stimulation of the parasympathetic system leads to:

- Decreased urination
- Dilation in salivary glands and external genitalia
- Secretion of watery saliva
- Decreasing heart rate and force of contraction
- Bronchiole constriction
- Increased GI motility, secretions, and relaxing of sphincters

In dentistry the need to decrease oral pharyngeal secretions has led to the use of parasympathetic blocking agents such as atropine to dry secretions. Atropine also is used to treat bradycardia such as might occur in a refractory vasovagal reaction.

**Part 2: The Respiratory System**

The respiratory system is involved with the exchange of oxygen and carbon dioxide and this process in general takes place unconsciously. The anatomical structures involved in respiration are numerous but the goal is simple: deliver oxygen to all cells in the human body and remove carbon dioxide by working in unison with the cardiovascular system.

Why is oxygen so important? It has to do with the production of key molecules used by all processes of human cellular life. A fundamental energy holding molecule of the human body is called adenosine triphosphate (ATP). Most processes in the human body are driven by the energy released when ATP loses a phosphate to form adenosine diphosphate. The production of ATP can occur in the presence or absence of oxygen. Table 1-A compares metabolism for both aerobic and anaerobic states. Of note is the fact that approximately 1/18th the number of ATP is produced without the presence of oxygen and that a byproduct of that production is lactic acid something harmful to the human body. In the presence of oxygen large amounts of ATP are produced and the byproducts of water and carbon dioxide are easily managed. The respiratory system is thus crucial to maintaining aerobic metabolism and the removal of CO₂, a byproduct of that metabolism.
Table 1-1: Aerobic and Anaerobic Metabolism in Comparison

<table>
<thead>
<tr>
<th></th>
<th>Aerobic Metabolism</th>
<th>Anaerobic Metabolism</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
<td>Metabolism in the presence of oxygen</td>
<td>Metabolism without oxygen</td>
</tr>
<tr>
<td><strong>Amount of Energy</strong></td>
<td><strong>36-38 ATP</strong></td>
<td><strong>2 ATP</strong></td>
</tr>
<tr>
<td><strong>Produced</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Byproducts</strong></td>
<td><strong>CO₂, H₂O</strong></td>
<td><strong>Lactic acid</strong></td>
</tr>
<tr>
<td><strong>Reactants</strong></td>
<td><strong>Glucose and oxygen</strong></td>
<td><strong>glucose</strong></td>
</tr>
<tr>
<td><strong>Sites of Reactions</strong></td>
<td>Cytoplasm and mitochondria of cells</td>
<td>Cytoplasm</td>
</tr>
</tbody>
</table>

Anatomy of the Pulmonary System

Upper Respiratory Tract

The upper airway starts at the nose and mouth. It includes the nasal passages, the four pairs of sinuses (frontal, maxillary, sphenoid, and ethmoid), and the pharynx. The pharynx is divided into three regions; the oropharynx, the nasopharynx, and the laryngopharynx. The nasopharynx differs from the other pharyngeal regions in that it is always patent (open). Air entering the nasopharynx is warmed, humidified, and filtered. The oropharynx lies between the soft palate and the epiglottis. The soft tissues of the oropharynx can contribute to airway obstruction in patients who have lost muscular tone during anaesthesia especially if they have obesity issues.

Lower Respiratory Tract

The lower respiratory tract starts at the opening to the trachea, called the glottal opening between the vocal cords. It consists of the trachea, the two mainstem bronchi, the bronchioles, and the lungs. The vocal cords are located at the larynx and are horizontal folds of tissue that attach to the thyroid cartilage. A rare but serious medical emergency can occur when the vocal cords go into what is known as laryngospasm. The vocal cords contract, often initiated by foreign materials in the laryngopharynx such as blood and mucus, and a severe airway obstruction occurs. This can happen as a patient is lightening after general anaesthesia (GA) or deep sedation.

Between the thyroid and cricoid cartilages anteriorly lies the cricothyroid membrane also known as the cricothyroid ligament. Creation of an opening through this membrane can be done if a patient experiences a severe upper airway obstruction. This is called a
cricothyrotomy. The most common way to achieve this is through what is known as the Melker technique.

Figure 1-5: Respiratory System
Figure 1-6: Upper Trachea
Remesz, Olek. Wikimedia Commons, 2008.

Figure 1-7: Melker Cricothyrotomy Set
Cook Medical; 2012
The cricoid cartilage is the only continuous circular cartilage in the trachea. It can be pushed on to compress the more posterior esophagus to stop or decrease the risk of gastric regurgitation during certain aspects of airway management.

The \textit{trachea} is about 12 cm in length and 2.5 cm in width. The walls of the trachea are made up of C-shaped rings of cartilage that keep it from collapsing. The internal ridge where the trachea bifurcates into the right and left bronchi is called the \textit{carina}. The \textbf{angle of bifurcation for the right bronchi is very shallow in comparison to the left bronchi}. This means that if a foreign body is aspirated it will often lodge in the right bronchial tree.

\begin{figure}[h]
\centering
\includegraphics[width=0.6\textwidth]{trachea.png}
\caption{Trachea and Bronchial Tree}
\end{figure}

Right lung aspiration pneumonia is much more common than left for this reason. The bronchi continue to divide into smaller and smaller bronchioles until when they are approximately 1 mm in diameter they terminate into \textbf{alveolar ducts and sacs}. Alveoli are the small grape like structures where gas exchange occurs with the capillaries of the bloodstream. Carbon dioxide rich blood is pumped from the rest of the body into the alveolar blood vessels where, through diffusion, it releases its carbon dioxide and absorbs oxygen. There are over 600 million alveoli in a human being.

\textbf{The Thoracic Cage}

How does air flow in and out of the complex respiratory system? The answer is in the creation of both negative and positive pressure through an intact \textbf{thoracic cage}. 
The thoracic cage is composed of the sternum, the ribs, the intercostal muscles, and the diaphragm. The diaphragm is a domed shaped muscle which when contracted is flattened in shape. **When the ribs are lifted upwards as the diaphragm contracts the volume of the “container” increases creating an area of negative pressure. The atmospheric pressure of the air is higher so air rushes in from higher pressure to lower pressure region. For expiration the opposite occurs. The diaphragm relaxes, the rib cage drops and the volume of the “container” decreases creating an area of higher pressure.**
Respiratory Physiology

There are two types of respiration. **External respiration** refers to the process of gas exchange at the alveolar level with oxygen and carbon dioxide. **Internal respiration** occurs at the cellular level when oxygen leaves the bloodstream and enters the cell and carbon dioxide leaves the cell and enters the bloodstream. Approximately 40% of blood is made up of **erythrocytes** (red blood cells). The percentage of RBC’s in the blood is referred to as **hematocrit**. A normal range is 42-52% in males and 37-48% in females. A component of RBC’s called hemoglobin is crucial to carrying oxygen. Each hemoglobin molecule can carry four oxygen molecules. Hemoglobin levels range from 12-18 mg/dl in healthy adults. A low hemoglobin concentration is called **anemia**.

Adults have approximately 20-30 trillion RBC’s at any given time each cell having a lifespan of 100-120 days. Around 2.4 million erythrocytes are produced every second and the time for each cell to do a complete circulatory loop through the body is around 20 seconds. Each RBC contains around 270 million hemoglobin molecules making up about a third of the cell volume. **This protein is responsible for the transport of 98% of the oxygen in the blood stream. The remaining oxygen is dissolved in the blood plasma.**

Carbon dioxide is picked up from cells and carried via the veins to the lungs in three forms. Thirty percent is carried attached to hemoglobin, seven to ten percent is carried dissolved in plasma, and the majority 60-70% is carried as bicarbonate through the bicarbonate buffer reaction. In normal RBC’s there is both oxygen and carbon dioxide attached to hemoglobin in both arterial and venous blood. The cells only use what oxygen is needed and release what carbon dioxide they produce thus leaving venous blood with oxygen but at a lower level.

Hemoglobin can carry a maximum of around 1.34 ml of oxygen. The maximum amount of oxygen a person’s blood can carry is called **oxygen capacity**. At any given moment the amount being carried may be equal to or less than this oxygen capacity. **Oxygen saturation is the percentage of actual oxygen carried compared with the oxygen capacity. Normal arterial oxygen saturation is ≥ (greater than or equal to) 95%.**

Tidal Volume and Dead Space

The total volume of air exchanged at rest when no extra effort is applied in a normal breath is referred to as **tidal volume**. It is approximately 7 ml/kg of body weight or 500 ml per inspiration. Some air that enters with respiration does not actually make it to the alveoli to participate in lung exchange. The volume of this space is referred to as **dead space** and is around 150 ml.

Respiratory Drive

The medulla oblongata in the brain stem controls the involuntary component of respiration. This respiratory center is very sensitive to blood carbon dioxide levels and **pH**, which is a logarithmic measurement of hydrogen ion concentration. **A higher hydrogen ion concentration means increased acidity and a lower pH. Things that bind with hydrogen ions are referred to as buffers and this leads to increased alkalinity and a higher pH.** Increases in hydrogen ion or carbon dioxide concentrations will cause the respiratory center to increase the rate of respiration. Peripheral sensors that control respirations also exist. These chemoreceptors are mainly sensitive to oxygen.
concentration but also to pH and carbon dioxide. The carotid body, one such cluster of chemoreceptors, is located near the bifurcation of the common carotid artery. When oxygen levels in the blood are detected to be low signals are sent to the brainstem to increase respirations. Another cluster of receptors is called the aortic body.

Part 3: The Cardiovascular System

The Heart: Structure

The heart is a fist sized organ that is located in the chest behind the sternum slightly to the left. It is a coned shaped organ with the bottom pointed portion (the apex) facing down and the base facing upward and tilted to the right. It is enclosed in a double walled sac called the pericardium.

In between the heart and the pericardial sac there is a small amount of lubricating pericardial fluid that allows the heart to move freely as it beats. The heart wall has several layers but the majority of its mass is made up of a type of specialized muscle called myocardium. Unique electrical centers and pathways within the heart mean that the myocardium can contract and beat without any outside stimulation. These electrical tissues therefore fire automatically and are said to have what is known as automaticity.

The heart is made up of four chambers. The chambers in the upper heart are called the right and left atria. In the lower heart are the right and left ventricles. The ventricles have approximately three times the volume capacity compared to the atria.
A series of valves keep the blood flowing in one direction. Between the atria and ventricles lay what are called the AV valves. The tricuspid valve (three cusps) separates the right atrium and ventricle, and the mitral valve separates the left atrium and the left ventricle. Another set of valves are associated with the flow of blood as it leaves the ventricles. As blood leaves the right ventricle it passes through the pulmonary valve, and as it leaves the left ventricle it passes through the aortic valve.

The heart has two distinct phases relating to the cardiac cycle. Systole is the contractile phase, a period of myocardial fiber shortening and the ejection of blood from the heart chambers. Diastole is the relaxation phase of the cardiac cycle, a period where the fibers are relaxing and the heart chambers increasing in their volume. This leads to the creation of lower pressures which helps draw blood into each chamber. The filling of the ventricles through open AV valves during diastole, as blood is sucked into the area of low
pressure, accounts for 65-85% of what is called **end-diastolic volume**. The atria enter into systole slightly before the ventricles and they push the remaining 15-35% of the volume in under positive pressure. This topping up of the ventricles is called **atrial kick**. This explains why the volume of the atria is only about 30% that of the ventricles.

The period of time for diastole to take place is called **diastolic filling time**. If heart rhythms occur that cause a heart to beat too fast this time may be so shortened that the chambers cannot be filled before the next systolic contraction. This partially explains why fast heart rhythms can be dangerous.

The heart is often thought of as a distinct right heart and a left heart for ease of understanding. **The right heart receives deoxygenated blood from the body by way of the inferior and superior vena cava.** As the right ventricle has finished systole it relaxes and the volume of the chamber increases creating low pressure. **This sucks the blood in from the vena cava through an open tricuspid valve.** Just before the ventricle contracts, the atria fires first pushing the remaining blood volume in (atrial kick). About a tenth of a second later the ventricle starts to contract (systole) and the building pressure causes the tricuspid valve to close and the pulmonary valve to open. Blood is then ejected into the pulmonary arteries where it travels to the lungs to release its carbon dioxide and to pick up more oxygen. **See Figure 1-13.** In review, the right heart functions to take deoxygenated blood from the venous system of the body and send it out to the lungs to be reoxygenated. The left heart similarly takes oxygenated blood from the lungs and then moves it out to the body to be distributed via the arterial system.

All blood vessels leading away from the heart are called **arteries** and all coming to the heart are **veins**. As blood returns from the lungs back to the heart rich with oxygen it travels down the four **pulmonary veins** into the left atria. **Again most of the flow occurs due to the lower pressure being created in the left ventricle as it increases in volume during diastolic relaxation.** This draws the blood through an open mitral valve from the atria and pulmonary veins. The left atria again contracts slightly ahead of the ventricle adding the atrial kick of 15-30% of the end-diastolic ventricular volume. As the left ventricle then enters systole (contraction) the mitral valve snaps shut and the aortic valve starts to open. Oxygen rich blood is then ejected out into the aorta to travel through the arterial system.

The cardiac cycle of the right and left sides of the heart occurs at the same time. Both atria contract at the same time followed a tenth of a second later by the contraction of both ventricles. **The volumes of blood ejected by the ventricles are equal also under normal circumstances.** Whatever the right sends to the left the left must be able to take and vice versa. So in a healthy heart the chambers are equal in volume (right to left) but they are not equal in thickness and muscle mass. The muscle mass of the left ventricle is approximately three times that of the right ventricle. The answer to why relates to the amount of force required to eject blood from the right and left ventricles.

**Afterload** is the amount of resistance there is to moving blood out of the heart. The bottom number of a person’s blood pressure is referred to as the diastolic pressure. It is the resting pressure in the arteries that the heart has to overcome to push blood out to the body. If a person has a “textbook” blood pressure of 120/80 that means that when the left ventricle contracts it must create a pressure somewhat greater than 80 mmHg to get the aortic valve to start to open. Therefore **diastolic blood pressure is a good indicator of a patient’s afterload.** To understand why the right ventricle has a smaller muscle mass compared to the left is easy when afterload is examined. A blood pressure...
can be measured within the pulmonary artery and the diastolic pressure in a healthy person is only around 10-12 mmHg. That means that the right ventricle only has to create a pressure slightly greater than 12 mmHg to get the pulmonary valve to start opening when it contracts. Less force means less muscle needed to do the work.

**Preload** is another important concept in understanding the heart as a pump. **Preload refers to the amount of stretch, pressure and volume in the ventricle just prior to systole.** Two factors affecting preload are vascular tone and blood volume. If a person has a vasovagal syncope reaction they have fainted partly due to the fact that they have dropped their preload suddenly as the “vaso” of vasovagal refers to the fact that they have lost vascular tone and vasodilated their blood vessels. This leads to the pooling of blood out in the periphery (arms and legs) with less returning to the heart. The “vagal” component leads to a dropping of heart rate at the same time. Combined this leads to inadequate blood flow to the brain and a loss of consciousness.

Like all organs, the heart has its own blood supply. The very first branches off of the aorta are the **right and left coronary arteries** which feed arterial blood to the heart myocardium itself. From there it enters the coronary veins where it empties back into the right atria via the coronary sinus. The heart has a very high oxygen demand and 75% of the available oxygen in coronary blood flow is removed as the arterial blood passes by the cells. In contrast, the kidney only uses 20% of available oxygen in the arterial blood supplied to it. The brain like the heart has very high oxygen demands. **The heart and brain therefore are very sensitive to ischemia. Ischemia is an inadequate blood flow to meet the oxygen demands of an area of tissue.**

In summary, the cardiac cycle results in the ejection of blood from both the right and left ventricles simultaneously. The pressure changes within the chambers causes the valves to open and close keeping the blood moving in one direction only. Even without the contraction of the atria, the ventricles can still eject about 65-85% of their normal volumes. This explains why when the atria are not contracting normally a patient will usually remain stable in terms of their ability to pump blood effectively. The heart ventricles have to overcome resistance to eject blood and this resistance is called afterload. Diastolic blood pressure is a good estimator of afterload. Preload is the “priming” of the pump; how much blood volume and pressure is bringing blood into the heart.

**The Heart: Cardiac Output**

**Cardiac output is the amount of blood ejected from the left ventricle in one minute.** For an adult an average cardiac output is about 5-8 liters per minute. During strenuous exercise this output can rise to over 18 liters/minute. Cardiac output is a product of heart rate and **stroke volume (SV),** the amount of blood ejected with one systolic contraction (beat) of the left ventricle. A normal SV is around 50-80 ml.

\[
CO = SV \times HR
\]

Cardiac output is so vital because it helps maintain the body in aerobic metabolism producing large amounts of ATP for the body’s metabolic needs. Several factors affect cardiac output, one being heart rate. As heart rate slows cardiac output will decrease. With a heart rate of 30 and a stroke volume of 70ml a person’s CO would be 2100
ml/min, less than one half of normal. On the other hand as heart rate increases so will cardiac output. There is a limiting factor though as to how high a person’s heart rate can climb where it is still leading to an improvement in cardiac output. This again relates to diastolic filling time. If the heart rate becomes too fast there is inadequate time to fill the chamber with blood before the next contraction so stroke volume starts to decline as does cardiac output. In many adults cardiac output will no longer increase at heart rates of greater than 150 bpm but will instead begin to drop.

![Figure 1-14: Cardiac Output and Heart Rate](image)

As a general rule, a patient with a heart rate that is too fast (> 150 bpm - not enough filling time), or too slow (<50 bpm - not enough rate) requires assessment for signs and symptoms of shock. Shock is defined as the inadequate perfusion of tissues and organs to allow for continued aerobic metabolism and cellular life. The term inadequate end-organ perfusion is often used. Signs and symptoms of shock include shortness of breath, chest pain, low blood pressure (hypotension), and altered level of consciousness. They are the signs and symptoms produced when any given tissue or organ is not getting enough oxygen.

The healthier a patient is the more of a bradycardia (heart rate less than 60) and tachycardia (heart rate over 100) they can tolerate. On the other hand a patient with an already damaged heart (example would be having had several heart attacks) may not tolerate a heart rate of less than 70 or over 100 without becoming very symptomatic.
Table 1-2: Parameters that Regulate Cardiac Output

<table>
<thead>
<tr>
<th>Parameters that Increase Cardiac Output</th>
<th>Parameters that Decrease Cardiac Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased heart rate up to 150 bpm</td>
<td>Heart rates less than 50 bpm</td>
</tr>
<tr>
<td>Having atrial kick</td>
<td>Loss of atrial kick (caused by certain heart arrhythmias)</td>
</tr>
<tr>
<td>Adequate diastolic filling time</td>
<td>Inadequate diastolic filling time</td>
</tr>
<tr>
<td>Increased preload</td>
<td>Decreased preload</td>
</tr>
<tr>
<td>Decreased afterload</td>
<td>Increased afterload</td>
</tr>
</tbody>
</table>

The Heart: Acute Coronary Syndrome (Coronary Artery Disease)

Many heart problems are related to issues with the coronary vasculature and problems with supplying oxygen rich blood to the myocardium. The primary cause of acute coronary syndrome (ACS) is the formation of plaque in the walls of coronary vessels.

Figure 1-15: Coronary Arteries

Blausen Medical Communications Inc. Wikimedia Commons; 2013.
Angina pectoris is one type of ACS in which there can be adequate blood flow to supply a region of the heart past a plaque deposit, but if the equilibrium between oxygen supply and demand (exercise, anxiety, stress) is impacted then signs and symptoms of ischemia will occur. The vessel does not have the ability to vasodilate open as there is an increased need for oxygen and blood flow so then tissues past (distal to) the narrowing are starved for oxygen. The tissue does not die as long as the equilibrium between oxygen supply and demand is reestablished. This is done by decreasing workload (rest, removal of emotional stressors) and increasing blood flow past the narrowing with drugs such as oxygen and nitroglycerin.

A myocardial infarction (MI) occurs when a coronary vessel is occluded across the full vessel lumen. This is caused when a plaque ruptures or erodes so that a thrombus forms and no blood flow occurs distal to the lesion. The muscle that the vessel feeds is then cut off to the point where myocardial tissue dies. The death of tissue is called necrosis.

Both angina and an MI can cause similar signs and symptoms such as chest discomfort (pain, pressure, heaviness, radiation of pain to other areas), shortness of breath, dizziness, nausea, and vomiting. During an MI rest and nitroglycerin will not relieve the signs and symptoms as it will with angina.
As arteries travel out further from the heart they become smaller and smaller in diameter and are then called **arterioles**. These arterioles then continue to become smaller until they terminate in very small vessels called **capillaries**. It is in the capillaries that the exchange of O₂, CO₂, nutrients and other waste products takes place at the cellular level. As the deoxygenated blood leaves the capillary beds it enters **venules** which combine together to form small veins, then larger veins, then finally the vena cava which takes venous blood back to the right atrium. Arteries are generally located more deep in the tissues whereas veins are much more superficial. **Arteries have much thicker muscular walls than veins and are more able to change their lumen diameter in response to nervous system innervation (connection); called vasoconstriction and vasodilation. Veins have one way valves that help keep blood moving towards the heart.**
Blood Pressure

Diastolic pressure has been discussed in relation to afterload. **Systolic blood pressure** is the peak wave of pressure created by the pulsation of blood flow created by systole as blood is ejected into the aorta. A normal blood pressure is dependent on the age of a patient. It is lower in children and as patient's age it gets higher. A typical textbook blood pressure (BP) is 120/80 mmHg. **A measurement very important in determining if the BP is causing enough pressure to perfuse all of the organs in the body is called Mean Arterial Pressure (MAP).** It is defined as the average arterial pressure during a cardiac cycle and is thought to be the perfusion pressure seen by organs in the body. It is calculated by a mathematical formula and all modern BP machines calculate MAP. A MAP of greater than 60 mmHg is thought to be high enough to ensure good organ perfusion. A normal MAP is between 70 and 110 mmHg.

Heart Rate, Electrical Pathways and Heart Rhythm

The human adult heart at rest beating normally, will have a rate of between 60-100 bpm. This rate can be taken manually as a pulse at key locations in the body. The pulse is a physical expansion of an artery that can be palpated (felt) where arteries run more superficially. Locations include the carotid artery in the neck, the radial artery at the wrist, and using the dorsalis pedis artery in the foot.
The electrical conduction system in the heart allows for the coordination of the impulses generated by the **sinoatrial node** (SA Node) to be propagated to and activate the myocardium in the correct sequence. **The SA node is the dominant pacemaker of the heart.** It is a specialized clump of cells that is located in the right atrium near where the superior vena cava enters the atrium. These cells have cell membranes that leak certain ions and this helps the cells to **depolarize** (fire) on their own in a cyclic repetitive manner. Adjustments for how fast the SA node fires are made by the medulla within the brain stem through the sympathetic and parasympathetic fibers that innervate the SA node.

Heart rhythms that start with impulses coming from the SA-node are called **sinus rhythms.** The electrical activity of the conduction system of the heart can be picked up on the skin of the body through an **electrocardiogram (ECG).** The impulse (wave of depolarization) travels out from the SA node across the right and left atria towards the next specialized clump of conductive tissue called the **atrioventricular node (AV node).**
The AV node can act as a backup pacemaker site if the SA node fails to work. It will fire at a rate of 40-60 bpm if it is not “dominated” by the higher pacemaker site, the SA node. It has another function which is to delay the wave of depolarization from traveling down into the ventricles by 1/10th of a second. This allows the atria to enter into systole slightly ahead of the ventricles so that they can contract and finish filling up the ventricles (atrial kick) before the ventricles enter into systole.

![Figure 1-20: Electrical Conduction Pathways of the Heart](image)

From the AV node the impulse travels down the Bundle of His and penetrates through the fibrous nonconductive tissues that separate the atria and ventricles. These fibers then branch into the right and left ventricles through what are known as bundle branches. The impulses then terminate in millions of micro-sized fibers called Purkinje fibers. The only way in a healthy heart for the atrial depolarization wave to enter into the ventricles is through the bundle of His. The purpose of the right and left bundle branch systems and the Purkinje fibers is to get the ventricles to contract in sync in a very short period of time; all fibers contract nearly simultaneously.

When patients undergo general anaesthesia or sedation they often have ECG monitoring during the procedure. The normal rhythm of a healthy heart under no stress is called a normal sinus rhythm (NSR). Figure 1-21 depicts one complete cardiac cycle.
The horizontal axis of an ECG represents time and the vertical axis voltage. The first wave is called the \textbf{P wave} and this represents the depolarization of the atria. The PR segment is the $1/10^{th}$ of a second delay for atrial kick from the AV node. The \textbf{QRS complex} is the depolarization of the ventricles that starts as the impulses leave the purkinje fibers. The \textbf{T wave} represents repolarization of the ventricles.

![Figure 1-21: The Normal ECG](Atkielski, Anthony. Wikimedia Commons; 2007)

\textbf{Vasculature Important to Dentistry}

After leaving the aorta the first branch on the right is the brachiocephalic artery. It branches into the \textbf{right common carotid artery} and the right subclavian artery. On the left the \textbf{left common carotid artery} branches directly off the aorta. It is the common carotid arteries that are very easy to palpate and pulses here can often be palpated even when a patient’s BP is very low. Just near the angle of the mandible, the common carotids bifurcate into the \textbf{internal carotid artery} and the \textbf{external carotid artery}. The external carotid artery has eight branches and several are important to the dental anaesthesia assistant.
The tongue and the floor of the mouth are supplied by the lingual artery. The facial artery starts behind the angle of the mandible and traverses across the face supplying it with blood. The maxillary artery supplies the maxilla, the teeth, the sinuses, and the nose.

The veins of the head and neck drain venous blood and in general follow similar pathways as the arteries. Superficial veins include the facial vein which drains blood from the face, the nose and the eyelids and then connects with the internal jugular vein. A deep component of the venous vasculature is the pterygoid plexus located behind the maxilla. See Figure 1-23. Local anaesthesia of the maxillary posterior teeth can lead to trauma of the plexus and the formation of a hematoma. A hematoma is a collection of blood outside of a blood vessel in liquid or clotted form.

Of particular importance for veins of the head and neck is the anatomical absence of the one way valves found in most veins. This allows for the tracking of infections backwards all the way to the brain in some instances. Infections of an odontogenic source must be treated aggressively and immediately or a fatal outcome can occur.
Part 4: Other Important Anatomical Systems and Organs

Immune System

The human immune system is a group of biological processes and structures that protect against disease. It protects through a series of layered defenses starting with surface barriers such as mucous membranes and skin. If a pathogen breaks through these barriers then the next response is from the innate immune system. It is known as a nonspecific immune system response that does not confer long lasting immunity for the host. Cells of the innate immune system recognize and respond to pathogens in a generic nonspecific way. Cells of the innate immune response include mast cells, phagocytes, basophils, eosinophils, and natural killer cells. The adaptive immune system is only found in vertebrates and it adapts its response to improve its recognition of pathogens. This response is retained in an immunological memory and each exposure to the same pathogen elicits a stronger response. Cells of this system include lymphocytes, killer T cells, and antibodies.
Endocrine System

Pancreas

The **Islets of Langerhans** are located in the pancreas. They are responsible for the secretion of insulin and glucagon. Insulin is the moderator of the uptake of glucose by the cells of the body and it keeps the blood sugar levels stable.

Adrenal Glands

Adrenal glands are endocrine glands that sit on top of the kidneys. They function in unison with the sympathetic nervous system releasing hormones in response to stress. These include corticosteroids such as **cortisol** and catecholamines such as **epinephrine and norepinephrine**. It is divided into two parts; the medulla and the cortex. The cortex is vital to the production of corticosteroids and androgen hormones. **Aldosterone** is one hormone responsible to long term control of blood pressure produced in the cortex. The cortex is also responsible to the body's control of carbohydrate metabolism and electrolyte balance. **The medulla secretes approximately 20% norepinephrine and 80% epinephrine and is the main source of the bodies circulating catecholamines, hormones critical to the fight or flight response.**

![Adrenal Gland](image)

**Thyroid Gland**

The thyroid gland is a u-shaped gland found in the anterior region of the neck. It secretes hormones such as thyroxin that regulates the body’s basal metabolic rate. If there is too much hormone released it is called hyperthyroidism or **Graves' disease**. If too little is released the result is myxedema. This gland can become much enlarged if there is a lack of iodine and is called **goiter**.
The Liver

The liver is the largest single organ in the human body. It has a variety of functions including protein synthesis, blood detoxification, and the production of biochemicals needed for digestion. The liver is responsible for over 500 separate functions. This includes protein synthesis, carbohydrate metabolism, lipid metabolism, coagulation factor production, red blood cell production, growth hormone production, storage of glycogen, the breakdown of toxic substances (many drugs are broken down by the liver), and the storage vitamins A, E, D, K and B12.

The Kidneys

The kidneys function to regulate body fluid composition and volume. They maintain homeostatic functions such as the regulation of electrolytes, acid-base balance, and blood pressure. They also serve as the body's main way to remove body toxins. Many drugs are broken down and excreted by the kidneys. The kidneys are also responsible for the reabsorption of water, glucose, and amino acids.

Module 1 Quiz

The module 1 quiz can be found at the following web address: https://testing.exambuilder.com/login

Contact Michael Dare at mmdare@dentaed.ca if you did not receive a student ID and password
Module 2: The Anaesthetic Spectrum of Care

Key Areas for Study

- Optimal characteristics of an Anaesthetic
- Balanced anaesthesia
- Types of anaesthetic care
- Stages of GA
- Levels of sedation
- The sedation continuum
- Indications for PSA
- Dental anaesthetic techniques
- Types of local dental anaesthesia
- Intravenous balanced anaesthesia
- Inhalation anaesthesia

Anaesthesia refers to the condition of having sensation removed or temporarily blocked. It is induced pharmacologically and can include amnesia, analgesia, unconsciousness, a decreased stress response, and the loss of skeletal muscle reflexes. Also under the umbrella of anaesthesiology are the realms of sedation and pain control. Anaesthesiologists are no longer limited to work in an operatory setting only. Anaesthesiologist can specialize in anaesthesia for special types of surgery (cardiac, obstetrical, thoracic, or pediatric), in regional anaesthesia, acute and chronic pain management, or in intensive care medicine.

This course focuses on outpatient office based anaesthesia, sedation and analgesia. It may involve care guided by an anaesthesiologist (usually the provision of general anaesthesia and deep sedation) or that provided by a dentist or oral maxillofacial surgeon who takes on the dual role of maintaining anaesthetic-sedative care with the help of his or her team while undertaking the dental/oral maxillofacial surgery itself. This dual role of undertaking the surgical care as well as the anaesthetic care is unique and not without certain challenges.

Part 1: The History of Anaesthesia

The advent of surgical anaesthesia did not occur until the mid-1800s. Two dentists in the 1840s Horace Wells and William T.G. Morton introduced the use of two gases, ether and nitrous oxide to the world as anaesthetics that might aid in surgery. Ether went on to be the primary anaesthetic agent used in surgery for the next century.

During the last half of the 19th century and into the early 20th century high concentration nitrous oxide was the main anaesthetic agent used in oral surgery. This was replaced in the 1930s by Sodium Pentothal which then became the dominant agent used until the 1960s. Sodium methohexital (Brevital) was introduced at that time and it remained the primary anaesthetic used until 2002 when a shortage of Brevital occurred. Propofol
started to be used as an alternative and most practitioners did not go back to Brevital when it again became available due to the much more optimal properties of propofol.

In current oral maxillofacial surgeries the most common agents used are propofol in conjunction with fentanyl and midazolam. Ketamine is also used in some practices. Since propofol can cause not only sedation but also full general anaesthesia it is limited in use to oral maxillofacial surgeons or to dentists who are working with an anaesthesiologist in the operatory. In most jurisdictions dentists are limited to moderate parenteral (IV) sedation using agents such as midazolam and fentanyl.

Part 2: The Goals of Anaesthetic Care

The general goals of anaesthesia are to provide a safe, comfortable and pain-free environment for the patient to undergo surgery. It may involve having a deep level of unconsciousness produced or inducing sedation with amnesia where the patient still maintains all of their own bodily functions such as breathing.

Optimal Characteristics of an Anaesthetic

- Have a desirable action- analgesia, disassociation, amnesia, sedation.
- Short term use should be non-addictive
- Non-toxic in nature
- Minimal allergenicity
- Non-flammable
- Have no negative impact on the cardiovascular system such as causing hypotension

Balanced Anaesthesia

Single agents (drugs) rarely have all the characteristics desired to obtain optimal anaesthesia. Balanced anaesthesia relies on the use of several agents to create a combination which takes from the best aspects of each agent. The components of balanced anesthesia include:

A. Analgesia

Providing relief from pain is a cornerstone to providing sound dental surgical care. This has traditionally been done in most dental practices by the use of local anaesthesia infiltration to block key nerves in the surgical area. As more involved procedures are undertaken such as occurs with oral maxillofacial surgery the addition of IV narcotics such as fentanyl can be added.

Local anaesthetics are cell membrane stabilizers; they reversibly stabilize and decrease the rates of depolarization and repolarization of excitable cells such as nerve cells. They often do this by inhibiting sodium influx by blocking sodium ion channels in the neuronal cell membrane. This stops a neuron or group of neurons from being able to fire so that pain signals cannot be transmitted. Marcaine and lidocaine are examples of sodium channel blockers used in local anaesthesia.
B. Amnesia
Certain drugs facilitate the creation of amnesia, the loss of memory. Agents like propofol have strong amnesic properties as do the benzodiazepines. Midazolam is a common short acting benzodiazepine used in sedation.

![Sodium Channels in a Cell Wall Membrane](image_url)

C. Relaxation and Immobility
Often surgical procedures necessitate that the patient be relaxed and even hindered immobile. Muscular paralytics, benzodiazepines, and propofol are some examples of drugs that do this.

D. Creation of Unconsciousness
Depending on the length and type of surgical procedure being done it may be desirable to create unconsciousness. Propofol in higher dosages is the most commonly used agent used at this time.

Other Optimal Characteristics of Anaesthetics

A. Be cost effective.
B. Easily administered.
C. Work well across all age groups.
D. Provide for rapid smooth and comfortable induction.
E. Be free of postoperative side effects such as nausea and vomiting.
F. In some instances allow the patient to retain protective gag reflexes and spontaneous breathing.
G. Rapid recovery and discharge.

Part 3: The Types of Anaesthetic Care

General Anaesthesia (GA)

GA is a medically induced coma with a loss of protective reflexes (ability to breath, maintain proper airway positioning on own, prevent aspiration with own gag reflex). This can be achieved with a variety of medications to ensure deep unconsciousness,
amnesia, analgesia, loss of control of the autonomic nervous system, and the relaxation or paralysis of skeletal muscles.

Stages of GA

A. Premedication
   Prior to the induction into GA a variety of medications can be given such as antiemetics, antihistamines, beta blockers, analgesics, and benzodiazepines to help obtain a balanced anaesthesia. Midazolam and propofol have amnesic properties but no analgesia so an opioid like fentanyl may be given. There may be synergistic effects with the medications given; the use of two or more medications together produces effects that are different or greater than the sum of their individual effects.

B. Induction
   This is the phase where agents are given and a loss of consciousness occurs. In modern anaesthesia this is done by either an intravenous route or by inhalation of a volatile anaesthetic such as Sevoflurane. Commonly used IV agents used include propofol, sodium thiopental, etomidate, and ketamine.

C. Maintenance
   Many anaesthetics have durations of action of only five to ten minutes. To maintain anaesthesia for the duration of a surgery the patient requires an ongoing controlled infusion of a drug such as propofol or the continued controlled inhalation of volatile anaesthetic gases.

D. Emergence
   Emergence refers to the return to baseline physiologic function for the organs and tissues of the body as anaesthetics are discontinued.

E. Postoperative care
   This care transitions the patient from anaesthesia to the post-surgical recovery and pain management.

Regional Anaesthesia (RA)

Regional anaesthesia is anaesthesia to a large area of the body such as a limb or to the lower half of the body. There are two categories of regional anaesthesia; central and peripheral regional anaesthesia.

Central Techniques

Neuraxial Blockade: The use of local anaesthetics placed around the nerves of the CNS such as spinal anaesthesia and epidural anaesthesia. More caesarean sections are now done under RA than GA.

Peripheral Techniques

These include plexus blocks and single nerve blocks where a local anaesthetic with epinephrine, a steroid, and/or an opioid is injected into a nerve or nerve plexus. As an example, various brachial plexus blocks exist for shoulder and arm surgeries.
Local Anaesthesia

Infiltration of local anaesthesia is a backbone of pain management in general dentistry. Local anaesthesia, in a true sense, is the anaesthesia of a small part of the body such as an area of skin or a tooth.

Procedural Sedation

Procedural sedation refers to a technique of administering sedatives or dissociative agents with or without analgesics to induce a state that allows the patient to tolerate unpleasant procedures while maintaining cardiorespiratory function. The objective is that the patient experiences a depressed level of consciousness but still maintains oxygenation and airway control independently. The vast majority of anaesthetic care that is done in dentistry beyond local anaesthesia is done in the realm of moderate to deep sedation.

Levels of Sedation

The American Society of Anaesthesiologists (ASA) defines the levels of sedation as follows:

A. Minimal sedation (anxiolysis) – a drug-induced state during which patients respond normally to verbal commands. Although cognitive function and coordination may be impaired, ventilatory and cardiovascular functions are unaffected.

B. Moderate sedation/analgesia (conscious sedation) – a drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.

C. Deep sedation/analgesia – 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 ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.

D. General anaesthesia – a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function 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. Cardiovascular function may be impaired.

E. Dissociative Sedation. The American College of Emergency Physicians also defines a level of sedation called dissociative sedation which is a trancelike cataleptic state characterized by profound analgesia and amnesia with retention of protective airway reflexes, spontaneous respirations and cardiopulmonary stability. Ketamine is the only approved dissociative agent.
Background History of Sedation

The practice of sedation has evolved significantly over the last several decades. Once under the direct control of anaesthesiology with legal responsibility for all sedation falling upon this specialty, expertise in procedural sedation is now part of the core competencies of several physician and dental specialties. As studies demonstrate the safety of performing various levels of sedation in both hospital and non-hospital settings, the scope of practice of providers has expanded and the demand for different procedures to now include sedation has grown.

Table 2-1: Continuum of depth of sedation.

<table>
<thead>
<tr>
<th></th>
<th>Minimal Sedation (Anxiolysis)</th>
<th>Moderate Sedation or Analgesia (Conscious Sedation)</th>
<th>Deep Sedation or Analgesia</th>
<th>General Anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Responsiveness</td>
<td>Normal Response to Verbal Stimulation</td>
<td>Purposeful* response to verbal or tactile stimulation</td>
<td>Purposeful response after repeated or painful stimulation</td>
<td>Unarousable, even with painful stimulus</td>
</tr>
<tr>
<td>Airway</td>
<td>Unaffected</td>
<td>No intervention required</td>
<td>Intervention may be required</td>
<td>Intervention often required</td>
</tr>
<tr>
<td>Spontaneous Ventilation</td>
<td>Unaffected</td>
<td>Adequate</td>
<td>May be inadequate</td>
<td>Frequently inadequate</td>
</tr>
<tr>
<td>Cardiovascular Function</td>
<td>Unaffected</td>
<td>Usually maintained</td>
<td>Usually maintained</td>
<td>May be impaired</td>
</tr>
</tbody>
</table>

Recent technological and pharmacological advances have drastically changed the practice of sedation. **Pulse oximetry** was developed during World War II but did not evolve into a common tool used in medicine until the early 1980’s. **It revolutionized the safety of procedural sedation.** By measuring the hemoglobin oxygen saturation, a wealth of information was provided to the clinician. Later **capnography**, the continuous monitoring of exhaled CO2, became the standard of care for all patients undergoing general anesthesia. ASA established it as a standard of basic anesthetic monitoring in 1999. In 2010 ASA amended standards for basic anesthetic monitoring and included a statement relating to monitoring exhaled CO2 during sedation. “During moderate or deep sedation the adequacy of ventilation shall be evaluated by continual observation of qualitative clinical signs and monitoring for the presence of exhaled carbon dioxide unless precluded or invalidated by the nature of the patient, procedure, or equipment.” Capnography and pulse oximetry together could have helped in the prevention of 93% of avoidable anesthesia mishaps according to a closed claim study done by ASA.

Pharmacological advances have led to several milestones in the evolution of sedative medications. **Early drugs generally had a slower onset of action and a prolonged duration of action along with having many undesirable side effects.** Their use exposed patients to increased risks in comparison to modern agents, for example long exposure to sedative action and associated risks such as nausea, vomiting and airway compromise. One of the most common modern sedative medications is **midazolam. It has a quick onset of action, a short duration of action, and a very favorable safety profile.** Although it
lacks analgesic properties it provides very good amnesia when dosed correctly. Currently the opioid of choice for many practitioners to provide analgesia is fentanyl. It also has a short duration of action and a rapid onset of action. Propofol can be used to induce general anesthesia, but subhypnotic doses have benefits of titratable sedation with rapid onset/offset. Its antiemetic properties are especially beneficial in the ambulatory setting, but it should be noted that it lacks analgesic effects.

The Sedation Continuum

Although the intent is to maintain the patient in a state of self-maintained cardiopulmonary function, the medications used and the patient's response to them may result in a deeper sedation level than intended. This places the patient at risk for airway and cardiopulmonary compromise. The important concept to remember here is that sedation is a continuum. The patient response may be unpredictable despite careful titration and monitoring. ASA states that if a patient's level of sedation progresses to a stage that is deeper than originally planned, the practitioner should be able to rescue the patient from the deeper level of sedation. For example, individuals who administer moderate sedation/analgesia (formerly known as “conscious sedation”) should be able to rescue patients who enter a state of deep sedation/analgesia, and those administering deep sedation/analgesia should be able to rescue patients who enter a state of general anaesthesia. Practitioners must be able to respond with the appropriate knowledge and skills to handle these emergencies.

Indications for PSA

A procedure doesn’t have to be painful to induce fear and anxiety in our patients. Previous experience with a noxious procedure or fear of the unknown can all contribute to how well the patient is able to tolerate our interventions.

Goals for IV Procedural Sedation

A. Provide the patient with a safe environment where a painful or unpleasant procedure is required.
B. Alleviate patient anxiety.
C. Minimize physical discomfort.
D. Maximize amnesia.
E. Control motor behavior and movement if necessary so as to perform painful/unpleasant procedures.
F. Minimize the risk of the procedure, and ensure safe discharge of the patient.

Potential Indications for Procedural Sedation and Analgesia in Dentistry

A. Anxiety and fear
A. Increased gag reflex
B. Prolonged or unpleasant treatments
C. Increased tooth sensitivity
D. Special needs patients
E. Movement disorders
Part 4: Dental Anaesthetic Techniques

Dental anaesthesia is a field of anaesthesia that includes several anaesthetic techniques such as local anaesthesia, PSA, and GA. The options chosen will depend on many factors such as the patient’s age, anxiety level, medical conditions, and the type of procedure being performed. Often several approaches are combined to give the most balanced anaesthetic care.

Dental Local and Regional Anaesthesia

The most commonly used local anaesthetic used in dentistry is **lidocaine**. It is a sodium channel blocker that has a **half-life** (the time for one half of the drug to be cleared or made inactive) of one and a half to two hours. Other modern agents include **articaine**, **marcaine** (a longer acting agent), and **mepivacaine**. Most come with and without epinephrine or another vasoconstrictor. **The addition of epinephrine has two purposes: it prolongs the duration of action of the local anaesthetic by slowing diffusion, and it can be used to decrease bleeding in a surgical area by vasoconstricting blood vessels.**

Types of Local Dental Anaesthesia

A. **Regional Nerve Blocks**

   The inferior alveolar nerve block quite possibly is the most anaesthetized nerve in the body. This nerve innervates the mandibular teeth, the chin, lower lip, and parts of the tongue. The lingual nerve is often anaesthetized with the same injection so the tongue and floor of the mouth are anaesthetized also. The superior alveolar nerves are usually not blocked directly as they are difficult to reach with a needle. **The maxillary arch is usually anaesthetized instead by inserting a needle below the oral mucosa surrounding the teeth so as to anaesthetize smaller branches of the nerve.**

B. **Infiltration**

   Anaesthetic given inferiorly to the root of the tooth to be worked on. Good for only minor dental procedures such as a dental restoration.

C. **Palatal Block**

   The greater palatine nerve block is useful for anesthetizing the palatal soft tissues. It is indicated when palatal soft tissue anesthesia is necessary for restorative treatment on more than two teeth and for periodontal and oral surgery.

D. **Intraosseous**

   Intraosseous anaesthesia is the injection of anesthetic agent into the cancellous bone surrounding the teeth being treated.

E. **Intapulpal**

   Injection of local anaesthesia into the pulp of the tooth to completely anaesthetize the tooth

F. **Pressure anaesthesia**

   Pressure with a cotton swab is applied in the area to distract the nerve sensation of pain when the needle enters.
Local Anaesthesia with Procedural Sedation and Analgesia

The combination of local anaesthetic techniques with intravenous sedation and analgesia is a very common method of balanced anaesthesia in the modern dental/oral surgery practice. A popular combination of agents is fentanyl and midazolam so that there is systemic analgesia and amnesia to the operatory experience. Often the goal is to achieve moderate sedation with local anaesthesia of the surgical field.

Nitrous oxide can also be used in conjunction with local anaesthesia for some patients. It is delivered through a nasal mask or cannula with oxygen concentrations of at least 30%. Following its use the patient must receive 100% oxygen for three to four minutes to avoid diffusion hypoxia which is when nitrous oxide rapidly leaves the bloodstream back into the alveoli of the lungs and displaces other gases including oxygen. The recovery from nitrous oxide usually takes a minimum of 15 minutes. Disadvantages of inhalation sedation include the need for expensive specialized equipment, the need to scavenge (collect) trace gases, and their potential risk to staff.

Intravenous Balanced Anaesthesia

Oral Maxillofacial Surgery (OMG) and General Dentistry often require a patient to be deeply sedated and/or unconscious (under GA). The equipment, protocols, and training of the team must be extensive to be able to effectively deliver deep sedation or GA in a safe manner in an office setting.

As a patient experiences a balanced anaesthesia they may go through many levels of anaesthetic care. They may receive a mild oral anxiolytic sedative that they are to take prior to arriving for their appointment. Such medications include triazolam, diazepam, and midazolam. The patient must have followed NPO (nothing by mouth) rules for the hours prior to the surgery; that is nothing to eat or drink for eight hours prior to the surgery other than clear fluids which can be consumed for up to two to four hours prior to
surgery. **Clear fluids** include water, tea, coffee, and apple juice. They do not include milk products or juices with pulp.

Prior to the IV insertion, the patient might receive a topical anaesthetic such as EMLA cream at the selected IV insertion site. Modern IVs use a plastic catheter over a needle with only the plastic catheter staying in place after insertion. Common locations for starting the IV include the back (*dorsum*) of the hand, the forearm, or the **antecubital fossa**. This is an area at the crease of the elbow.

**Prior to induction an IV opioid analgesic and anxiolytic may be given.** Next induction is undertaken with an IV agent such as propofol, methohexital, and/or ketamine. During the surgery a continuous infusion of an IV solution is given at a rate that just keeps the vein open (TKVO), the flow keeps the IV catheter tip from clotting off. A solution of 9% NaCl (sodium chloride) called *Normal Saline* or an electrolyte balanced solution called *Ringers Lactate* is used.

**With the ability to provide IV sedation and GA, the local anaesthetic injections are much better tolerated by the patient.** If adequate local anaesthesia is achieved then the maintenance and emergence phases of anaesthetic care are generally uneventful. If not then heavier doses of anaesthetics tend to be given to overcome the inadequate pain control.

**One of the biggest negative side effects of propofol is respiratory depression and hypotension.** Local anaesthetics with epinephrine can offset this depression. A common combination is to give ketamine and propofol together at lower doses (*synergistic action of two drugs together*). Ketamine is one of only a few anaesthetics that have the property of increasing blood pressure so that when given with propofol it overrides the hypotensive properties of the propofol.

During the surgery a level of anaesthesia which is in the realm of being deep sedation bordering on GA is maintained usually by way of a continuous infusion of the main anaesthetic agent chosen. This can be achieved through **small incremental boluses** (small doses frequently given by syringe into a port on the IV line) or by use of an **infusion pump** like the syringe pump shown in Figure 2-3.

**When the procedure is completed it is desirable to have the patient recover quickly with minimal side effects post operatively.** Older drugs often had a long half-life and the patient remained sedated for prolonged periods of time. Also undesirable side effects such as nausea and vomiting were common. When short acting drugs such as propofol, ketamine, and remifentanil are used the patient usually awakens quickly after the drugs are discontinued. The patient’s reflexes are still depressed so they must be carefully monitored until discharge.

**Inhalation Anaesthesia**

Historically the trend for advanced dental anaesthetic care has been to use an intravenous approach for the delivery of medications. In the early 1990s a newer inhalation anaesthetic was introduced called *sevofluorane*. It had much more desirable characteristics than its predecessors such as a rapid onset of emergence when the gas is discontinued, a lack of myocardial irritability, a pleasant smell, and no airway irritability. Like all inhalation agents it does put the patient at risk for a life threatening emergency...
called *malignant hyperthermia (MH)*. Practices using inhalation anaesthetics must have a MH kit and protocol in place to treat the emergency. A drug called *dantrolene* is used to treat MH.

![A Modern Syringe Pump](image)

**Figure 2-3: A Modern Syringe Pump**

**Module 2 Quiz**

The module 2 quiz can be found at the following web address:
[https://testing.exambuilder.com/login](https://testing.exambuilder.com/login)

Contact Michael Dare at *mdare@dentaled.ca* if you did not receive a student ID and password
Module 3: Anaesthetic Pharmacology and Administrative Techniques

Key Areas for Study

- Pharmacokinetics and pharmacodynamics
- Routes of Administration
- Nerve impulse transmission
- Main CNS target areas for anaesthetics
- Narcotic Receptor sites
- Key drugs
  - Propofol
  - Ketamine
  - The amides
  - Epinephrine
  - Midazolam
  - Fentanyl
  - Naloxone
  - Flumazenil
  - Gravol
- Fentanyl and midazolam combination
- Propofol and Fentanyl in combination

Part 1: The Absorption and Action of Drugs

Two common terms of pharmacology are **pharmacokinetics** and **pharmacodynamics**. These terms are used to describe the actions of a drug on the body but also what the body does to the drug.

**Pharmacokinetics**

Pharmacokinetics refers to what effects the body has on the drug. For a drug to work it must be absorbed into the body, be carried to the site of action, be eventually metabolized so that the effect of the drug ends, and to be eliminated from the body.

Drug absorption can occur in many ways. Oral medications must be dissolved and absorbed by the stomach or the intestines. **Intramuscular** (IM) injections can get a drug into circulation quicker as the drug molecules move into the bloodstream. The most common and effective method for administering anaesthetic medications is by the **intravenous route** (IV). It also allows for the most accurate titration of the drug to get the desired action.

After a drug has been absorbed, it must travel to the site of action. Often this is done through the bloodstream. Sometimes a drug is acting at the site of absorption itself.
Topical anaesthetics such as ELMA work in this way as do drugs injected into a targeted site such as a nerve block or the injection of a steroid into a joint. Drugs like ketamine, propofol and midazolam work centrally on the brain to produce their actions. Anaesthetic agents exert their effects by attaching to receptor sites on neural cell membranes where they alter the movement of ions through what are called ion channels. Drugs that are long acting will stay attached to these receptors for longer periods of time before the drug redistributes to other areas of the body. A drug like propofol has a rapid redistribution profile so that its action is short in duration because the drug only attaches to receptors for a short period of time before being redistributed to other tissues like fat and muscle.

Most anaesthetic medications used are broken down (metabolized) by the liver. The metabolites of most drugs are not pharmacologically active (they have no action). Some metabolites however are pharmacologically active and the metabolites have actions of their own that may be desirable or not. Patients with a history of liver disease may not be able to metabolize drugs normally and this can lead to prolonged or shortened action for a drug.

Once the drug has been cleaved into metabolite subunits the body will eliminate it. Much elimination is done through the kidneys and the urine. Inhalation agents are often eliminated out through the lungs. Some elimination can occur through the GI tract and feces.

Pharmacodynamics

Pharmacodynamics refers to the effects a drug has on the body. Drugs can have actions which are the primary and secondary in nature. The primary action or effect of a drug is the desired action for which we are using it for. Secondary actions are the side effects which are actions that are generally unwanted. In balanced anaesthesia the drugs used in combination can actually be used to minimize the overall side effects. Ketamine and propofol used together is again a good example. The side effect of hypotension from propofol is cancelled out by the side effect of hypertension from the ketamine giving this drug combination a relatively neutral hemodynamic profile.

The duration of action is the amount of time that a drug is effective and producing its desired action. The therapeutic window is the dosage range of a medication that gives the desired effect. Anaesthetic drugs can have dangerous unwanted side effects when given in amounts outside their therapeutic range. If a drug has a narrow therapeutic window it must be administered with great care. When a drug has a wide therapeutic safety window, this means that the dosage can be varied greatly before an undesirable and possibly dangerous side effect occurs. For example propofol has a narrow safety window for causing respiratory arrest in comparison to midazolam.

Routes of Drug Administration

By Mouth (PO)

This route of administration is acceptable for many medications but when powerful sedatives, analgesics, and anaesthetics are being given in the operatory issues of absorption, delayed absorption, and highly variable absorption make this route
unacceptable. Using a PO route is good for ongoing pain management post operatively, antibiotic administration, and anxiolytic administration preoperatively.

Figure 3-1: A Chemically Gated Channel and Receptor Sites
Blaus, Bruce. Wikimedia Commons: 2013.
**Topical**

This route can be used favorably for certain medications, but few are used in OMS. The advantage of this route is that the drug is passed into systemic circulation without passing through the liver first. Topical anaesthetics used before venipuncture or local anaesthetic injections are some of the few uses of the topical route in OMS.

**Subcutaneous (SQ)**

This route uses the injection into fat tissues located below the skin. Medications given this way have a slower rate of absorption when compared to IM or IV administration. Very few drugs used in anaesthesia are given by this route.

**Rectal**

This route is highly vascular so medications placed rectally can be very rapidly absorbed. Most patients consider this route to be unpleasant so it is not often used. The rectal route has applications when a patient is very nauseated and cannot take an antiemetic medication PO. In the OMS/Dental sedation setting there is usually an IV in place so that is the preferred route to give antiemetics such as Gravol or Ondansetron.

**Intramuscular (IM)**

Like other routes mentioned this route has variable absorption and onset of action. In addition the dosage of the drug cannot be titrated over a period of time.

**Intravenous (IV)**

This route is the main route of anaesthetic drug administration due to several distinct advantages over other routes.

- **Rapid route of administration**
- **Rapid onset of action**
- **Easily titrated**
- **Nearly all emergency drugs are given IV**
- **Blood can be easily taken from an IV site to do tests like a blood glucose level**

**Intranasal (IN)**

The injection of fine droplets of a drug into the nares of a patient has gained popularity especially for use in pediatric sedation. **The droplets are atomized (misted into micro droplets) as they are injected into the nares.** A drug molecule can be transferred quickly across the single epithelial cell layer of the nasal passage directly to the systemic blood circulation with an absorption profile very similar to IV. The drug misses what is called first pass liver and intestinal metabolism. Many analgesics, sedatives and anaesthetics can be given this way including midazolam, fentanyl, and ketamine.
Inhalation Route

The inhalation route provides very rapid and titratable administration of anaesthetics. These anaesthetics are for GA use with the exception of nitrous oxide. Volatile gases are generally reserved for the operating room environment of a hospital. Sevoflurane has begun to enjoy some usage in office settings though most inhaled GA requires management of the patient’s airway and breathing to be artificially maintained.

![Figure 3-2: Intranasal Atomization Device](image)

Part 2: Anaesthetic Actions and the CNS

Mechanism of Action

The mechanism of action of many anaesthetics is not well known. In the last 50 years much progress has been made in understanding how anaesthetics exert their effects. Many anaesthetics exert their action by influencing the movement of ions through ion channels in the membranes of neurons.

Nerve Impulse Transmission

Nerves conduct their impulses through electrical means. Various charged ions are found on the inside and outside of neuron cell membranes in various concentrations so that at rest **the outside (extracellular) of a nerve membrane is positively charged in relation to the inside (intracellular)**. As a cell is triggered to fire, certain ions such as sodium (Na+) move through channels and rapidly enter the cell. This changes the outside positive charge to a negative one. **This process is called depolarization.** After a wave of depolarization travels down a cell that cell begins to pump ions back to reestablish its
resting state. This is called repolarization. Many local anaesthetics exert their action by blocking Na+ channels thus slowing or blocking nerve transmissions altogether.

**The Nerve Synapse**

The junction that occurs between two nerves is called a synapse. Neurons can also form connections (synapses) with other types of cells such as muscle fibers as well as directly with the blood stream and intracellular fluid. As a neuron terminates at a synapse it forms a bulge called a terminal button. The nerve before the synaptic gap is called the presynaptic nerve and the one after the post-synaptic nerve. The gap between the nerves at the synapse is called the synaptic cleft. To transmit the impulse across the synaptic cleft the presynaptic neuron releases neurotransmitters which cross the gap and attach to receptor sites on the post-synaptic neuron. These receptor sites are located on the tops of ion channels or gates. The neurotransmitter attaches to these receptors and activates the gates to open. This in turn starts a new wave of depolarization in the post-synaptic neuron.

Most of the intravenous, inhalation drugs and local anaesthetics used act through the alteration of nerve conduction. They modify the action of neurotransmitters and/or the movement of ions through neuron membranes.

![Figure 3-3: Neuron Chemical Synapse](National Institute of Aging, Wikimedia Commons; 2009)
Where Anaesthetics Act in the CNS

There are several areas within the central nervous system where the various drugs used in balanced anaesthesia act.

The Wakefulness System

There is no distinct sleep/wake promoting centers within the brain. The function is undertaken by several interconnected neuronal systems. The system has at its core the brainstem from which radiate outward neurons that innervate the cerebrum. The reticular activating system (RAS) is composed of several neuronal circuits connecting the brainstem to the cerebral cortex. When these circuits are intact the brain is in a state of wakefulness. **When hypnotic (sleep inducing) drugs are used to interrupt these pathways a patient loses consciousness. Drugs such as propofol work in this manner.**

The Centers of Emotion; the Limbic System

The emotions such as calmness, rage, anger, anxiety and sadness are dependent on a group of structures called the **limbic system.** The balance between the emotions is often upset when a patient is faced with the uncertainty of surgery. **Anxiety reducing (anxiolytic) medications such as midazolam targets these centers.**
The Relay Center for Sensory Inputs; the Thalamus

All inputs coming into the brain from sensory neurons pass through the thalamus located at the top of the brainstem. Some of its functions include the relaying of sensory and motor signals to the cerebral cortex, and the regulation of consciousness, sleep, and alertness. The use of drugs like ketamine, block the relaying of sensory information to the cortex. This disconnection of the brain from all sensory inputs creates a dissociative state.

Narcotic Receptor Sites

Opioids drugs exert their action at three levels. They affect transmission of painful impulses from the site of injury itself; they affect transmission within the spinal cord; and finally they affect receptor sites in the brain. The general feeling of euphoria often felt with the use of opioids is attributed to its central brain actions. Drugs like morphine, and fentanyl work by attaching to narcotic receptor sites and blocking pain impulses.

The Vital Centers of the Pons and Medulla

Most anaesthetic agents and narcotics have undesired side effects to some degree that target the centers in the brain that control the autonomic functions for respirations and circulation. The pons and medulla oblongata in the brainstem maintain homeostasis for circulatory and respiratory function and many analgesics, sedatives, and anaesthetics tend to alter that state. The common side effect of these drugs is to produce hypotension and respiratory depression. One exception is ketamine which is a sympathetic stimulant.

Part 3: Anaesthetics and Ancillary Medications Used in OMS and General Dentistry

Pharmacology Basics

- **Agonist**: a drug that interacts and binds with a receptor site and activates that receptor.
- **Antagonist**: a drug that binds with a receptor site without activating the receptor while simultaneously blocking an agonist from activating the receptor.
- **Synergism**: the effect of two drugs given together is greater than the sum effect of the two drugs. This is often seen when opioids and benzodiazepines when they are used in combination.
- **Pharmacokinetics**: refers to the properties such as onset of action and duration of action of a drug. It describes the absorption, distribution, metabolism and elimination of a drug. This is affected by the route of administration.
- **Pharmacodynamics**: refers to the responsiveness of receptors to a medication and the mechanism through which these effects occur.
- **Volume distribution**: the distribution of a medication between plasma and the rest of the body when a dose is administered. It is influenced by the characteristics of the drug such as lipid solubility, binding to plasma proteins, ionization rate, and molecular size.
- **Elimination half-life**: the time it takes for plasma concentration of a drug to reach 50% of its original concentration.

### Intravenous Balanced Anaesthesia

### IV General Anaesthetic Agents for Office Practice

#### A. Propofol

Propofol started to replace barbiturates in the 1980s and 90s because of its more desirable drug profile. Like barbiturates it is rapidly metabolized and redistributed but it also has antiemetic properties. It does not cause laryngospasm, and the patient wakes up rapidly feeling very well. Propofol is insoluble in water so it is mixed in a water-soluble emulsion that includes egg lecithin, soybean oil, and glycerol. The emulsion supports bacterial growth and is perishable so Propofol must be used within six hours of opening a vial.

Propofol is very lipid soluble and it is rapidly redistributed to fat, muscle, and skin within minutes of injection. It is metabolized mainly by the liver in a rapid fashion while some metabolism occurs in the lungs. Moderate liver dysfunction does not seem to effect the drugs action or duration of action. The metabolites are inactive and are rapidly eliminated by the kidneys.

At lower infusion rates propofol acts as a sedative (moderate or deep sedation), but at higher dosages it induces full GA. The therapeutic window for sedation is narrow so it is used by mainly by practitioners with anaesthesia training. It has anticonvulsant properties, and it decreases intracranial pressures and cerebral blood flow. Propofol's primary side effect on the cardiovascular system is to decreases arterial blood pressure through inhibition of the sympathetic nervous system. This leads to blood vessel vasodilation. The problem is accentuated in the elderly. Hypotension can be minimized my avoiding rapid administration of large doses especially in the elderly. Heart rates in patients may show a mild bradycardia during the first 10 minutes of the infusion.

Propofol is a respiratory depressant especially at induction dosages and apnea (cessation of breathing) can occur after administration. Propofol blunts the response to high CO2 levels (hypercarbia) in the blood stream. After discontinuation of propofol, respiratory physiology returns to normal within approximately 30 minutes. Unlike earlier barbiturate agents propofol is a bronchial relaxant and it can lead to a decrease in the incidence of intraoperative wheezing in patients with reactive airway diseases (COPD, asthma). It is also associated with a very low incidence of laryngospasm. In dentistry the upper
Airway reflexes are often blunted enough during deep sedation to allow for pharyngeal suctioning.

The emulsion used to deliver the drug can be associated with allergic reactions though the incidence seems to be rare. Patients allergic to eggs are most often allergic to the albumen found in egg whites as opposed to the lecithin which is derived from the yolk. In individuals allergic to soybeans, the soy oil used does not seem to produce an allergic response. Another common issue is that propofol causes pain throughout the vessel of injection. This can be blunted through dilution; speeding up the IV fluid that the syringe-pump is piggy backed into, so that even the amount of propofol per minute is unchanged. A pretreatment dose of 0.1 mg/kg of IV lidocaine is effective in decreasing the pain at the injection site.

B. Methohexital

Intravenous barbiturates were once one of the most common agents used in sedation and outpatient office anaesthesia. They provided a more pleasant experience when compared to the available volatile anaesthetic gases of the day. Undesirable side effects though, meant that with the increasing comfort gained with the use of drugs such as propofol, barbiturates have nearly ceased being used in anaesthesia settings. These side effects include respiratory depression, myocardial depression with a reflex tachycardia, hypotension, laryngospasm, bronchospasm, and a slow recovery period.

Under the brand name Brevital, methohexital is an ultra-short-acting barbiturate that is not as highly bound to fat as other barbiturates. This allows the drug to be cleared and eliminated faster than other drugs in this class. It has all of the side effects listed above therefore its use should be avoided in patients with reactive airway diseases. It can induce seizures in patients with epilepsy.

C. Ketamine

Ketamine is a medication in a class of its own. It is the only dissociative anaesthetic currently marketed. A dissociative anaesthetic is one which produces dissociation between the brain and all sensory inputs. The patient appears catatonic with no awareness and no perception of their environment around them. This includes any perception of pain. The patient maintains critical reflexes that are suppressed with most other anaesthetics such as eye, cough, and swallowing reflexes. The patient’s eyes remain open with a nystagmic gaze; a distant wandering gaze. It produces potent analgesia and amnesia even at low dosages.

Ketamine is highly lipid-soluble and the action of ketamine peaks within one minute of administration. It is rapidly redistributed from the brain to tissues throughout the body which leads the patient to be totally awake and oriented within 15-30 minutes. The drug is metabolized and cleaved into inactive metabolites by the liver then excreted by the kidneys.

Unlike most anaesthetics, ketamine is a cardiovascular stimulant and is associated with increased blood pressure, heart rate, and cardiac output. Whereas increasing cardiac stimulation is often desirable, it may not be so in patients with hypertension or a preexisting tachycardia state. Benzodiazepines seem to blunt this effect even in low doses. Another positive property of ketamine
is that it has a minimal effect on the brain's control of respirations. It also is a bronchial smooth muscle relaxant which makes its use in asthmatics something to be considered.

When administered alone ketamine cause emergence reactions in some patients such as hallucinations and vivid dreams. These reactions are rare in children and more frequent in women than men. Using lower dosages and slower rates of administration along with synergistic dosing with drugs such as midazolam and propofol can minimize the problem of emergence reactions.

Ketamine can cause hypersalivation but this can be reduced by the use of agents such as glycopyrrolate or atropine, anticholinergic drugs that dry secretions. Contraindications to ketamine include severe cardiac disease, severe hypertension, uncontrolled glaucoma, pregnancy, and poorly controlled psychotic illnesses.

Ketamine has seen a surge in use in the last ten years as it was found that the detrimental side effect of delirium was not as common as previously reported. Using a balanced approach in combination with other drugs such as propofol has led to more use of Ketamine. See figure Table 3-1.

### Table 3-1: Complementary Effects of Propofol and Ketamine

<table>
<thead>
<tr>
<th>Complementary Effect</th>
<th>Propofol</th>
<th>Ketamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analgesia</td>
<td>None, Pain on injection</td>
<td>+++</td>
</tr>
<tr>
<td>Relaxation</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Amnesia</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>No effect</td>
<td>++</td>
</tr>
<tr>
<td>BP</td>
<td>(-) (-) (-)</td>
<td>++</td>
</tr>
<tr>
<td>Respirations</td>
<td>(-) (-) (-)</td>
<td>(-)</td>
</tr>
<tr>
<td>Bronchodilation</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Cerebral Blood Flow</td>
<td>(-) (-) (-)</td>
<td>+++</td>
</tr>
<tr>
<td>Intracranial Pressure</td>
<td>(-) (-) (-)</td>
<td>+++</td>
</tr>
<tr>
<td>Antiemetic</td>
<td>++</td>
<td>(-)</td>
</tr>
<tr>
<td>Pharyngeal Reflexes</td>
<td>(-)</td>
<td>+</td>
</tr>
<tr>
<td>Emergence Euphoria</td>
<td>(-) (-)</td>
<td>++</td>
</tr>
</tbody>
</table>

The combination of these agents reduces the fluctuations in both cardiac and respiratory parameters seen when the drugs are used alone, and there is a rapid uneventful recovery for the patient.

**Local Anaesthetics**

Local Anaesthetics are the most commonly used drugs in the dental and OMS setting. They block key ion transport across neural cell membranes thus inhibiting nerve transmissions. They are administered by syringe with aspiration always occurring first to ensure that injection of the agent and often an accompanying vasoconstrictor is not done directly into the blood stream.
A. Esters
This group of drugs has generally been replaced by the amide group. Ester compounds have a high risk of causing allergic reactions due to their chemical makeup. Two examples of ester anaesthetics are cocaine and procaine. These drugs not only cause anaesthesia but also potent vasoconstriction of vascular beds. Cocaine has been used with nasotracheal intubation to anaesthetize the nasal passages and shrink tissues so that the endotracheal tube can be more easily passed.

B. Amides
The amide group of local anaesthetics is the most commonly used in dentistry. These drugs are metabolized in the liver like most medications.

a. Lidocaine (Xylocaine)
Lidocaine is a short acting local anaesthetic that can have its action prolonged with the addition of epinephrine which slows its diffusion into central circulation. It is available as a 2% solution with epinephrine 1:100,000 or 1:50,000, or without epinephrine. A maximum of 10 carpules can be used on an average adult. Excess amounts of this drug can lead to lidocaine toxicity which affects the CNS and cardiovascular systems.

b. Mepivacaine (Carbocaine)
This drug is similar to lidocaine but with a slightly longer duration of action. It can be combined also with a vasoconstrictor to prolong its action, in this case neo-cobefrin. Carbocaine, a trademarked preparation of the drug, comes as a 2% solution with neo-cobefrin 1:20,000 or without the vasoconstrictor as a 3% solution. No more than 7-8 carpules of 3% Carbocaine should be given to an average weight adult. Calculations to prevent systemic overdose need to be done to safely use the drug with pediatric patients.

c. Prilocaine (Citanest)
Prilocaine is a short acting agent that is most often used without a vasoconstrictor. It is packaged as a 4% solution with or without epinephrine 1:200,000. A maximum of six carpules should be used for an adult patient.

d. Bupivacaine (Marcaine)
This is a long acting agent and when combined with a vasoconstrictor it can give several hours of anaesthesia at the site of injection. With a slightly delayed onset of action it is not considered as potent in its anaesthetic properties. It comes as a 0.5% solution combined with epinephrine 1:200,000. A maximum of ten carpules should be used for an adult patient.

C. Vasoconstrictors
Nearly all local anaesthetics with the exception of cocaine cause slight vasodilation. Vasoconstrictors are often added to prolong the action of the anaesthetic by slowing its distribution back into the bloodstream. With better local pain control at the surgical site the patient needs less sedation or GA to complete the surgery. This leads to fewer intraoperative complications and a
quicker postoperative recovery. **Another positive effect of using vasoconstrictors is that they significantly decrease bleeding at the surgical site.**

a. **Epinephrine**  
Also known as adrenalin, this drug is the most common vasoconstrictor used. It is a sympatho-mimetic (mimics the stimulation of the SNS) drug with strong alpha, beta-1, and beta-2 properties. Accidental injection into the bloodstream directly can lead to severe hypertension, tachycardia, and heart palpitations. Cardiac patients should have this drug used with caution if at all in a dental setting.

b. **Levonordefrin (Neo-cobefrin)**  
This is a vasoconstrictor that is less effective than epinephrine (1/6th as potent) and is compounded with mepivacaine. It is thought to have less cardiac and CNS stimulation than epinephrine.

**Adjunctive Agents**

A. Inhaled Agents

a. **Nitrous Oxide**  
This is one of the earliest anaesthetics discovered and it still has a limited role in many practices today. It is a very weak anaesthetic but does have some analgesic properties. It is administered in concentrations of 30-70%. Long term staff exposure can lead to obstetrical and neurological problems so a scavenger recovery system is mandatory. It is a compressed gas in liquid form under pressure supplied in blue cylinders.

b. **Oxygen**  
Oxygen is included because of its important role in sedation and anaesthetic care. Room air contains 20% oxygen. If a patient is given oxygen at a higher concentration it can allow for their blood to become hyperoxygenated, allowing for a greater oxygen reserve if there is a period of apnea or respiratory compromise. If used in too high of a concentration, it may mask issues with the sedation/GA being undertaken. Thus when the patient shows signs of respiratory compromise there is no physiological reserve left to work with. All patients should be on some oxygen during sedations or GA but the amount may be kept low so the team may detect issues compromising the patient early. **Nasal prongs delivering oxygen at 1-4 liters per minute (lpm) is often suitable.** Oxygen comes in green cylinders and should be used with caution. While it is not flammable itself it does support and accelerate combustion so it should not be used in the presence of flames or sparks if possible.

B. **Topical Agents**

a. **Ethyl Chloride**  
This compound is used as a vapocoolant by many dentists and surgeons who wish to use a topical agent before injections or starting a venipuncture. Other agents often have a significant delay before taking
effect. The spray should be applied for 5-10 seconds and the venipuncture initiated within 60 seconds.

b. EMLA
Used for full local anaesthesia of a venipuncture site like is often desired with pediatric patients. It needs to be applied as a thick layer under an occlusive dressing with a contact time of at least one hour. When combined with the use of nitrous oxide it can lead to a near total pain free venipuncture experience for children.

c. Topical Oral Anaesthetics
Benzocaine and lidocaine without any vasoconstrictor are the two most common intraoral topical agents used to decrease the pain of injections. Higher concentrations are used since the anaesthetic must transverse the mucosal membrane. Toxicity can be an issue since no vasoconstrictors are used so a limited amount should be applied at any given time. When topical oral anaesthetics are used the application should occur 2-3 minutes prior to injection.

C. Anxiolysis

The term anxiolysis means to dissolve anxiety. The most common class of drugs used to treat anxiety is the benzodiazepines. They can be used to treat anxiety but they can also be used alone or in combination to provide various levels of sedation and amnesia. Their use is questionable for patients with narrow angle glaucoma as they increase intraocular pressure.

a. Diazepam (Valium)
This drug has seen a decline in use due to several characteristics that make it a less favorable drug in comparison to midazolam. These include
high levels of vein irritation due to the substrate diazepam is compounded with. Also the metabolites that diazepam is broken down into are active themselves so prolonged action can be seen especially in the elderly. On occasion patients receiving valium will enter a hyperactive state instead of becoming sedated.

b. Midazolam (Versed)
Midazolam is the most common amnesic sedative agent used for moderate IV sedation in dentistry. This drug is a short acting, water-soluble benzodiazepine possessing the properties of sedation, amnesia, and anxiolysis. It is also an anticonvulsant and muscle relaxant. It is metabolized by the liver and excreted by the kidneys. It has an onset of action of 1.5 to 5 minutes, a peak action within 10-15 minutes and duration of action of 30-60 minutes. Adverse effects include respiratory depression and apnea at higher doses, hypotension, diminished reflexes, and impaired coordination. It has no analgesia properties.

**Dental dosing of midazolam as a single sedation agent in adults**

- Create a 10 ml syringe with 1 mg/ml concentration.
- Give a small test dose, 0.5 mg and wait 1 minute to test for unusual response such as hypersensitivity or allergy.
- Start sedating at a rate of 1 mg/min until the desired level of sedation is reached, usually 4-8 mg. If at 8 mg some sedation is achieved but not optimal sedation, then further titration to 10 mg can be undertaken. If at 8 mg no sedation is noticed then the dentist should stop at that amount as further dosing is unlikely to produce the desired effect.
- If no signs of sedation occur at 8 mg, or if 10 mg seems to produce less than optimal sedation then the dentist should cease sedation attempts and proceed with the procedure. Amnesia can be profound and the dental work may still be successfully done.
- Procedures taking up to an hour in duration can be done with this single drug technique.
- Clinical signs of adequate sedation include:
  - Patient starts to act in a more relaxed fashion
  - Response to questions starts to become much slower, may have trouble expressing themselves clearly
  - May begin to have trouble keeping eyes open and appears to dose off

D. Narcotics

Narcotic analgesia is a key component to a balanced approach to sedation and anaesthesia. Many of our key anaesthetics have no analgesic properties including propofol. Most often in sedation dentistry opioid agents are given by IV. They often potentiate when combined with other drugs so that anaesthetic dosages are lowered leading to less side effects. The most common unwanted side effect of all opioid based narcotics is respiratory depression and to a lesser extent hypotension from vasodilation. Several opioids also cause nausea and vomiting if an antiemetic is not given. Opioid based narcotics are one of the few
drug categories which have a reversal agent if a patient experiences an overdose. The drug is called naloxone (Narcan).

a. Morphine Sulphate (MS)
Morphine produces major central nervous system effects with its major effect being analgesia. It also produces drowsiness, sedation, and euphoria. Prolonged use can cause physical dependence and tolerance but like all narcotics, short term use is not associated with these issues. It is dosed in small increments of 2-4 mg IV until the desired effect is achieved. Morphine has action duration of one to two hours.

b. Meperidine (Demerol)
Demerol is a synthetic analgesic which has fallen out of use in the last decade in most settings. It has a longer than desired duration of action of three to four hours. It is associated with the release of histamines and their untoward side effects.

c. Fentanyl (Sublimaze)
Fentanyl has become the main analgesic used in sedation, analgesia, and anaesthesia for the intraoperative period due to several favorable characteristics. This drug is a very potent synthetic opioid that is 100 times more potent than morphine. It has favorable characteristics for use in procedural sedation and analgesia (PSA) and in anaesthesia such as a rapid onset of action, short duration of action, and lessor negative impact on the cardiovascular system than other opioids. It is the analgesic of choice in PSA when used in combination with sedatives such as midazolam, propofol, or etomidate. It produces no active metabolites and causes much less histamine release compared to morphine, hence less hypotension from vasodilation. Rapid IV administration may cause muscle rigidity, severe respiratory depression, or cardiovascular collapse. For adult use fentanyl is usually given in 50-100 mcg increments until the desired effect is reached.

Table 3-2: Drug classes commonly used in sedation and their effects

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Sedation Effect</th>
<th>Anxiolysis Effect</th>
<th>Pain Control</th>
<th>Cardiovascular Depression</th>
<th>Respiratory Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzodiazepines</td>
<td>++</td>
<td>++</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Opioids</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Propofol</td>
<td>++</td>
<td>+</td>
<td>-</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Ketamine</td>
<td>++</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

d. Remifentanil (Ultiva)
Remifentanil is a newer potent ultra-short-acting synthetic opioid which has several characteristics that are very desirable in sedation and anaesthesia. Remifentanil is approximately twice as potent as fentanyl, and 100-200 times as potent as morphine. It is metabolized by enzymes located throughout the body and is not dependent on the liver and kidneys. Patients with liver and renal disease can have remifentanil used. The drug has a very rapid onset of action of one minute and a half-life of 4 minutes. Due to its enzyme breakdown, long time infusions of this drug do
not lead to the tissue accumulation issues seen with fentanyl. It is metabolized to a compound that has only 1/4600 the potency of its parent. Like all opioids remifentanil depresses sympathetic tone which can lead to respiratory depression, hypotension, and a decreased heart rate. Intense pruritus (itching), often around the face, is a side effect relating to increased serum levels of histamines. This drug is most often given as an infusion at rates ranging from 0.1 mcg/kg/min to 0.5 mcg/kg/min. It is most often used in conjunction with a sedative or hypnotic medication such as propofol.

E. Reversal Agents

There are two drugs used as reversal agents in anaesthetic care. They can be used to reverse the actions of two drug classes, the opioids and the benzodiazepines. Reversal agents should not be used to expedite recovery times and their routine use should be avoided. The duration and action of the following reversal agents is often shorter than the opioids and benzodiazepines that they are being given for. This can lead to a patient relapsing into a deep state of sedation after being given the reversal drug. Many guidelines state that a patient must be monitored for a minimum of two hours after the last dose of a reversal agent is given. A preferred method is to undertake a partial reversal with the reversal agent administered in small doses titrated to desired effect such as an increased LOC and an increased respiratory rate without conducting a full reversal. This often will avoid adverse events such as signs of acute withdrawal to opioids or benzodiazepines or the initiation of severe pain due to full narcotic blockade.

a. Naloxone

This agent is an opioid antagonist which is used to reverse respiratory depression, apnea, chest wall rigidity, pruritus, and hypotension from opioid use.

- Dose: 0.2mg IV may repeat every three minutes.
- Onset of action: Less than one minute.
- Duration of action: 15-30 minutes.
- Adverse effects: narcotic withdrawal, analgesic cessation.

b. Flumazenil

Flumazenil is used to reverse serious respiratory depression related to benzodiazepine use. It may precipitate seizures in some patients and panic attacks in those with an underlying panic disorder.

- Dose:
  - 0.2 mg IV slowly over 15 seconds.
  - May repeat at 1-minute interval.
  - Maximum cumulative dose is 1 mg.
- Onset: <1 minute.
- Duration of action: 30-45 minutes.
- Contraindications:
  - Hypersensitivity to flumazenil.
  - Use of benzodiazepines to control seizures or increased ICP.
• Use with caution in patients who may be dependent on benzodiazepines or alcohol.
• Adverse effects:
  • Seizures.
  • Nausea/vomiting.
  • Hyperventilation.
  • Emotional liability, anxiety.
  • Sweating.

F. Anticholinergics

This refers to drugs that block the action of the parasympathetic nervous system. A main neurotransmitter of the parasympathetic nervous system is acetylcholine. Drugs that stimulate this side of the autonomic nervous system are said to have cholinergic effects. The main use of anticholinergics in anesthesia is to dry oral secretions. Another use is to increase a dangerously bradycardic (slow) heart rate in a patient during an emergency. Two drugs in this category are atropine and glycopyrrolate.

G. Antihistamines

Antihistamines can be used when a patient exhibits signs of an allergy response such as pruritus. They also have sedative properties and may potentiate the action of both hypnotics and sedatives, so care must be taken when using this class of drugs concurrently with sedatives and hypnotics. Diphenhydramine (Benadryl) and Promethazine (Phenergan) are examples of this drug class.

H. Antiemetics

Nausea and vomiting have historically been of great concern in anaesthesiology as they are common side effects of agents used. Modern drugs such as fentanyl and propofol do not possess these side effects to the degree of other medications. Still the use of antiemetics is common during sedation and anesthesia. Agents such as dimehydrinate (Gravol), metoclopramide, and ondansetron are used to treat the multiple triggers that could be leading to a patient’s nausea.

Common Agent Combinations used in PSA

The practice of combining agents to get the best PSA profile possible is a very common balanced anaesthesia practice as has been discussed. The following special considerations should be reviewed whenever combination therapy is being used:

• The risk of adverse events is increased when combination drug therapy is used.
• The agent that poses the greatest risk of respiratory depression should always be given first.
• The longer acting of the two agents should be administered first.
• Allow for enough time to pass after administering the first agent to evaluate its effect before giving the second agent.
Fentanyl and Midazolam

The combination of opioids and benzodiazepines was the first combination therapy used in PSA. Midazolam and fentanyl have been used in such areas as the ER and ambulatory daycare for several decades and have a proven safety record. They combine the sedative, amnestic, and anxiolytic properties of benzodiazepines with the analgesic properties of opioids. Fentanyl should be administered first with sufficient time given to evaluate for respiratory depression before the midazolam is given. The risk of cardiac and respiratory depression increases when these agents are used together and respiratory depression can occur in up to 25% of patients. This combination is a good choice for moderate sedation, but if used for deep sedation there is a risk that as soon as the stimulus of the procedure itself is over the patient will then suffer from possible respiratory depression or even apnea until the medications wear off.

Propofol and Fentanyl

This combination is very popular when doing painful short procedures as is often done in locations such as the emergency department. While propofol has potent sedative and amnesic properties it lacks any analgesic properties. When fentanyl is combined with propofol instead of midazolam the interval from the start of procedure to discharge is shortened significantly. In dentistry a propofol infusion is used to create prolonged moderate to deep sedation and small boluses of fentanyl are given throughout the procedure as needed for analgesia.

Ketamine and Propofol (Ketofol)

The first article describing the combination of ketamine and propofol for use outside the OR, particularly in the ER, was published in 2007. The authors from the University of British Columbia in Vancouver coined the term ketofol. Propofol provides excellent sedation but no analgesia. Ketamine provides both good sedation and analgesia but many practitioners are hesitant to use it outside of the pediatric population due to a higher incidence of emergence reactions reported in the adult population. The addition of ketamine to propofol provides for an analgesic effect while permitting a lower dose of each agent. Moreover, the two agents are complementary in their adverse effect profiles - propofol lowers blood pressure and pulse whereas ketamine raises both, propofol is an antiemetic while ketamine can cause nausea and vomiting. The end result in theory is a potent sedative-analgesic-amnesic drug combination with a neutral hemodynamic profile and a very low rate of respiratory depression or airway compromise.
Inhalation Anaesthetics

Inhaled agents are used for general anaesthesia and are usually classified as halogenated hydrocarbons. They come in a liquid form and must be placed in a vaporizer which allows for a controlled evaporation of the particular gas it was designed for. The vapors generated are inhaled and cross into the bloodstream rapidly. When the gas is turned off the process is rapidly reversed and the patient awakens.

Figure 3-7: Inhaled General Anaesthetics

Most often the patient has an advanced airway in place such as an endotracheal tube or a laryngeal mask airway for the delivery of the gas and maintenance of ventilations.

A. Sevoflurane (Ultane)
Sevoflurane is the newest of the inhaled anaesthetics. It is not cardiac proarythmic, it does not irritate the airways, and it has a low tissue solubility and rapid elimination therefore the patient awakens quickly. It is sometimes used for mask induction in pediatrics.

B. Desflurane (Suprane)
Desflurane requires a specially heated vaporiser. It can cause coughing and during the induction phase the patient can become excited. This requires the use of an IV anaesthetic at the same time. This gas is administered in high concentrations and is expensive.
Module 3 Quiz

The module 3 quiz can be found at the following web address:
https://testing.exambuilder.com/login

Contact Michael Dare at mdare@dentaed.ca if you did not receive a student ID and password
Module 4: Preoperative/Postoperative Care

Key Areas for Study

- Aldrete Score
- ASA classification system
- Mallampati Score
- 3-3-2 Rule
- BMI
- MOANS
- Consent
- ACS
- Prophylactic antibiotic therapy criteria
- HF
- Asthma
- Diabetes
- CVA
- Pt. recovery

Patient selection for PSA and anaesthesia in dentistry and OMS is based on long established anaesthesiology guidelines from organizations such as the American Society of Anaesthesiologists (ASA). Staff (often the dentist or oral maxillofacial surgeon themselves, or an RN) needs to obtain a history and perform a physical examination to identify medical illnesses, medications, allergies, and anatomical features that may affect safety such as if there is a need to provide emergency airway management for the patient. A formal screening questionnaire can be employed. Patient screening may start from the first phone contact by asking simple screening questions about snoring/sleep apnea, age, height and weight, the presence of common comorbid diseases such as diabetes, heart and lung disease, and routine use of narcotics or sedatives.

Part 1: Physical Examination

It is important to uncover positioning issues, neck range of motion, sleep apnea/snoring, difficult intubation or any history of unexpected events during procedures or surgeries. The patient assessment should include a baseline measurement of a consciousness-sedation score (Table 4-1), which should also be monitored and documented during PSA and through recovery. In addition, it should include a baseline Modified Aldrete Score or an equivalent, so that the patient can be compared against baseline during anaesthesia and while being recovered in order to determine criteria for discharge from post-sedation monitoring (Table 4-2).
Table 4-1: Richmond Agitation-Sedation Scale (RASS)

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>+4</td>
<td>Combative, violent, a danger to staff</td>
</tr>
<tr>
<td>+3</td>
<td>Aggressive, pulls or removes tubes or catheters</td>
</tr>
<tr>
<td>+2</td>
<td>Frequent non-purposeful movements, fights ventilator</td>
</tr>
<tr>
<td>+1</td>
<td>Anxious, apprehensive, but not aggressive</td>
</tr>
<tr>
<td>0</td>
<td>Alert and calm</td>
</tr>
<tr>
<td>-1</td>
<td>Awakens to voice (eye opening/contact) for &gt;10 seconds</td>
</tr>
<tr>
<td>-2</td>
<td>Light sedation, briefly awakens to voice (eye opening/contact) for &lt;10 seconds</td>
</tr>
<tr>
<td>-3</td>
<td>Moderate sedation, movement or eye opening; no eye contact</td>
</tr>
<tr>
<td>-4</td>
<td>Deep sedation, no response to voice, but movement or eye opening to physical stimulation</td>
</tr>
<tr>
<td>-5</td>
<td>Unarousable, no response to voice or physical stimulation</td>
</tr>
</tbody>
</table>

Screening and assessment also involves classification of the patient by means of the ASA physical status classification. In the field of moderate IV sedation in dentistry most guidelines suggest only ASA level I-II patients be considered for office sedation. ASA III patients should only be considered after consultation with the patient’s primary care physician or specialist. Another key aspect of patient assessment is to evaluate the patient’s airway anatomy to estimate the difficulty of airway management should an emergency airway crisis develop. This includes the completion of a Mallampati score. Parts of these scores are incorporated into a commonly used mnemonic LEMON. LEMON was developed by Walls and Murphy in their Manual of Difficult Airway Management and has been externally validated.

Table 4-2: The Modified Adult Aldrete Score

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Ability</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activity</td>
<td>Able to move voluntarily or on command:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4 extremities</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>2 extremities</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>0 extremities</td>
<td>0</td>
</tr>
<tr>
<td>Respiration</td>
<td>Able to breath and cough freely</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dyspnea; shallow or limited breathing, tachypnea</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Apneic</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Circulation</td>
<td>BP +/- 20 mmHg of presedation level</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>BP +/- 21-49 mmHg of presedation level</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>BP +/- 50 mmHg of presedation level</td>
<td>0</td>
</tr>
<tr>
<td>Consciousness</td>
<td>Fully awake</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Arousable or calling</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Not responding</td>
<td>0</td>
</tr>
<tr>
<td>O2 Saturation</td>
<td>Able to maintain saturation &gt;92% on room air</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Needs oxygen to maintain saturation &gt;90%</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Oxygen saturation &lt;90% even with supplemental oxygen</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 4-3: ASA Patient Classification Status

<table>
<thead>
<tr>
<th>Score</th>
<th>Classification</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA-I</td>
<td>A normal healthy patient</td>
<td>No significant medical history</td>
</tr>
<tr>
<td>ASA-II</td>
<td>A patient with mild to moderate systemic disease.</td>
<td>Controlled hypertension, controlled diabetes, upper respiratory infection, smoking, thyroid tumor that does not threaten the airway. Pregnancy and extremes of age are sometimes included in this category.</td>
</tr>
<tr>
<td>ASA-III</td>
<td>A patient with severe systemic disease that limits normal daily activity.</td>
<td>Chronic obstructive pulmonary disease, chronic stable angina, obesity (which is a multisystem disease), lung tumor that decreases pulmonary function</td>
</tr>
<tr>
<td>ASA-IV</td>
<td>A patient with incapacitating disease that is in constant threat to life.</td>
<td>Congestive heart failure, unstable angina, severe pulmonary or hepatic dysfunction, major trauma, prematurity with respiratory distress and necrotizing enterocolitis</td>
</tr>
<tr>
<td>ASA-V</td>
<td>A moribund patient not expected to survive 24 hours with or without surgery.</td>
<td>Ruptured aneurysm, major trauma, massive intracerebral injury</td>
</tr>
</tbody>
</table>

The Mallampati score is used to estimate the difficulty of intubation. This may be necessary if an emergency situation develops or if the patient is undergoing GA. It is accomplished by having the patient sit and open their mouth with their tongue protruding as much as possible. The anatomy of the oral cavity is visualized; specifically, whether the base of the uvula, faucial pillars (the arches in front of and behind the tonsils) and soft palate are visible. See Figure 4-1. Scores of III or IV are associated with a difficult intubation.

The LEMON mnemonic helps the team predict the difficulty of airway management if there is an emergency airway situation such as a planned deep sedation becoming one of general anesthesia. Another mnemonic, possibly even more important than LEMON is MOANS. The MOANS mnemonic lists the validated indicators of difficult bag-valve-mask (BVM) use.
Figure 4-1: Mallampati Score
Marchin, Jordi. Wikimedia Commons
Table 4-4: Lemon Mnemonic (Estimating a difficult airway management situation)

<table>
<thead>
<tr>
<th>Letter</th>
<th>Refers to</th>
</tr>
</thead>
<tbody>
<tr>
<td>L</td>
<td>Look externally- If it looks like a difficult airway it probably is. Bleeding, trauma, small jaw, large tongue, short neck, large teeth, obesity, agitation state, beard.</td>
</tr>
</tbody>
</table>
| E      | Evaluate the 3-3-2 rule: (Fig. 4.7)  
- Interior incisor distance – 3 finger breadths (less is worse)  
- Hyoid-mental distance – 3 finger breadths (less is worse)  
- Thyroid to floor of mouth – 2 finger breadths (more or less=bad) |
| M      | Mallampati score (Classes 3 and 4 are difficult airway views) |
| O      | Obstruction/obesity. Obstruction: Muffled voice, difficulty swallowing, stridor, sensation of dyspnea. |
| N      | Neck Mobility - cervical spine immobilization, intrinsic spine immobility such as ankylosing spondylitis or rheumatoid arthritis can make intubation extremely difficult. |

Figure 4-2: The 3-3-2 Rule (Significantly more or less than these numbers indicates a difficult airway)
Table 4-5: MOANS mnemonic predicting difficulty of BVM use

<table>
<thead>
<tr>
<th>Letter</th>
<th>Refers to</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>Mask seal/male face/Mallampati class 3-4. Trauma to the face, blood and debris, beards all can make getting a BVM seal difficult.</td>
</tr>
<tr>
<td>O</td>
<td>Obesity/Obstruction. BMI &gt; 26 kg/m² indicates likely difficulty using BVM.</td>
</tr>
<tr>
<td>A</td>
<td>Age &gt; 55 yo, perhaps because of a loss of muscle and tissue tone in the upper airway.</td>
</tr>
<tr>
<td>N</td>
<td>No teeth. Leave dentures in situ if possible.</td>
</tr>
<tr>
<td>S</td>
<td>Stiff/Snoring - patients with reactive airway disease such as COPD, asthma, ARDS, and pulmonary edema where high pressures are needed to ventilate and get chest rise. History of snoring or sleep apnea.</td>
</tr>
</tbody>
</table>

One last assessment measurement is usually calculated for patients having anaesthesia called the Body Mass Index (BMI). It is a measure for human body shape based on an individual's mass and height. Patients with a BMI over 40 should be approached with caution as obesity is considered to be equivalent to a multisystem disease and obesity is linked to difficult airway management in the case of an emergency.

![Figure 4-3: Body Mass Index](image)

The physical exam should include a baseline set of vital signs including pulse oximetry. Abnormalities in blood pressure and heart rate need to be assessed. One last part of the physical exam is to listen to the breathing (auscultation) with a stethoscope to see if the patient has wheezes or crackles. To decide to auscultate lung sounds can be based on what medical history the patient has - asthma or COPD for example. If the heart rate is abnormal the patient may have an ECG tracing done.
Consent

At the time of the physical exam and history taking the practitioner will review the intended treatment and obtain consent to treat. A consent form describes:

- The nature of the treatment proposed
- Alternatives to the proposed treatment
- The anticipated outcome and benefits of the treatment
- The risks and potential complications of the proposed treatment
- The risks and potential complications of the anaesthesia itself

Part 2: History

Taking a medical history is an essential part of a preoperative patient evaluation. Most practices use a preprinted dental history form that the patient fills out which is then reviewed before or after the physical exam. A check box type form works well. The following is a review of various medical problems and how they can affect a patient’s care during anesthesia.

Cardiovascular Disease

Acute Coronary Syndrome

A. Angina pectoris

Angina refers to the chest pain and other symptoms a patient experiences due to a narrowing or spasm of a coronary artery. The decreased blood flow causes ischemia but not necrosis (death) of cardiac tissues. With atherosclerotic disease the vessel has lost much of its ability to vasodilate and the lumen is narrowed. In certain conditions of high oxygen demand the blood stream cannot deliver the required flow and chest pain is felt. By decreasing the workload and resting, the pain is resolved without cellular death. If the pattern of what precipitates an episode and what treatment is required to alleviate the symptoms is the same each time the patient is said to have stable angina. If the pattern is changing—less stressors are needed to get an episode to start and more treatment is needed to get relief of symptoms, then the patient is said to have unstable angina. Unstable angina should be immediately assessed by the patient’s doctor and the patient is not an appropriate client for dental treatment until their cardiac issues are treated. The main treatment medication during an episode of angina is rest and the use of a vasodilator, nitroglycerin.

B. Myocardial Infarction (MI)

An MI is a heart attack. It refers to the death of myocardial tissue that occurs from a thrombus (clot) formation which blocks the lumen of a vessel leading to myocardial cellular death (necrosis) distal to the clot. If a patient has a history of an MI, more investigation is usually required. What area of the heart was damaged? How large was the MI? Has it led to the patient having what is known as heart failure? It is recommended that elective surgery not be performed in the first 6 months following an MI.
Valvular Heart Disease

A. Rheumatic Heart Disease
This is a condition of the heart which occurs when a person has had rheumatic fever. It is caused by a bacterial infection of the heart from beta-hemolytic streptococci bacteria. It primarily causes damage to heart valves, especially the mitral valve with the potential for backwards regurgitative blood flow and some degree of heart failure. Patients often have a heart murmur, sounds heard on auscultation of heart sounds.

B. Mitral Valve Prolapse (MVP)
MVP occurs when a valve leaflet is damaged and can prolapse into the left atrium. It can be caused by a number of things such as a myocardial infarction or endocarditis. It affects around 5% of the population, mostly women. It used to be standard practice that patients with MVP would receive prophylactic antibiotic therapy. This would prevent bacterial endocarditis if some bacteria from the surgical site entered the bloodstream. These guidelines have been revised and only select patients now receive antibiotic therapy. They include patients with

- Artificial heart valves
- History of infective endocarditis
- Cardiac transplant patients who develop heart valve issues
- Certain congenital heart conditions

C. Prosthetic Heart Valves
Patients with artificial heart valves will often require prophylactic antibiotic therapy. They are most often on anticoagulants and require careful management prior to any surgery.

Hypertension

Hypertension refers to the elevation of blood pressure to levels which are beyond normal for a particular age of patient. In adults it is considered to be any BP > 140/90. Most people with hypertension have primary idiopathic (no known cause) hypertension. Patients with hypertension are usually on medications such as beta-blockers like atenolol, vasodilators like ACE inhibitors, and diuretics such as Lasix and HCTZ.

Heart Failure (HF)

This condition occurs when the heart is unable to pump adequate cardiac output to meet the needs of the body. It can be caused by many things but most often the cause is from such things as an MI, cardiomyopathy, or problems with the valves of the heart. Treatment is focused on maintaining a state of homeostasis so that the body’s organs can be adequately perfused. First-line therapy includes the use of ACE inhibitors such as ramipril, enalapril or captopril. This drug category has been shown to improve survival and quality of life for HF patients. Diuretics are used to maintain normal blood volumes. Other drugs used include beta blockers and AR blockers.

Heart failure can involve either the right heart or the left heart. Left sided failure means that the left heart cannot pump out blood to keep up with the venous return from the lungs. A higher pressure in the pulmonary vessels leads to fluid accumulating in the
alveolar sacs. This in turn causes the patient to experiencing shortness of breath. In right heart failure the venous blood returning to the right atrium and ventricle cannot be effectively pumped out to the lungs. Higher venous pressures lead to fluids shifting out into the tissues and forming what is called peripheral edema in areas like the lower legs and ankles.

Post Heart Surgery Patients

Patients who have had open heart surgery are often on many cardiac medications including anticoagulants. Management of their anticoagulant medications and the use of prophylactic antibiotics may be needed.

Pulmonary Disease

Diseases of the respiratory system can significantly impact a patient’s ability to have office based anaesthetic procedures. A physician consult is often required along with the completion of certain pulmonary function tests.

Asthma

Asthma is a common chronic inflammatory disease of the airways. It is caused by a combination of environmental factors and having a genetic predisposition. It is characterized by wheezing, coughing, shortness of breath, and chest tightness. The patient needs to be assessed as to the severity of their asthma. Have they ever been hospitalized for asthma overnight? Have they ever been placed on a ventilator for their asthma? How often do they need to use their inhaler? The anaesthetic team must be prepared to deal with an asthmatic episode if one occurs. Minor respiratory illnesses can precipitate an increased frequency and severity of a patient’s asthma.

Chronic Obstructive Pulmonary Disease (COPD)

The presenting ongoing chief complaint of someone with what is commonly known as emphysema is dyspnea (difficulty breathing). It is usually associated with heavy cigarette smoking.
Enlargement of alveolar sacs and extensive scarring leads to a markedly reduced surface area for gas exchange in the lungs. COPD patients are poor candidates for outpatient anaesthesia and sedation.

Bronchitis

This is the usually temporary condition of an inflammatory state of the bronchial mucus membranes. On occasion it can progress to a more chronic state. It is associated with increased secretions and a narrowing of bronchial airways. **Any surgery under sedation or anaesthesia should be delayed if possible until a resolution of the bronchitis.**

Smoking

Patients who smoke are much more likely to have significant bronchial secretions along with being at increased risk for a cardiac or respiratory event perioperatively.

Upper Respiratory Infection (URI)

Having a cough and cold usually leads to increased airway secretions. **While ill these patients are poor candidates for sedation or GA.**

Endocrine Disorders

Diabetes Mellitus

Insulin, which is produced in the pancreas, is crucial for cells and their ability to metabolize carbohydrates. The disorder is characterized by an inability for glucose to be metabolized by cells due to low insulin production. This leads to a high blood sugar level called **hyperglycemia.** There are two basic types of diabetes;

- **Type I Diabetes**
  - Patients have very low or no production of insulin therefore they require insulin injections to regulate their glucose metabolism.
- **Type II Diabetes**
  - This is the most common form of diabetes accounting for 85-95% of all cases.
  - Patients produce enough insulin to not require injections of insulin.
  - Part of the issue is that cells fail to use the insulin properly.
  - They usually take oral medications such as metformin.

The typical signs and symptoms of uncontrolled diabetes are those of hyperglycemia. **This includes weight loss, frequent urination, increased thirst, blury vision, and skin rashes. If a patient's sugar is too low (hypoglycemia) due to inadequate food intake in relation to the amount of insulin taken, then different more acute signs and symptoms can occur such as restlessness, tremor, weakness, altered level of consciousness, coma, diaphoresis (sweating), irritability, and ataxia (loss of balance). If left untreated both hyperglycemia and hypoglycemia are life threatening.**

Patients with diabetes create challenges for a dental team. Firstly they do not generally heal well and they have an increased rate of infections. Many practitioners will prescribe
prophylactic antibiotic therapy for diabetic patients. Secondly, they are usually on oral medications or subcutaneous insulin injections. Many patients are now using a continuous insulin pump. The management of their NPO status and adjustments to their medications is required. Booking of these patients as the first cases of the morning is often a good choice.

Most often a physician consult is a good idea prior to undertaking anaesthesia for these patients. Testing can help tell how well the patient’s diabetes is being controlled.

- Fasting blood glucose. This should be less than 7.0 mmol/liter
- HbA1C level tells how well the diabetes has been controlled over recent months.

Blood sugar levels should be checked before, during, and after the sedation or GA. Intermediate and long acting types of insulin are generally not held for type I diabetics. The use of sugar containing IV fluids may be considered such as D5W. Here is an example insulin regime for a patient NPO at midnight.

- Night before procedure
  - Patient taking evening or bedtime insulin
    - NPH/Levemir® (detemir) – Give usual dose
    - Mixed insulin (70/30, 75/25 etc.) – Give usual dose
    - Lantus® (glargine) – Give 80% of usual dose
  - Patients using insulin pump
    - Continue basal rate

- Morning of procedure (for patients who are NPO)
  - Morning insulin injections
    - Morning intermediate or long-acting insulin
      - NPH/Levemir® (detemir) – Give 1/2 of usual morning dose
      - Lantus® (glargine) – Give 80% of usual morning dose
      - Mixed insulin – Give 1/3 usual morning dose
  - Morning short-acting insulin (Novolog®, Humalog®, Apidra®, Regular)
    - Hold all short acting insulin
  - Patients using insulin pump
    - Continue basal insulin rate

- Intra-operative and postoperative use of the pump needs to be addressed on an individual basis.

If a patient at any time starts to show signs of hypoglycemia and they are awake then they can be given sugar, oral glucose gels, or juices. If they are more obtunded then IV glucose can be given.

Adrenal Disorders

The cortex of the adrenal gland secretes hormones called corticosteroids. If a patient has a low level of production for this hormone they have what is called Addison’s
disease. This state is a life threatening condition because the patient cannot respond appropriately to even minor physical or mental stressors. They usually take a daily preparation of a cortisone medication. Consultation with the patient’s physician is recommended before undergoing dental surgery.

Thyroid Disorders

The main function of the thyroid gland is to regulate the body's basal metabolic rate. It does this through the hormone release of thyroid hormones T3 and T4.

A. Hypothyroidism

Patients with hypothyroidism can have an increased sensitivity to sedatives, narcotics, and anaesthetic agents. Untreated patients may be lethargic, have an increased weight gain, and have intolerance to cold.

B. Hyperthyroidism

Patients may show symptoms of insomnia, tremors, agitation, tachycardia, and weight loss. They are at risk of having a **Thyroid Storm** while under GA. It is characterized by a cardiac arrhythmias, **pyrexia (fever)**, mental agitation, and vomiting. It is a medical emergency.

All patients with known thyroid dysfunction should have a physician consultation.

Liver Disease

**Cirrhosis of the liver is the formation of scar tissue within the liver.** It can be caused by many things such as viral infections or alcoholism. **Since the liver is key to the metabolism of many drugs, liver dysfunction is of concern for patients undergoing sedation or GA and for postoperative pain management.** Medications can have a more pronounced action and a prolonged action such as respiratory depression. Postoperative and intraoperative bleeding can be a concern because the liver produces many of the clotting factors key to blood clotting. Liver disease can also put strain on the cardiovascular system as fluid shifts such as ascites make the heart work harder. Some liver diseases such as the various types of viral hepatitis are very contagious. **Patients with advanced liver disease should not undergo outpatient surgeries under GA or sedation without careful consideration to the risks involved.**

Kidney Disease

The kidneys are responsible for the elimination of wastes from the body including some medications. The practitioner must take this into account when considering what medications should be used. The patient may be undergoing renal dialysis and this often means the use of a short acting anticoagulant. **Dental procedures should not be booked on the same day as dialysis for this reason.**
Nervous System Disorders

Seizure Disorders

Many patients have seizure disorders. They can be the result of several different underlying systemic disorders. These patients may be on one or more seizure medications. The patient should be questioned about the type and frequency of their seizures. If a seizure develops intraoperatively IV ativan or midazolam may be given to treat the seizure.

Cerebrovascular Accidents (CVA)

Patients can have a history of stroke or transient ischemic attack (TIA) which is signs and symptoms of a stroke that resolve within 24 hours of onset. Most experience an ischemic stroke due to an occluded blood vessel in the brain while a hemorrhagic stroke (ruptured blood vessel) is less common. Patients often are on anticoagulants post CVA/TIA. These drugs may need to be discontinued preoperatively for a short period of time. Another issue may be related to the deficits the patient has suffered from their CVA. They may have swallowing dysfunctions and respiratory compromise. Patients with a history of CVA may also suffer from other cardiovascular diseases such as hypertension, ACS, heart failure or diabetes. A specialist consult is often required for these patients.

Other Medical Issues

Pregnancy

Most often OMS and dental procedures can be delayed until after a pregnancy. If circumstances are such that dental/OMS must be undertaken then the second trimester is often the best period to undertake care. A consultation with an obstetrician is recommended. The first trimester should be avoided as the greatest surgical and anaesthetic risks occur at that time. In the last trimester patient positioning is an issue as the weight of the uterus can compress the inferior vena cava so the patient should not be placed in a low supine position but instead be in a left tilted position to keep weight off of the vena cava.

Obesity and Sleep Apnea

Obesity and all of the multisystem diseases it is associated with such as diabetes, hypertension, cardiovascular disease, stroke and sleep apnea make obese patients difficult to safely manage in an outpatient setting. A BMI of 38-40 indicates a level of obesity that puts patients at too high of a risk for office based sedation and GA. Consider using local anaesthesia when possible.

Consider the following for these patients:

- Use of an appropriately sized BP cuff
- Venous access may be very difficult
- Treat these patients in an upright or semi-fowlers position if possible to reduce respiratory compromise.
HIV

Current medications used to treat HIV can have a profound effect on the anaesthetics agents used, prolonging their action substantially. The protease inhibitors Norvir, Invirase, and Crixivan have been shown to greatly lengthen the actions of benzodiazepines and barbiturates. These sedative agents should not be administered to patients taking protease inhibitors.

Drug Addiction

People suffering from addictions often have a built up tolerance to sedatives and analgesic drugs. This can make their management as patients during sedation and GA difficult. Careful questioning about drug use in the 24 hours prior to their appointment should be done. If the patient is suspected of being under the influence of drugs the surgery should be postponed.

Part 3: Postoperative Care

The purpose of the recovery period and the associated post anaesthetic monitoring is to ensure the return of normal physiologic function before the patient is discharged. The environment must be comfortable and well lit with the appropriate monitoring and resuscitation equipment available. In some smaller offices the patient is recovered in the operatory itself.

Equipment Needs of the Recovery Area

- Oxygen supply
- Oxygen delivery devices
  - BVM
  - Face Masks
  - Nasal cannula
  - Oropharyngeal airways
  - Advanced airways and their adjuncts needed for insertion
- Suction unit
- Patient monitor
  - ECG
  - BP
  - SpO₂
- Emergency crash cart
  - Defibrillator/AED
  - Resuscitation medications
  - Airway management equipment
  - IV supplies

Patient Assessment

Transfer of care should start with an assessment of the patients' level of consciousness and their A, B, C's (airway, breathing, and circulation). Vital signs should be repeated and monitored as dictated by the patient's condition. The surgical sites should be
assessed for any issues such as bleeding. Pain control for the postoperative period should be managed and discharge scripts organized.

Discharge Criteria

- Full recovery to their pre-sedation/anaesthesia level of consciousness
- Recovered cardiovascular function
- Stable airway and breathing
- Adequate state of hydration
- Patient can ambulate at their pre-sedation level

Postoperative Patient Instructions

Written instructions are given to accompanying person escorting the patient from the office. They should include:

- A 24 hour follow-up number for the practitioner
- Return to normal diet instructions
- Pain management instructions
- Limitation of activities instructions (considered impaired for 24 hours postoperatively)
- Anticipated patient behavior in the hours following surgery relating to the sedation or GA.

Module 4 Quiz

The module 4 quiz can be found at the following web address: https://testing.exambuilder.com/login

Contact Michael Dare at mdare@dentaed.ca if you did not receive a student ID and password
Module 5: Anaesthesia Equipment

Key Areas for Study

- Monitoring Requirements
- Pulse oximetry
- ETCO₂ monitoring
- Pretracheal stethoscope
- Cardiac monitoring
- NSR- normal electrical system firing and structures.
- Parts of an ECG complex
- Key rhythms: atrial fibrillation, SVT, VT, VF, bradycardias, asystole
- Basics of Inhalation anaesthesia machine
- Jaw thrust, head tilt-chin lift, oral airways, BVM use
- Nasal cannula, SFM
- Advanced airways
- Verification of correct endotracheal tube placement

Part 1: Monitoring for PSA

The monitoring of patients during PSA is one of the most important aspects of patient safety. The most indispensable monitor is the qualified staff member who observes the patient for skin and mucosal color changes, visual signs of pain and lightening of sedation, respiratory rate and depth, and audible (ear only needed) adventitious (abnormal) respiratory signs. Monitoring equipment is there to supplement the observational skills of the staff. Team members must be able to operate and interpret the information these adjunctive devices provide. They must also understand and be able to act on the information gathered appropriately to keep patients safe.

Monitoring Requirements

Monitoring requirements for various levels of PSA change depending on the location of services, the institutional guidelines, regional guidelines, and guidelines of the specialty overseeing delivery (dentistry, ER, gastroenterology etc.) Typical monitoring guidelines include the following monitoring parameters:

- ECG (for ASA levels II and III)
- Capnography for all moderate and deep sedation
- Noninvasive BP
- Respirations
- Heart rate
- Pulse oximetry
- LOC

Initiate, monitor and interpret ECG rhythms for ASA scores II-III, history of previous cardiac disease and/or dysrhythmias, and for patient complaints and/or observations of cardiorespiratory symptoms.
Establish End-Tidal CO₂ monitoring for:

- **Deep sedation**
- Also consider if available for moderate or dissociative sedation
- If respiratory rate and depth assessment may be difficult due to procedural draping or positioning.

The ASA has established the following as required monitoring parameters:

- LOC
- ECG: Must be used for all patients undergoing deep sedation
- ETCO₂: Quantitative capnography required for all moderate and deep PSA as of July, 2011
- Pulse oximetry
- Respiration evaluation by auscultation and observation
- Noninvasive BP
- Heart rate

Much controversy has accompanied the guideline for the use of ETCO₂ monitoring put forward by the ASA in 2011. A variety of governing bodies and specialty associations have challenged the guideline as not being evidence based. They also state that hospital areas caring for patients of much higher risk do not have a mandate to use capnography on deeply sedated patients and patients still deep from general anesthesia such as seen in recovery rooms.

For both moderate and deep PSA an individual dedicated to monitoring the patient should be present other than the practitioner performing the procedure. For deep sedation that individual should have no other responsibilities. During moderate sedation that person can assist with minor tasks that are interruptible after the PSA level has been stabilized along with the patient’s vital signs as long as sufficient monitoring of the patient’s sedation level can be maintained.

**Monitoring for Oxygenation and Ventilation**

The most likely adverse event of serious threat to patient morbidity and mortality is one of a respiratory nature. The monitoring of a patient’s respiratory status has two components: ventilation and oxygenation. There seems to be much confusion about the use of pulse oximetry and whether or not it is an effective tool to monitor ventilation and detect apneic episodes. The most important monitoring tool is the practitioner themselves. Observation of chest rise-rate and depth, and skin color can be made by team members. **Cyanosis is a bluish color that appears in the skin, mucous membranes, and nail beds. It is an extremely late sign of hypoxemia (low blood oxygen levels).**

**Pulse Oximetry**

Hemoglobin saturation (SaO₂) is the amount of oxygen bound to hemoglobin in arterial blood expressed as a percentage. A normal saturation reading is > 95%. Pulse oximetry allows for a noninvasive continuous measurement of this parameter at the bedside. It is now commonly referred to as the fifth vital sign and it measures pulse rate also. Oximeters are made up of a light source (LED), a photo detector and a microprocessor. It relies on a process called spectral analysis which uses light absorption characteristics
that are unique in all matter types to determine the physiochemical properties of a substance. The response time of the oximetry reading lags behind the patient’s physiologic status from 4-20 seconds in most monitors. Centrally located probes tend to respond faster than the commonly used finger probe.

The use of oximetry is to give information on arterial blood oxygen content. When pulse oximetry is used to detect hypoventilation and apnea it must be noted there can be a significant delay between an apnea/hypoventilation episode starting and that event then being reflected by decreasing oxygen saturation levels. The addition of supplemental oxygen to the equation can even further delay apnea/hypoventilation detection. A patient may be apneic for several minutes (In excess of 5 minutes) before the oxygen saturation drops significantly (<92%).

Table 5-1: Causes of Oximetry Errors

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Examples</th>
</tr>
</thead>
</table>
| Sensor Location           | • Poor blood flow to extremities in critical illnesses. Forehead reflective oximetry best.  
                            | • Extraneous light sources                                    |
| Motion artifact           | • Exercise                                                  |
                            | • CPR                                                       |
                            | • Shivering                                                 |
                            | • Seizures                                                  |
                            | • Tremor                                                    |
| Signal degradation        | • Poor peripheral perfusion                                 |
                            | • Hypotension                                               |
                            | • Hypoperfusion                                             |
                            | • Vasoconstriction                                          |
                            | • Nail polish                                              |
| Physiologic range         | • Inaccurate if systolic BP < 80 mmHg                         |
                            | • Increasingly inaccurate at SaO2 < 75%                      |
| Dyshemoglobinemia         | • Met-Hgb                                                   |
                            | • CO-Hgb                                                    |
| Intravenous dye           | • Methylene blue                                            |
                            | • Indocyanine green                                         |

End-Tidal CO₂ Monitoring

Relying on pulse oximetry is a late determinate of ventilation and respiratory compromise. Capnography is the measurement of exhaled CO₂ in a patient’s breath. CO₂ monitors measure the partial pressure of CO₂ in expired gas expressed in mmHg. When measured at the end of expiration it is referred to as end-tidal CO₂ (ETCO₂) which approximates alveolar CO₂. Wave form ETCO₂ machines give the most data on metabolism, perfusion, and ventilation.
Figure 5-1: A Bedside Pulse Oximeter

Figure 5-2: Colorimetric CO2 detector used to confirm intubation of the trachea. It is a one-time use device.
Most of these devices use an infrared sensor, which measures the amount of infrared light absorbed by the passing CO₂ gas. **Nonintubated patients undergoing sedation are usually monitored with a sidestream capnographer through a nasal cannula.** This is done most often with a set of specialized nasal cannula which includes a port which sucks in gases from nasal exhalation. These are somewhat mixed with room air and inspired oxygen so the CO₂ level is somewhat diluted. A normal **ETCO₂ is 35-45 mmHg** but with sidestream cannula use the reading is somewhat lower; **25-35 mmHg.** The main use of the nasal cannula system is to detect apnea, **bradypnea** (slow breathing), and to give a count of the respiratory rate.

The other method of use is with patients who are under GA and have an advanced airway in place. Here the gas is drawn off at the endotracheal connector itself and will not be diluted with oxygen or room air.
Figure 5-5: Endotracheal tube adapter for ETCO₂ measurement

A normal capnogram has a typical rectangular shaped waveform that should start from 0 mmHg and end at 0 mmHg. If the baseline maintains itself at above zero CO₂ rebreathing or hypoventilation is occurring. The plateau has a gentle positive slope with ETCO₂ been measured at the end of exhalation (black arrow, fig. 5-6). With the beginning of inspiration there is a rapid drop back to baseline.

Figure 5-6: A Normal Capnogram Waveform

In the setting of PSA the use of capnography is the earliest and most sensitive indicator of apnea or hypoventilation. Several studies have shown that patients undergoing procedural sedation have a high rate of hypoxic events including hypoventilation and apnea, and clinical assessment of chest rise is not sensitive for detecting these events. Oxygen desaturation is a late finding in hypoventilation, especially in patients receiving supplemental oxygen. Addition of capnography to standard monitoring provides advanced warning and reduces unwanted hypoxic events.
When using capnography to assess for respiratory depression it must be understood that both increases and decreases in expired CO₂ can mean a patient is experiencing hypoventilation issues.

**Capnographic evidence of hypoventilation can include:**

- ETCO₂ > 50 mmHg.
- A change of 10% or more from baseline.
- An absolute change of 10 mmHg or more.
- Loss of waveform.

Classic hypoventilation results in a waveform with a wide base and increased amplitude as seen in Figure 5-7. Shallow ineffective breathing will give a low amplitude waveform as CO₂ levels are diluted by dead space gases. See Figure 5-8.

**In sedation one of the greatest uses of capnography is to detect apnea. If the patient stops breathing the system will lose the waveform and alarm immediately. Oxygen saturation (known as SpO₂) can continue to read normal for several minutes. During that time dangerously high levels of CO₂ can develop in the bloodstream.**

![Figure 5-7: Classic Hypoventilation Waveform Capnogram](image)
Figure 5-8: Shallow Ineffective Breathing Capnogram

**Transthoracic Resistance Respiratory Rate**

Another modality the monitors breathing is the measurement of *transthoracic resistance* changes that occur while breathing as detected through the ECG electrodes of an ECG monitor. As a person breathes in and out the electrical resistance changes across the chest/abdomen and the monitoring system calculates the patient’s respiratory rate from this information.

**Pretracheal Stethoscope**

This type of stethoscope is designed to listen to breath sounds continuously during anaesthesia. The preferred location is to place the bell of the stethoscope in the *suprasternal notch* (the notch at the base of the neck just above the sternum). A *course sound of tracheal air movement is heard* that gives information as to respiratory rate and flow.

Figure 5-9: A Bluetooth Pretracheal Stethoscope
**Cardiovascular Monitoring**

**Electrocardiograph (ECG)**

All patients under deep sedation and GA require cardiac monitoring. Patients with higher ASA classifications or cardiac risk factors under moderate sedation should be monitored also.

![Modern Cardiac Monitor](https://commons.wikimedia.org/wiki/File:Modern_Cardiac_Monitor.jpg)

*Figure 5-10: Modern Cardiac Monitor (Has built in pace-maker and defibrillation capabilities)*

Wikimedia Commons.

Anaesthesia assistants are not the primary team members responsible for ECG interpretation and treatment, **but as a team member they should be able to recognize key rhythms and anticipate how they would be treated. They also should understand how to connect the patient to the monitor and how to adjust the monitoring parameters.**

![Three Lead Cable ECG Electrode Positions](https://commons.wikimedia.org/wiki/File:Three_lead_cable_ECG_Electrode_Positions.png)

*Figure 5-11: Three Lead Cable ECG Electrode Positions*

Wikimedia Commons.
Figure 5-12: Five Lead Cable ECG Electrode Position

Figure 5-13: Five Lead ECG Electrode Cable and a “Sticky” ECG Foam Electrode
To monitor the ECG, the patient has sticky electrodes attached to their skin as shown in Figures 5-11 and 5-12 depending on whether the cable is a three or five lead system. The leads show the electrical picture of the heart from different views so a five lead cable gives more “views” than a three lead cable. On the machine itself you can select what view you want to look at and whether or not you want to amplify the size of the ECG. Less electricity makes it to the surface with larger patients and this can make the ECG difficult to see and interpret, so this is why an ECG size/amplitude adjustment is available. Most often the ECG view used is Lead II. There is a lead select function/dial on the monitor also.

Blood Pressure and Pulse

Most offices now use automatic blood pressure machines. These machines will automatically record pressures at preselected intervals and keep several readings in memory. This function is included in multi parameter monitors like the one shown in figure 5-14. Often the pulse is given as a reading also if it is a stand-alone BP machine.

![Figure 5-14: A Multi-function Monitor- ECG, BP, Oxygen Saturation, Respirations](image)

A Crash Course on Interpreting Bad ECG Rhythms

Courses that teach healthcare providers how to interpret ECGs are multiple days in length and the books used to teach those courses hundreds of pages long. Learning to interpret ECGs is like learning to speak a new language. It takes time. Many other health care professionals are not taught how to interpret ECGs as part of their regular education, including nurses.

The goal in this manual is to teach the reader the key components that make a particular heart rhythm dangerous and what the treatments are for some of those rhythms. Let’s first look at a normal sinus rhythm, the normal heart rhythm that most patients will be in.
Figure 5-15: A Normal Sinus Rhythm

Figure 5-15 shows a sinus rhythm at a rate of around 90 beats per minute (BPM). The P wave (A) represents the depolarization of the atria. This wave should be an upright positive deflection in the lead II view. The second wave is the QRS wave and it represents the depolarization of the ventricles. The width of the QRS wave (D) should not be more than three small boxes (each box represents 0.04 seconds on the horizontal axis) wide. QRSs that are abnormally wide (>3 small boxes or 0.12 seconds) often indicate that the rhythm is not starting in the sinus node but instead down in the ventricles. Generally the wider the QRS the poorer the contractility of the ventricle, the poorer the stroke volume and hence the poorer the cardiac output. The time from the beginning of the P wave to the beginning of the QRS is called the PR interval (C). It should not be wider than five small boxes or one large box on the ECG paper (0.20 seconds). This represents the time delay from the beginning of atrial depolarization to the beginning of ventricular depolarization and it allows for atrial kick. Repolarization of the atria occurs but the wave is buried inside the QRS. The repolarization of the ventricles is visible as the T wave (E).

Normally the sinus node fires causing atrial depolarization. The wave of depolarization then travels down to the AV node and is delayed by 1/10th of a second before travelling down the Bundle of His and into the right and left bundle branches. From there it travels into smaller and smaller branches until it terminates in the Purkinje fibers. This causes a very fast synchronous depolarization of the ventricles and from that an efficient contraction of the ventricles with maximum stroke volume.

The naming of rhythms consists of two main components. The first part describes the location where the rhythm started in the heart. The second part describes the rate of firing for the rhythm in relation to the intrinsic firing rate of that region of the heart.

What Makes a Rhythm Dangerous?

The reasons an ECG heart rhythm may be dangerous can be related back to the anatomy and physiology review in module one. If a heart beats too slowly then despite good stroke volume cardiac output will be low. If a heart starts to beat too fast then stroke volume suffers due to inadequate diastolic filling time. Lastly if the QRS is wide then (>0.12 sec) there is a loss of efficiency to the contractions of the ventricles. The wider the QRS, the worse the stroke volume.
Negative factors affecting cardiac output when looking at an ECG

- Heart rate too slow (< 50 BPM)
- Heart rate too fast (> 150 BPM)
- QRS wide (> 0.12 sec)
- Loss of atrial kick (least negative impact of the listed conditions)

The bookends for bradycardia and tachycardia vary depending on the age and health of the patient’s heart. Someone who has extensive cardiac disease may only tolerate a heart rate between 70-90 bpm whereas an endurance athlete may tolerate a range between 30 and 200 bpm.

Key Cardiac Rhythm Examples

A. Atrial Fibrillation
Atrial fibrillation is the most common “need to treat” rhythm in western society. It rarely causes gross instability of a patient’s cardiac output unless the ventricular rate is too slow or too fast. The upper chamber is fibrillating and no coordinated atrial contraction occurs.

![Atrial Fibrillation Diagram](image)

Figure 5-16: Atrial Fibrillation
If the rate is adequate the only loss to cardiac output is a loss of atrial kick. There are no P waves but instead an undulating wavy baseline with small fibrillation waves. The impulses that make it down into the ventricles are absolutely random and therefore irregular. This leads to a totally chaotic pattern of QRSs in terms of their rate and hence an irregular pulse. Patients with chronic atrial fibrillation are on oral anticoagulants since there is an increased risk of thrombus formation in the left atrium. This can lead to emboli (a floating clot) breaking free and traveling to the brain causing a CVA.

![Figure 5-17: Atrial Fibrillation (AF)](image)

B. Supraventricular Tachycardia
This rhythm is also common and is often precipitated by increased catecholamine levels as seen when a patient is anxious or stressed. It rarely causes any serious instability in cardiac output as it usually occurs in younger patients with healthy hearts. The QRS is most often narrow so ventricular contraction is efficient. There may be a loss of atrial kick and some decreased filling time but most patients tolerate this rhythm well. It is characterized by sudden paroxysmal onset, a regular rapid rate (usually around 160-200 bpm), and an absence of visible P waves. It can often be terminated by stimulating the vagus nerve and increasing parasympathetic tone with what are known as vagal maneuvers.

![Figure 5-18: Supraventricular Tachycardia (SVT)](image)

C. Ventricular Tachycardia (VT)
VT is a dangerous rhythm for several reasons. It is associated with ischemia and it is very unstable electrically. It will deteriorate into ventricular fibrillation (a cardiac arrest rhythm) eventually. Some patients will have little or no cardiac output and be pulseless. Other patients may have a pulse and may even be awake when in VT. This rhythm is characterized by wide regular QRSs at a rate usually over 150 bpm and no visible P waves.
Figure 5-19: Ventricular Tachycardia (VT)

A patient in this rhythm most often needs to be shocked with a defibrillator. If the patient has a pulse the shock is called a cardioversion, if they are pulseless it is called a defibrillation. Promptly starting CPR should occur until the patient can be defibrillated if they are pulseless.

D. Bradycardias
Many different bradycardia rhythms exist and it is beyond the scope of learning of this manual to learn them all. As a general rule the slower the rate and the wider the QRS the worse the cardiac output. The following are examples of various bradycardias.

Figure 5-20: Sinus bradycardia at 52bpm (At rest patient would most likely be stable)
E. Ventricular Fibrillation (VF)

VF is a lethal heart rhythm where the ventricles are fibrillating at between 400-600 impulses a minute with no coordinated cardiac depolarization and hence no cardiac output. The patient will be in cardiac arrest requiring immediate CPR and defibrillation as soon as it is available. The rhythm has no QRSs or P waves visible and a fibrillating waveform.
F. Asystole

Asystole is the absence of all electrical activity in the heart. There is no pulse and CPR should be immediately started.

![Figure 5-21: A Sinus Rhythm terminating in Asystole.](image)

Part 2: Anaesthetic Equipment and Monitoring for GA

Anaesthetic Machines

Basic for Nitrous Oxide Use

These machines supply oxygen and nitrous oxide through supply lines. A series of flow meters and valves on the machine deliver the gases to the patient as a liters/minute measurement. They also control the ratio of delivery of the gases. There is a nasal mask system usually for delivery of the gases and a scavenger system to collect excess gases. These systems usually have a failsafe shutoff for the nitrous oxide if the concentration of oxygen falls below 22%

Inhalation Anaesthesia Machine

Full function anaesthesia machines are made up of two main parts, a vaporizer and the ventilator. They also incorporate a suction unit and patient monitoring devices. The most common type in use is called a continuous-flow anaesthetic machine which provides an accurate and continuous supply of medical gases along with an accurate concentration of anaesthesia vapor. A modern machine contains the following components:

- Connections to piped hospital oxygen, medical air, and nitrous oxide.
- Gas cylinders of oxygen, air, and nitrous oxide attached via a specific yoke.
- Pressure gauges, and regulators with “pop-off” valves
- A high-flow oxygen system which provides pure oxygen at 30-75 liters/minute
- Vaporizers to provide accurate dosage control when using volatile anaesthetics such as Isoflurane and Sevoflurane
- Flow meters for oxygen, air, and nitrous oxide
- An integrated ventilator to properly ventilate the patient
- A manual ventilation bag
- Systems for monitoring the gases being administered to, and exhaled by the patient
- Systems for monitoring the patient's heart rate, ECG, blood pressure and oxygen saturation, in some cases with additional options for monitoring end-tidal carbon dioxide and temperature

![Nitrous oxide Oxygen Delivery System](image)

**Figure 5-22: Nitrous oxide Oxygen Delivery System**

**Airway Equipment**

**Suction**

Adequate suction should be available in both the recovery area and in the operatory. In addition the crash cart should have a portable battery operated suction unit. Suction function should be checked daily.

**Airway Adjuncts**

**A. Basic Airway Management**

Dealing with an anatomical functional blockage of the airway from relaxation of patient tissues and their tongue is a common situation staff must be able to respond to. The first steps if signs of a partial or complete airway obstruction occur are to reposition the patient's head, jaw, and neck. The **head tilt-chin lift** is the initial maneuver used to open the airway in these situations (Fig. 5-23). If the patient is not supine then a modified jaw thrust with some head tilt can be applied. If necessary the patient may need to be quickly brought into a supine position. The **modified jaw thrust** refers to a jaw thrust with the addition of head tilt. The **jaw thrust with or without head tilt** is more effective in displacing the tongue anteriorly with the mandible and hyoid than is the head tilt/chin lift.
If the situation dictates that the patient will have a loss of protective reflexes for a prolonged period of time then the use of oral or nasal pharyngeal airway should be considered. Both prevent the tongue from occluding the airway and provide an opening for spontaneous breathing or positive pressure ventilation with a BVM.

Figure 5-23: Head tilt-chin lift

Figure 5-24: Modified Jaw Thrust
Oral airways (Fig. 5-25) are placed in adults upside down then rotated into position as the person inserting feels the tip of the airway hitting the soft palate (Fig. 5-26). In toddlers they are placed in with the same orientation as they will sit in (without the upside down rotation) to minimize the risk of soft palate trauma. Sizing is from the lips to the angle of the jaw (Fig. 5-27).

Once a patent airway is established the patient may begin adequate spontaneous breathing. As these airway procedures are being undertaken other staff must decide to use reversal agents if opioids or benzodiazepines were used.
If it is determined that the patient is apneic or hypoventilating then staff will have to ventilate the patient with a bag-valve-mask (BVM). This device is not easy to use for many individuals. The flow of oxygen from the outlet should be sufficient so that the reservoir bag stays filled or partially filled at all times. Flows of 12-15 LPM will insure 100% oxygenation in adult patients. The use of two people to ventilate will allow for successful delivery of respirations in most patients. The person squeezing the bag should deliver each breath over 1-2 seconds using enough volume to just see the chest rise. Excessive inspiratory pressures can lead to the passage of air down the esophagus and subsequent regurgitation. Ventilate at 10-12 breaths per minute or to pulse oximetry readings. Avoid hyperventilation. The second person holds the mask forming a seal with two hands (Fig. 5-29).
Figure 5-29: BVM Technique

For the general management of hypoxia both nasal cannula and face masks can be used to increase the inspired percentage of oxygen. **Nasal Cannula (Fig. 5-30) delivers oxygen concentrations of between 28-44%, at flow rates of 1-5 lpm.**
Simple face masks (SFM) can be used with oxygen flow rates of 6-10 lpm. The concentration of inspired O2 is determined by how much air the patient is breathing at any given moment which leads to mixing with room air. Estimates are between 40-60% oxygen concentrations with a SFM.

Figure 5-31: Simple Face Mask (SFM)

A nonrebreather mask is similar to a SFM except it has an oxygen reservoir bag and valves to prevent the breathing of room air as long as the mask is tight fitting. Oxygen flow rates should be between 12-15 lpm, the key being that the reservoir bag never collapses flat. Concentrations of inspired oxygen range from 60-80%.

Figure 5-32: Nonrebreather Mask
B. Advanced Airway Management

Advanced airways are placed in non-emergent situations as part of the induction GA process. They can also be placed in emergency situations. Most patients undergoing anaesthetic procedures in a dental/OMS setting do not have full GA but instead moderate or deep sedation. This means that they have an open patent airway and are breathing on their own.

When staff is failing to ventilate a patient in respiratory arrest a decision has to be made to place an advanced airway. An advanced airway is a device that isolates the trachea from the esophagus and directly allows for ventilation of only the lungs. It is fitted with a universal adapter on the end to attach to a bagging unit without a mask on the face (failure to get a seal with the mask is the leading reason for BVM failure). Traditionally the advanced airway was an endotracheal tube placed by intubation into the trachea, but now several easier to place devices are available such as the Laryngeal Mask Airway (LMA) and the King Airway. See Figures 5-33 and 5-34. These devices do not go past the vocal cords and into the trachea. Such devices are called supraglottic or extraglottic airways.

![Figure 5-33: The King Laryngeal Airway](image)

**Distal Cuff**
Inflates in the esophagus. Isolates the laryngopharynx from the esophagus.

**Proximal Cuff**
Inflates at the base of the tongue. Isolates the laryngopharynx from the oropharynx and nasopharynx.
The issues with intubation, the “golden” standard for advanced airway management, are that many healthcare providers who are allowed to intubate cannot easily undertake a direct laryngoscopy intubation as they do not do the skill frequently enough to maintain a high level of efficiency and expertise. The LMA and King Airway are easier to place requiring no direct visualization of the glottal opening. These newer types of alternative airways should be readily available in the operatory.

If intubation is undertaken for a GA it is usually a nasal intubation so that the endotracheal tube does not interfere with the surgical site. After insertion, great care must be taken to ensure that the tube is in the right position. If it has been accidentally placed in the esophagus then the patient will receive no ventilations. If it is placed too
deep into the trachea then only one lung may be ventilated (due to the lesser angle of the right mainstem bronchi the tube usually goes down the right side).

To ensure correct placement: the chest is auscultated bilaterally for breath sounds, the upper left quadrant of the abdomen is auscultated for gurgling sounds indicating an esophageal intubation, the chest is visualized for equal expansion, and a good ETCO₂ wave form is confirmed via capnography.

Figure 5-36: Nasal Intubation

Module 5 Quiz

The module 5 quiz can be found at the following web address: https://testing.exambuilder.com/login

Contact Michael Dare at mdare@dentaled.ca if you did not receive a student ID and password.
Module 6: Anaesthetic Emergencies

Key Areas for Study

- Hypoventilation, hypoxemia, hypercarbia
- Airway Obstruction, all causes
- Hyperventilation
- Hypotension
- ACS
- Hypoglycemia
- Seizures
- Syncope
- Allergic reactions and anaphylaxis

Part 1: Emergency Equipment Needs

Emergency equipment needs depend on the type of PSA/GA that is being delivered along with the depth of sedation being undertaken. During moderate and deep sedation the ASA recommends that a defibrillator be immediately available for all patients. Pharmacological agents used to reverse both opioids and benzodiazepines need to be available along with the resuscitation medications common to a cardiac arrest crash cart. Appropriately sized airway equipment for delivering oxygen, suction, positive pressure ventilation, and the establishment of a patent airway need also be available.

Example of emergency equipment needed for PSA/GA

- Intravenous equipment
- Gloves
- Tourniquets
- Alcohol wipes
- Sterile gauze pads
- Intravenous catheters
- Intravenous tubing sets
- Intravenous fluids
- Assorted needles for drug aspiration, intramuscular injections
- Intravenous access device
- Appropriately sized syringes
- Basic airway management equipment
- Source of compressed oxygen (tank with regulator or pipeline supply with flowmeter)
- Source of suction
- Suction catheters
- Yankauer-type suction
- Face masks
- Self-inflating breathing bag-valve set
- Oral and nasal airways
- Lubricant
- Advanced airway management equipment
- Laryngeal mask airways
- Laryngoscope handles (tested)
- Laryngoscope blades
- Endotracheal tubes
- Pharmacologic Antagonists
  - Naloxone
  - Flumazenil
- Emergency medications
  - Epinephrine
  - Ephedrine
  - Vasopressin
  - Atropine
  - Nitroglycerin (tablets or spray)
  - Amiodarone
  - Lidocaine
  - Glucose 10, 20, or 50%
  - Diphenhydramine
  - Hydrocortisone, methylprednisolone, or dexamethasone
  - Diazepam or midazolam
  - Malignant hyperthermia kit

Part 2: Adverse Events in PSA/GA

This section describes the factors that lead to adverse events, what those adverse events are, and the likelihood of those events occurring.

Safety: Human and Organizational Factors

Delivering PSA/GA safely is a multistage process that starts before even meeting the patient. There needs to be a safe and reliable anaesthesia service design which allows for the free flow of communication, a team which adopts a culture of safety.

A clinical microsystem (CM) is a group of staff and clinicians working together to provide care to a population of patients with a shared clinical purpose. A PSA/GA team is an example of such a microsystem. CM provides a conceptual framework for approaching organizational learning and the delivery of safe PSA/GA. To improve safety it is necessary to study the components that make up the system which are humans, technologies and their complex interactions.

There are two reasons for adverse events during PSA/GA

- All human beings, regardless of their skills, abilities, and specialist training, make poor decisions and commit unsafe acts. This human tendency for committing errors and violating safety procedures during tasks can be moderated by selection, training, well-designed equipment, and good management, but it can never be entirely eliminated.
• No matter how well designed, constructed, operated, and maintained they may be, all man-made systems possess latent failures to some degree.

Human error and performance limitations have been extensively studied in several industries such as aviation, road and rail travel, and nuclear power for several decades. Theories of error and accident causation have been developed for other human activities although they have just started to be applied to the field of medicine. One common realization from this type of research is that errors are rarely isolated to one individual’s actions. Adverse event analysis may often reveal deep-rooted unsafe features of an organization. This has led to a much broader understanding of accident causation, with less focus on the individual who makes an error and more on the organizational structures that allow the error to occur.

Adverse events that occur with systems/organizations which have a wide number of technical and procedural safeguards have been termed organizational accidents. These events do not arise from a single error but instead by the accumulation of errors that started well before the event in question. One of the basic principles of error management is that the transitory mental states associated with error production – momentary inattention, distraction, preoccupation, forgetting – are the least manageable links in the error chain because they are unintended and largely unpredictable.

The Design of a Safe Anaesthesia/Dental Team

The delivery of safe PSA/GA relies on coordinated teamwork, good communication skills, a culture of learning not a culture of blame, and trust between team members. Factors that lead to errors include:

• Poor communication between team members.
• Poor training of the team leader in team dynamics and leadership.
• Not having enough dedicated team members.
• Reluctance to question the leader or other senior team members.
• Failure to establish and maintain consistent supportive organizational infrastructure.
• Absence of experienced team members.
• Failure of members to function as part of a team.
• Failure to establish and maintain consistent supportive organizational infrastructure.

One application of processes established in anesthesiology is a modification of the “time out” team concept of preoperative verification of the correct patient, procedure, site, and implants. There is an active discussion among all team members and the procedure is not started until all questions and concerns are resolved. A similar “time out” approach for a dental OMS team might include:

• Correct patient.
• Correct procedure.
• Patient appropriate for PSA/GA.
• Correct drug(s) for type and length of procedure.
• Correct drug dosages checked by two team members.
• Correct safety equipment operable and ready.
• Correct location for the patient and the type of procedure.
• Adequate staffing for the procedure and PSA/GA.

All team members understand that they are all safety “gatekeepers” and that all concerns and questions will be answered before starting.

Wherever PSA/GA is undertaken, there must be emergency protocols in place which delineate the actions necessary in the event of an emergency, what the individual staff responsibilities are, what emergency equipment is required, where it is located, and what training program is needed to maintain emergency response competencies. In an out of hospital setting such as a dental office these protocols would look vastly different to those of a large hospital emergency department. Who calls 911 if there is an emergency? Who goes down to guide the paramedics up to the office? Who brings the crash cart into the operatory? All of these issues must be dealt with and an emergency plan put into place. Staff should be trained appropriately for their roles- CPR, use of an AED or manual defibrillator, and use of a BVM. Mock simulations on dealing with PSA emergencies should be regularly undertaken with all staff that provides PSA/GA.

Specific Emergencies That Occur with PSA/GA

Respiratory System Complications

The vast majority of adverse events in PSA are of a respiratory nature. They can usually be attributed to oversedation or an unexpected response to the agents by the patient. The signs and symptoms include respiratory depression, hypoventilation, apnea, hypoxemia, hypercapnia, laryngospasm, bronchospasm and complete airway obstruction.

A. Hypoventilation, Hypoxemia and Hypercapnia

Hypoventilation and the resulting issues of decreased blood oxygen levels and rising blood CO₂ content are a potentially serious adverse event of PSA/GA. Hypoventilation can be attributed to relaxed laryngeal tissues that increase airflow resistance and obstruction, along with a decreased CNS respiratory drive caused by the agents used. Patients who are ill and/or have chronic disease processes may not metabolize medications in a predictable manner. The complex pharmacokinetics and pharmacodynamics of agents with these patients can lead to cardiopulmonary compromise at doses not normally attributed to causing those types of events. Benzodiazepines and opioids are two PSA/GA drug categories that are often linked to respiratory adverse events. When used in combination, their effects are synergistic on the respiratory system. Oversedation can be prevented by:

• Slow titration of agents over a period of time. Avoid rapid boluses.
• Avoid giving multiple agents simultaneously. Administer the agent with the most depressing action on the respiratory system first and then after a period of time the second agent is given again using small slowly administered boluses.
• Take into consideration the physiological status of the patient (renal and hepatic issues, BMI, dehydration, poor cardiac function, age) and adjust drug choices and dosing accordingly.
Treatment usually involves stimulation of the patient by asking them to breathe deeply combined if needed with mild to moderate tactile stimulation. Increasing supplemental oxygen flow rates or switching delivery method may assist with hypoxemia. Repositioning of the patients airway if it is partially blocked may be needed. If these interventions do not work then the consideration should be given to the use of reversal agents such as naloxone or flumazenil. If needed assisted ventilations using a BVM can be undertaken.

B. Airway Obstruction

Obstruction of a patient's airway can be caused by relaxed muscle tone of the submandibular muscle and surrounding tissues leading to upper airway obstruction from the tongue or a closed epiglottis, laryngospasm, bronchospasm, secretions, or foreign body obstruction (FBO). Obstruction from the patient's own anatomy can usually be managed with proper patient position and simple airway maneuvers.

a. Foreign Body Airway Obstruction (FBAO)

The risk of this emergency is heightened in dental/OMS settings due to the nature of the surgical procedures being done and their location. Signs of an acute FBAO include

- If 100% obstructed no ability to cough or vocalize in any way
- Paradoxical chest wall and abdominal motions (seesaw motions)
- Apnea alarm from ETCO₂ monitor
- Upper airway high pitched noises called stridor if partially obstructed.
- The indrawing of soft tissues in the thoracic cage such as between the ribs (intercostal indrawing), in the suprasternal notch, and supra-claviculary.
- Use of accessory muscles of breathing
- The patient will become tachycardic and eventually their oxygen saturation will start dropping
- Late stages: loss of consciousness and full cardiovascular collapse

For a full obstruction treatment includes:

- Doing the Heimlich maneuver for the awake patient
- For the patient under moderate or deep sedation compressions of the chest or an upward series of abdominal thrusts can be attempted with the patient in the dental chair or supine. The idea is to create positive pressure in the thoracic cage to blow the obstruction outwards like an artificial cough.
- Suctioning or the oropharynx
- Use of Magill forceps to manually remove the object if accessible
- Direct laryngoscopy to see if removal of the foreign body can be done with suction or forceps
- Cricothyrotomy- most often done with a special kit which has all of the required items.

b. Laryngospasm

This is a form of airway obstruction caused by a tonic contraction of the glottis muscles including the true and false cords. It is much less common
with PSA than general anesthesia. Factors associated with causing laryngospasm are:

- Airway manipulation including with the use of oral or nasal pharyngeal airways
- Excessive secretions
- Vomitus
- Suctioning
- Preexisting respiratory infection

Laryngospasm can usually be managed with:

- Deliver 100% oxygen
- Establish proper patient positioning for an open airway such as a head tilt chin lift or jaw thrust.
- Stop bleeding from the surgical site
- Suction the deep posterior pharynx
- **Positive pressure ventilation with a BVM**
- On occasions medications are used and the patient is paralyzed and intubated.
- **Application of firm pressure bilaterally in the laryngospasm notch** (Fig. 6-1)

Figure 6-1: Laryngospasm Notch

c. **Bronchospasm**
This occurs as a lower airway obstruction due to increased bronchial smooth muscle tone. The signs and symptoms the patient presents with depend on the degree of bronchospasm. **Mild signs may be a slight wheeze only detectable by stethoscope. More severe bronchospasm may cause audible (heard without a stethoscope) wheezing, chest tightness, use of accessory muscles, indrawing, and severe dyspnea.**
Factors for bronchospasm development include having reactive airway disease, release of histamines, upper respiratory infections, aspiration, excessive secretions, and bronchial irritation from suctioning. Propofol has been linked to causing bronchospasm. It may result from an anaphylactic allergic reaction independently or in combination with laryngeal edema. More commonly, it is a consequence of the hyperreactive airway typical in patients with asthma. Regardless of the cause for bronchospasm, the patient will exhibit dyspnea and wheezing attributed to obstruction in the chest, not the throat or mouth. Bronchial smooth muscle is under autonomic nervous control and requires beta-2 sympathomimetics for relaxation such as ventolin. Following primary assessment, including oxygen supplementation, ventolin should be administered by a metered inhaler or by nebulization. Other treatments such as steroids can wait until later as their onset of action is much delayed. If ventolin therapy and oxygen do not alleviate the symptoms activating 911 may be needed.

C. Negative pressure pulmonary edema (NPPE)
This is a pulmonary complication that can rarely occur in patients when a patient breathes against an acute upper airway obstruction creating high negative intrathoracic pressures. This leads to a shift of fluids from pulmonary capillary beds into alveoli. Patients at risk of developing NPPE include those with a history of sleep apnea, laryngospasm, FBO, obesity, and opioid use. In dentistry sedation the risk of foreign body obstruction and aspiration can be significant due to the location of the procedure. Management is to maintain an airway free of obstruction and provide high flow oxygenation. On occasion patients who do not improve must be placed on a ventilator and have other management. This would be done in a hospital setting.

D. Hyperventilation
Hyperventilation occurs when a patient is breathing deeper and faster than required leading to the exhalation of too much CO2. It may be caused by anxiety, fear, pain, or medications the patient has taken. When the patient exhales too much CO2 they may feel light headed and anxious and may start to have tingling in their fingers and even tetany, the involuntary contraction of muscles. Treatment consists of terminating whatever is happening that may be causing increased fear and anxiety and trying to calm the patient and coach them to breathe slower. Do not give oxygen (unless hypoxia has been confirmed) or have the patient breath into a paper bag. If another more serious cause of hyperventilation is occurring or the patient has been misdiagnosed the use of a paper bag can be dangerous and harmful. The use of anxiolytics can be attempted if more serious causes of tachypnea have been ruled out.

E. Pulmonary Aspiration
The incidence of pulmonary aspiration in PSA/GA is low. If it does occur pulmonary aspiration can cause a variety of sequelae depending on the type of aspirate. Signs and symptoms include crackles, dyspnea, wheezes, tachycardia and tachypnea, and oxygen desaturation. Many drugs used for PSA/GA can induce nausea and vomiting while obtunding (blocking, blunting) protective reflexes. Patients at risk for aspiration are those with increased intra-abdominal pressure, poor gastric emptying, or gastric esophageal reflux disease (GERD). Increases in intra-abdominal pressure can be found in pregnant patients and in patients with ascites for example. Aspiration of GI particulate leads to acidic
gastric contents damaging the endothelial lining of the lungs. Teeth and instrument aspiration can lead to blockages within bronchi or bronchioles.

Treatment for aspiration includes:
- Protect A, B, Cs
- 100% oxygen
- Activate ambulance as warranted
- Oropharyngeal suctioning, laryngoscopy, forceps removal of objects within reach
- Turn patient onto their right side in a slight Trendelenburg position
- Intubation or other advanced airway insertion along with ventilation support as needed depending on severity of situation

Cardiovascular System Complications

A. Hypotension

Hypotension is the most common cardiovascular adverse event found with PSA/GA. The mechanism for hypotension is a decreased sympathetic tone which leads to vasodilation of vascular beds. This in turn leads to peripheral pooling of blood and decreased preload. The other mechanism for some PSA agents is that they have a direct negative contractility effect on the heart. The physiologic status of the patient can contribute to the degree of hypotension also—preexisting heart disease, dehydration, blood loss, and vasovagal response. Treatment should be directed at identifying and treating the causative factor for the hypotension. General management includes;

- Consider IV fluid bolus (rapid infusion of a specific quantity of fluid)
- Placing the patient in a modified Trendelenburg position
- Support A, B, Cs and give 100% oxygen
- Give drugs such as Phenylephrine or Ephedrine if it is determined that hypotension is due to the standard negative properties of the PSA/GA agents.
  - Phenylephrine 50-100 mcg IV
  - Ephedrine 2.5-5 mg IV
- Active 911 if a serious cause is identified that cannot be easily managed in the outpatient setting.

Figure 6-2: Modified Trendelenburg Position
B. Hypertension (HTN)
HTN can occur during PSA/GA. It may be an indication that the patient has had inadequate PSA analgesia for a given procedure. The stress and pain then leads to an autonomic sympathetic release. Epinephrine containing local anesthetics can also lead to hypertension when larger quantities are used. This can precipitate a hypertensive crisis in patients with preexisting hypertension. Generally a systolic pressure of 200 mmHg or higher or a diastolic pressure of 100 mmHg or higher requires evaluation and treatment. The patient may have signs and symptoms such as dizziness, headache, chest pain, and shortness of breath or they may be asymptomatic. Treatment should be conservative in an office setting but may include;

- Treat underlying causes if one can be identified
- Consider giving drugs such as beta blockers (labetalol, esmolol)
- Consider 911 activation

C. Acute Coronary Syndrome (ACS)
The signs and symptoms of angina and MI are similar and these two types of ACS cannot be necessarily differentiated in an office setting. A series of 12-18 lead ECGs and cardiac blood work are needed to determine the category of ACS a patient is experiencing. One of the questions that often needs answering is whether or not a chest pain episode is serious or not? Let's look at the signs and symptoms that occur and when a procedure needs to be terminated and an ambulance called.

a. Stable Angina
Angina is a category of ACS that is further divided into stable and unstable angina. It is a symptom and not a diagnosis. It is associated with chronic and gradual narrowing of one or more coronary arteries and a loss of the ability to fully vasodilate in the region of the narrowing. The signs and symptoms of angina include chest pain, heaviness, tightness, squeezing or discomfort that may radiate to the neck, jaw, teeth, shoulders, and/or arms. It is brought on by exertion or emotion and it is relieved by rest and/or nitroglycerin. If the same pattern causes the discomfort each time, if the discomfort is described as the same and the same treatment gives relief then the patient has stable angina. If a patient has an episode of stable angina at the dental/OMS office and it responds as stable angina then the surgery/procedure may still be carried out at the discretion of the team.

b. Unstable Angina
Unstable angina has the same signs and symptoms as stable angina but

- The chest pain has changed in severity, is more prolonged and usually lasts for > than 15-20 minutes.
- What initiates the onset of pain has changed and pain can occur at rest or awaken the patient at night
- The amount of nitrates needed to get relief from signs and symptoms is increasing

The pattern of the patient’s angina is not stable. Also any new onset of angina like symptoms is considered unstable angina in a patient who has
never experienced angina symptoms before. A patient experiencing the signs and symptoms of unstable angina needs immediate medical treatment and an ambulance should be called.

c. Myocardial infarction
The diagnosis of an MI will not be made in the dental office setting; however the signs and symptoms of an MI do differ from those of angina. Signs and symptoms typical of an MI are
- Chest Pain or discomfort which may be described as a heaviness, tightness, fullness, or squeezing sensation. The pain does not go away with rest and nitrates. It persists and is most often worse than that of angina.
- Radiation of the discomfort to other areas like the jaw, neck, shoulders, back, abdomen, and arms.
- SOB
- Dizziness
- Nausea and vomiting
- Sweating

Women, the elderly and diabetics often have the most atypical signs and symptoms. Diabetics can have a silent MI which is only detected at a later date usually by accident.

Other medical conditions can cause ischemic like symptoms such as an aortic dissection, a pulmonary embolism, a tension pneumothorax, or a musculoskeletal injury. When in doubt it is always best to seek medical attention for the patient

Treatment for unstable angina and signs and symptoms suspicious of an MI include

- Stopping the procedure.
- Attach to cardiac monitor and obtain a full set of vital signs and repeat frequently. Make sure crash cart is readily accessible.
- Ensure good IV access.
- Give a 0.4 mg dose of nitroglycerin SL by tab or spray after checking that the patients' BP is not hypotensive.
- If the chest pain is worse or unchanged after five minutes then 911 should be activated. Nitroglycerin 0.4 mg SL can be given every five minutes as long as the patient does not become hypotensive. (Systolic BP<100 mmHg or MAP < 60 mmHg).
- Apply oxygen to maintain oxygen saturation to between 94-98% Avoid hyper-oxygenation (O₂ Sat > 98%) as there is evidence that this can be harmful.
- Have patient chew and swallow 325 mg ASA (four baby aspirin) if there are no contraindications. Make sure drug is clear of teeth so that the full dose is received.
- Allow patient to assume a position of comfort.
- Monitor for dangerous/lethal arrhythmias and treat as needed.
D. Arrhythmias
Many cardiac arrhythmias are of no life threatening consequence, and are best not managed in the office setting. Only rhythms which put the patient in immediate danger should be managed in an outpatient setting.

a. Sinus Tachycardia
The most common arrhythmia seen in PSA/GA is a sinus tachycardia. Like with hypertension, the most common cause is inadequate levels of PSA/GA. Other more ominous causes need to be ruled out such as hypotension and hypoxemia. Appropriate treatments for these underlying causes should be instituted.

b. Symptomatic Bradycardia
If the bradycardia is caused by vagal stimulation stop whatever is producing the reaction. Most often the patient’s blood pressure and heart rate will recover with no treatment. If it persists glycopyrrolate or atropine may be given. If the bradycardia is more ominous then calling 911 may be necessary. Certain bradycardic rhythms are more dangerous. One is called a complete heart block and it may require a pacemaker. If people trained in Advanced Cardiac Life Support (ACLS) are present other interventions may be undertaken like cardiac pacing of the patient using the crash cart monitor.
c. **Supraventricular Tachycardia (SVT)**

This rhythm rarely causes enough instability to need treating in an office setting. If the patient has a history of SVT’s they may be trained to do **vagal maneuvers** such as holding their breath and bearing down which may terminate the SVT. If this is the first time for the arrhythmia then management is best done at a hospital. 911 should be called if the patient shows any signs of poor cardiac output. If the patient is experiencing hypotension the dentist or surgeon may administer a medication to terminate the rhythm such as **Adenosine**.

d. **Ventricular Tachycardia**

This is a very dangerous heart rhythm. 911 should be immediately called. If the patient is pulseless then CPR should be started and the patient defibrillated. The ACLS algorithm called **VF/Pulseless VT** should be followed and the team optimally led by an ACLS trained practitioner. **Support staff should be doing highly effective CPR with minimal interruptions and changing the chest compressor every two minutes. This will mean moving the patient to the floor as soon as possible**

If the patient has a pulse and is in VT then the decision may be made to sedate the patient (if they are not already sedated or under GA) and do a **shock similar to a defibrillation called a cardioversion.**

e. **Atrial Fibrillation and Atrial Flutter**

These two rhythms rarely cause serious patient instability needing treatment in an office setting. Treatment may be needed if the ventricles are firing too fast. To slow the firing of the ventricles, the patient may be given an **IV beta blocker or calcium channel blocker.** If a patient suddenly goes into a rapidly firing atrial fibrillation or flutter they will need to be cardioverted with a shock. Most often the patient will be stable and therefore should be transported to the closest ER if warranted.
Endocrine Disorders

A. Hypoglycemia

If a patient’s blood sugar falls below 4 mmol/l they may begin to show signs and symptoms of hypoglycemia. Initial symptoms may include nausea, tachycardia and a feeling of hunger. As the sugar continues to drop the signs and symptoms will worsen and include restlessness, agitation, anxiety and lethargy. At dangerously low levels the patient will lose consciousness and perhaps have seizures.

Treatment includes

- Call an ambulance if the reaction is severe
- Establish an IV
- Give IV glucose solution such as D50W, D10W, or D5W
- If no IV access give Glucagon
- If patient awake and can eat and drink give a high sugar containing product like juice as a drink followed by complex carbohydrates like crackers and cheese.
B. Acute Adrenal Insufficiency
This occurs when there is inadequate production of cortisol by the adrenal gland. It can occur when a patient's own production is inadequate or when the taking of steroid medications has suppressed production. Symptoms include hypotension, tachycardia, confusion, diaphoresis, fatigue, fever, nausea and abdominal pain. Treatment includes

- Stop procedure
- Activate 911
- IV access and fluid bolus if hypotensive
- Modified Trendelenburg position
- Give Dexamethasone 4 mg IV or Hydrocortisone 100 mg IV
- Monitor vitals

The Nervous System

A. Seizures
A full body generalized tonic-clonic (previously called a Grand Mal) seizure can be alarming to see. It involves a loss of consciousness and abnormal muscle contractions which leads to flailing and abnormal body posturing. Most seizures are self-limiting and last only one or two minutes. The primary goal while the seizure is occurring is to protect the patient from injury. Loosely control limbs as much as possible. Remove any objects and instruments from the area around the patient. Loosen tight clothing. During the seizure the jaws are predominately locked shut and the respiratory muscles are in clonic contractions so no effective breathing is occurring and no airway management can be done. If the seizure becomes prolonged treatment with a benzodiazepine such as midazolam 3-6 mg IV or Ativan 1-2 mg IV may be needed. When generalized seizures last greater than five minutes it is called status epilepticus. If breathing is interfered with for too long the patient may show signs of severe hypoxemia such as cyanosis. The patient is often incontinent of urine. Once the seizure stops the patient enters into what is known as a postictal state in which they have an altered level of consciousness that lasts between 5-30 minutes. Initially they should be assessed for adequate breathing and have breathing assisted with a BVM if needed. They should be placed in a recovery position until they are awake enough to maintain their own airway and assessed for injury. If they are experiencing a refractory seizure or having repeat seizures then 911 should be called.

B. Syncope and Vasovagal Reactions
Syncope is one of the most common cardiovascular complications of PSA. It can be associated with hypotension, hypoxemia, or even on occasion cardiac dysrhythmias. Most syncope related to PSA is a combination or syncope due to drug induced orthostatic hypotension and reflex syncope from a vasovagal source. The vasovagal reflex syncope is induced mainly from a vagal tone induced bradycardia and resulting low cardiac output. This may be exacerbated by drug induced or volume depletion hypotension leading to decreased venous return. Presyncopal signs and symptoms include pallor, nausea, and lightheadedness. Seizure activity can occur briefly with a syncopal episode due to lack of blood flow temporarily to the brain. If the presyncopal signs and symptoms are recognized early enough syncope can be avoided. Rapidly place
the patient supine or in a slight Trendelenburg position, open the IV wide if one is in place. Apply cold compresses to the forehead and administer oxygen.

Treatment includes:

- Maintain A, B, C’s.
- Place patient in a supine position.
- Consider modified Trendelenburg.
- Consider fluid boluses as per hypotension treatment guidelines.
- Immediately stop any triggering activity.
- For persistent bradycardia consider atropine.
- Vitals, cardiac monitoring.
- If dysrhythmias detected treat as per ACLS guidelines.
- If a serious causative factor is identified consider calling 911.

C. Cerebrovascular Accident (CVA)
A CVA or stroke can be caused by either a rupture of a blood vessel (hemorrhagic CVA) or a clot occluding a vessel (ischemic CVA). If the neurological impairments persist for greater than 24 hours it is defined to be a CVA. If the signs and symptoms resolve before 24 hours it is called a transient ischemic attack (TIA). Patients can present with unilateral (one sided) weakness or paralysis, headache, inability to speak or slurred speech, numbness, and ataxia (poor balance). Other symptoms can be present; it is dependent on the location of the CVA. “Time is brain” is the saying when a CVA is suspected. Some of the treatments are most effective when given early and they cannot be given at all if a certain amount of time has passed. Treatment for a CVA in an office setting includes:

- Rapid activation of 911
- Support A, B, C’s and place patient in position of comfort
- Oxygen by mask or nasal prongs
- IV Access

The Immune System

A. Mild Allergic Reactions
Signs and symptoms of an allergic reaction include hives, pruritus (itching), and angioedema (swelling- face, lips, perioral areas), and erythema.

Treatment includes:

- Benadryl 25 mg IV or IM, or 50 mg po
- Possibly epinephrine 1:1000 0.3 mg IM if more severe symptoms
- IV access maintained
- Monitor vitals

B. Anaphylaxis
Anaphylaxis is likely when all of the following 3 criteria are met:

- Sudden onset/rapid progression of symptoms.
- Life-threatening airway and/or breathing and/or circulation problems.
• Skin and/or mucosal changes such as flushing, urticaria, angioedema are present

Remember:
• Skin or mucosal changes alone are not a sign of an anaphylactic reaction.
• Skin and mucosal changes can be subtle or absent in up to 20% of reactions. (some patients can have only a decrease in blood pressure)
• There can also be gastrointestinal symptoms (e.g. vomiting, abdominal pain, incontinence).

Signs and symptoms include skin reactions, severe angioedema, gastrointestinal cramping, bronchoconstriction, progressive upper airway obstruction, urinary incontinence, and cardiovascular collapse. Treatment includes:

- Call 911
- Maintain or establish IV
- Epinephrine 1:1000 0.3-0.5 mg IM or epinephrine 1:10000 0.2-0.5 mg IV
- Place patient supine
- IV fluid boluses for hypotension
- Maintain a patent airway with advanced airway management or cricothyrotomy
- Benadryl 50 mg IV
- Consider corticosteroid IV

Other Emergencies

**Malignant Hyperthermia (MH)**
This is a genetically transmitted disorder of the muscles that can be triggered by certain anaesthetic agents (volatile anaesthetics) and by succinylcholine, a muscle paralyzing agent used in anaesthesia and airway management. It leads to a hypermetabolic state of skeletal muscle oxidative metabolism. The first sign
of MH is tachycardia followed after by masseter muscle rigidity, pyrexia, total body rigidity, increasing ETCO₂ levels, and tachypnea. If untreated it rapidly leads to death.

Treatment:

- Activate 911
- Immediately administer the muscle relaxant Dantrolene rapid IV push, 2.5 mg/kg
- Hyperventilate with 100% oxygen
- Icepacks to groin, axilla, and neck.
- Cold NS IV fluids 15ml/kg every 15 minutes x 3
- Rapid transport to hospital

Module 6 Quiz

The module 6 quiz can be found at the following web address: https://testing.exambuilder.com/login

Contact Michael Dare at mdare@dentaled.ca if you did not receive a student ID and password
Glossary of Terms

1. **Acute coronary syndrome (ACS):** Encompasses all of the heart diseases related to the formation of plaque in the walls of coronary vessels. This includes the various types of angina and heart attacks.

2. **Adenosine triphosphate (ATP):** A fundamental energy holding molecule of the human body. Most processes in the human body are driven by the energy released when ATP loses a phosphate to form adenosine diphosphate.

3. **Adrenergic Receptors:** Receptor sites throughout the human body that are targets of the catecholamines, especially norepinephrine and epinephrine.

4. **Aerobic:** In the presence of oxygen.

5. **Afferent:** Carrying information from the body back to the CNS.

6. **Afterload:** The amount of resistance there is to moving blood out of the heart. Diastolic blood pressure is a good indicator of a patient’s afterload.

7. **Agonist:** A chemical that binds to a receptor and activates the receptor to produce a biological response.

8. **Alveoli:** The small grape-like structures in the lungs where gas exchange occurs with the capillaries of the bloodstream.

9. **Anaerobic:** In the absence of oxygen.

10. **Anaesthesia:** The condition of having sensation removed or temporarily blocked. It is induced pharmacologically and can include amnesia, analgesia, unconsciousness, a decreased stress response, and the loss of skeletal muscle reflexes.

11. **Anemia:** A low hemoglobin concentration.

12. **Angina:** One type of ACS in which there can be adequate blood flow to supply a region of the heart past a plaque deposit, but if the equilibrium between oxygen supply and demand (exercise, anxiety, stress) is impacted then signs and symptoms of ischemia will occur.

13. **Antagonist:** A chemical that binds to a receptor site and blocks the normal agonist chemicals from activating the receptor.

14. **Anticholinergics:** Refers to drugs that block the action of the parasympathetic nervous system.

15. **Anxiolytic:** An antianxiety agent.

16. **Aortic valve:** The valve located in the left ventricle as blood leaves to enter the aorta.

17. **Apnea:** The cessation of breathing.

18. **Auscultation:** The act of listening to sounds arising within organs (as the lungs or heart) as an aid to diagnosis and treatment.

19. **Automaticity:** The characteristic of cells to fire electrically on their own without any outside stimulation.

20. **Autonomic nervous system (ANS):** Controls body functions below the level of consciousness such as respiratory and cardiac regulation. Contains the sympathetic and parasympathetic nervous system divisions.

21. **Arrhythmia:** An abnormal heart electrical rhythm.

22. **ASA:** American Society of Anaesthesiologists.
23. **Ataxia:** Loss of balance leading to a poor gait.
24. **Atria:** Upper chambers of the heart.
25. **Atrial kick:** The blood that the atria injects into the ventricle before ventricular systole. Makes up between 15-30% of the volume in the ventricle before ventricular systole.
26. **Atrioventricular node (AV node):**
27. **Atropine:** A drug that block the parasympathetic nervous system’s actions commonly used to dry secretions in the mouth and to speed up heart rates.
28. **Axon:** Neuron impulses initiated/transferred are carried onwards down a long neural fiber called an axon.

**B-C**

1. **Balanced anaesthesia:** The use of several agents to create a combination which takes from the best aspects of each agent.
2. **Bolus:** A dose of a medication or other pharmaceutical preparation injected all at once intravenously.
3. **Buffer:** A chemical that binds with hydrogen ions.
4. **Bundle of His:** A tract of conductive tissue that branches down from the AV node and penetrates through the fibrous nonconductive tissues that separate the atria and ventricles.
5. **Brain stem:** Area at the base of the brain containing the pons, the medulla oblongata, and the midbrain. Plays an important role in the control of cardiac, vasomotor and respiratory function.
6. **Bronchospasm:** Constriction of airway passages called bronchioles with diseases such as asthma.
7. **BVM:** Bag valve mask. Used to ventilate a patient who is not breathing.
8. **Capnography:** The continuous monitoring of exhaled CO2 in a patient’s breath.
9. **Cardiac output (CO):** The amount of blood ejected from the left ventricle in one minute.
10. **Carina:** The internal ridge where the trachea bifurcates into the right and left bronchi.
11. **Catecholamine:** Chemical compounds associated with the sympathetic nervous system such as epinephrine, dopamine, and norepinephrine.
12. **Cerebellum:** An area of the brain involved in motor function; aiding in precision, coordination, and motor timing.
13. **Cerebrum:** The area of “higher thinking” such as learning, memory, emotions, self-control, hearing, and sight within the brain.
14. **CNS:** Central Nervous system composed of the brain and spinal cord.
15. **Coronary arteries:** These are the first two arteries in the arterial system that branch off of the aorta and feed oxygen rich blood to the heart itself.
16. **Crackles:** The clicking, rattling, or crackling noises that may be made by a patient with a respiratory disease during inhalation.
17. **Cranial nerves:** A set of 12 nerves that originate from the base of the brain.
18. **Cricothyroid membrane:** Membrane located between the thyroid and cricoid cartilages of the neck. This is the location for the emergency procedure called a cricothyrotomy.
19. **Cyanosis**: a bluish discoloration of mucus membranes and the skin due to the tissues near the skin surface having a low oxygen saturation.

**D-F**

1. **Dead space**: Air that enters with respiration but does not actually make it to the alveoli to participate in lung exchange.
2. **Dendrites**: Long tentacle like branches of a neuron that carry impulse in towards the body.
3. **Depolarize**: The changing of a voltage of a cell, often to the point where the cell fires electrically stimulating cells adjacent to it.
4. **Desaturation**: In pulse oximetry, the condition of a low blood oxygen concentration.
5. **Diaphoresis**: Sweating. Moisture formation on the skin.
6. **Diastole**: The relaxation phase of the cardiac cycle, a period where the fibers are relaxing and the heart chambers increasing in their volume.
7. **Diastolic filling time**: The period of time between two systolic contractions for blood to move into a heart chamber either sucked in through negative pressure or pushed in like with atrial kick.
8. **Diuretics**: Medications which lead to increased urination allowing for the removal of excess fluids from the body.
9. **Dissociative anaesthetic**: Produces dissociation between the brain and all sensory inputs. The patient appears catatonic with no awareness and no perception of their environment around them. This includes any perception of pain.
10. **Distal**: Further out or past some reference point.
11. **Duration of action**: The length of time that a particular drug is effective.
12. **Dyspnea**: Difficulty breathing.
13. **Efferent**: Carrying information from the CNS out to the body.
14. **Emergence**: Refers to the return to baseline physiologic function for the organs and tissues of the body as anaesthetics are discontinued.
15. **End-diastolic volume**: The blood volume in a chamber at the end of diastole just before systolic contraction.
16. **Epiglottis**: A connective tissue covering that closes over the tracheal opening during swallowing to prevent the aspiration of food or liquids.
17. **Erythrocytes**: Red blood cells.

**G-I**

1. **General Anaesthesia (GA)**: A medically induced coma with a loss of protective reflexes such as the ability to breath, maintain proper airway positioning on own, prevent aspiration with own gag reflex.
2. **Glottal opening**: The opening to the trachea.
3. **Graves' disease**: Hyperthyroidism of the thyroid gland.
4. **Half-life**: The time for one half of the drug to be cleared or made inactive.
5. **Hematocrit**: The percentage of red blood cells in the blood.
6. **Hematoma**: A collection of blood outside of a blood vessel in liquid or clotted form.
7. **Hemoglobin**: A molecule in RBCs that is crucial to carrying oxygen. Each hemoglobin molecule can carry four oxygen molecules. This protein is responsible for the transport of 98% of the oxygen in the bloodstream.

8. **Hypercapnia**: High levels of CO₂ within the bloodstream.

9. **Hypoventilation**: Inadequate rate and or volume of respirations.

10. **Hypoxemia**: Low amounts of oxygen within the bloodstream.

11. **Idiopathic**: No known cause.

12. **Induction**: This is the phase where agents are given and a loss of consciousness occurs in GA.

13. **Innate immune system**: A nonspecific immune system response that does not confer long lasting immunity for the host.

14. **Innervate**: To supply an organ or body part with nerves.

15. **Intubate**: To place a breathing tube called an endotracheal tube into the trachea.

16. **Ion channel**: Pore forming membrane proteins that control the movement of chemical ions through cell membranes. Present in the membranes of all cells.

17. **Ischemia**: An inadequate blood flow to meet the oxygen demands of an area of tissue.

18. **Islets of Langerhans**: Cells in the pancreas responsible for the secretion of insulin and glucagon.

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L-P

1. **Laryngospasm**: A medical emergency in which the vocal cords contract, often initiated by foreign materials in the laryngopharynx such as blood and mucus, and a severe airway obstruction occurs.

2. **Limbic system**: A group of structures in the brain responsible for emotions including anxiety.

3. **Lumen**: Inside space of a tubular structure, such as an artery.

4. **Mean Arterial Pressure (MAP)**: Defined as the average arterial pressure during a cardiac cycle and is thought to be the perfusion pressure seen by organs in the body.

5. **Mitrval valve**: A bicuspid one way valve located between the left atria and ventricle in the human heart.

6. **Motor nerves**: Nerves that carry impulses away from the CNS.

7. **Myelin sheath**: An electrically insulating material that surrounds sections of axons. This sheath speeds up electrical conduction down axon pathways.

8. **Myocardial infarction (MI)**: Commonly known as a heart attack. Occurs when a coronary vessel is occluded across the full vessel lumen. This is caused when a plaque ruptures or erodes so that a thrombus forms and no blood flow occurs distal to the lesion. The muscle that the vessel feeds is then cut off to the point where myocardial tissue dies.

9. **Myocardium**: Muscle cells of the heart.

10. **Necrosis**: The death of cells.

11. **Neurotransmitter**: Chemicals that transmit signals across a synaptic cleft.

12. **Normal saline (NS)**: A solution of sterile water and 0.9% NaCl.

13. **NPO**: Nothing by mouth.
14. **Obtundation**: Refers to less than full alertness (altered level of consciousness), typically as a result of a medical condition or trauma.

15. **Onset of Action**: The time from drug administration until the drug exerts an observable specific effect or response.

16. **Oxygen saturation**: The percentage of actual oxygen carried compared with the oxygen capacity. Normal arterial oxygen saturation is $> 95\%$.

17. **P wave**: A ECG wave that represents depolarization of the atria.

18. **Parasympathetic Nervous System**: The system that slows down the body; it induces the body to be “at rest”.


21. **Pericardial fluid**: The small amount of lubricating fluid found in the pericardial sac.

22. **Pericardium**: The double walled sac that surrounds the heart.

23. **Peripheral Nervous System (PNS)**: Composed of sensory nerves that carry information from our environment to the CNS (afferent nerves) and motor nerves which carry the response from the CNS back out to the body (efferent nerves) to create such things as body movement.

24. **pH**: A logarithmic measurement of hydrogen ion concentration. The lower the pH the more acidity there is.

25. **Pharmacodynamics**: refers to the responsiveness of receptors to a medication and the mechanism through which these effects occur.

26. **Pharmacokinetics**: Refers to what effects the body has on the drug. It describes the absorption, distribution, metabolism and elimination of a drug.

27. **Potentiate**: To make more effective or more active; to augment the activity of a drug synergistically.

28. **Pulse oximetry**: The measurement of the hemoglobin oxygen saturation.

29. **Proarrythmic**: To cause abnormal heart ECG rhythms.

30. **Pruritus**: Localized or generalized itching.

31. **Purkinje fibers**: The millions of small fibers at the end of the bundle branches that transmit the wave of depolarization to the ventricles.

32. **Preload**: Refers to the amount of stretch, pressure and volume in the ventricle just prior to systole.

33. **Proximal**: Happening closer in or before a specific reference point.

34. **Pulmonary valve**: The valve located where blood leaves the right ventricle and enters the pulmonary arteries.

**Q-Z**

1. **QRS complex**: The ECG wave that represents depolarization of the ventricles.
2. **Ringers Lactate**: An IV solution of water and a balanced number of electrolytes.
3. **Sensory nerves**: Nerves that carry information (impulses) towards the central nervous system.
4. **Shock**: Defined as the inadequate perfusion of tissues and organs to allow for continued aerobic metabolism and cellular life. Signs and symptoms of shock include shortness of breath, chest pain, low blood pressure (hypotension), and altered level of consciousness. They are the signs and symptoms produced when any given tissue or organ is not getting enough oxygen.
5. **Sinoatrial node (SA):** The dominant pacemaker of the heart. It is a specialized clump of cells that is located in the right atrium near where the superior vena cava enters the atrium.

6. **Sinus rhythm:** Heart rhythms that start with impulses coming from the SA-node.

7. **Soma:** A neuron cell body.

8. **Somatic nervous system (SNS):** Part of the PNS associated with the voluntary control of movement via skeletal muscles.

9. **Status epilepticus:** A seizure that lasts more than 5 minutes.

10. **Stridor:** High-pitched musical breath sound resulting from turbulent air flow in the larynx or lower in the bronchial airways.

11. **Stroke volume (SV):** The amount of blood ejected with one systolic contraction (beat) of the left ventricle. A normal SV is around 50-80 ml.

12. **Sympathetic Nervous System:** Part of the ANS that leads to what is called the “flight or fight” response when stimulated.

13. **Sympathomimetic:** Something that mimics and stimulates the SNS

14. **Synapse:** When traveling from the axon terminal to the dendrite of another neuron the impulse crosses a small gap called a synapse. Nerve scans also form synapses with other types of cells such as muscle cells.

15. **Synaptic cleft:** The gap between the nerve and the cell it is innervating at the synapse.

16. **Synergistic:** The effect of two drugs given together is greater than the sum effect of the two drugs. This is often seen when opioids and benzodiazepines when they are used in combination.

17. **Systole:** The contractile phase of the heart, a period of myocardial fiber shortening and the ejection of blood from the heart chambers.

18. **T wave:** Represents repolarization of the ventricles on an ECG.

19. **Tachypnea:** Rapid breathing.

20. **Terminal button:** A bulge that forms as a neuron terminates at a synapse.

21. **Tetany:** The involuntary contraction of muscle fibers.

22. **Therapeutic window:** The dosage range of a medication that gives the desired effect.

23. **Thoracic cage:** The sternum, the ribs, the intercostal muscles, and the diaphragm. Responsible for the mechanics of breathing.

24. **TKVO:** To keep vein open. A very slow infusion rate of an IV solution so the catheter does not clot off.

25. **Tidal volume:** The total volume of air exchanged at rest when no extra effort is applied in a normal breath.

26. **Titration:** The incremental increase in drug dosage to a level that provides the optimal therapeutic effect.

27. **Trendelenburg position:** The body is laid flat on the back (supine position) with the feet higher than the head by 15-30 degrees.

28. **Tricuspid valve:** The three cusp one way valve that is located between the right atria and ventricle in the human heart.

29. **Vagal maneuvers:** Procedures that a patient can do or that can be done to a patient to stimulate their vagus nerve. Used to terminate a type of abnormal ECG called an SVT.

30. **Vagus nerve:** A main parasympathetic nerve is the 10th cranial nerve, the vagus nerve. The vagus nerve leaves the brain stem and innervates many organs before
terminating in the rectum. Vagal stimulation leads to a decreasing heart rate “bradycardia”.

31. **Volume distribution**: the distribution of a medication between plasma and the rest of the body when a dose is administered. It is influenced by the characteristics of the drug such as lipid solubility, binding to plasma proteins, ionization rate, and molecular size.