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(CONDENSED FOR DENTAL BOARD)

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THE HISTORY OF ANESTHESIA

Anesthetic practices date from ancient times, yet the evolution of the specialty only began in the mid-nineteenth century and only became firmly established in the last 60 years. Ancient civilizations had used opium poppy, coca leaves, mandrake root, alcohol, and even phlebotomy (to the point of unconsciousness) to allow surgeons to operate. It is interesting that the ancient Egyptians used the combination of opium poppy (morphine) and hyoscyamus (hyoscyamine and scopolamine); a similar combination, morphine and scopolamine, is still used parenterally for premedication. Regional anesthesia in ancient times consisted of compression of nerve trunks (nerve ischemia) or the application of cold (cryoanalgesia). The Incas may have practiced local anesthesia as their surgeons chewed coca leaves and spat saliva into the operative wound. Surgical procedures were, for the most part, limited to caring for fractures, traumatic wounds, amputations, and the removal of bladder calculi. Amazingly, some civilizations were also able to perform trephination of the skull. The mark of a successful surgeon was his ability to work quickly.

The evolution of modern surgery was hampered not only by a poor understanding of disease processes, anatomy, and surgical asepsis but also by the lack of reliable and safe anesthetic techniques. These techniques evolved first with inhalation anesthesia, followed by local and regional anesthesia, and finally intravenous anesthesia.

Inhalational Anesthesia History
The first general anesthetics were inhalational agents: ether, nitrous oxide, and chloroform. Ether or diethyl ether (C-C-O-C-C) was originally prepared by Valerius Cordus in 1540 but was not used as an anesthetic agent in humans until 1842, when Crawford W. Long and William E. Clark used it independently on patients. A short four years later, Boston’s William T.G. Morton conducted the first public demonstration of general anesthesia using ether. Joseph Priestley produced nitrous oxide in 1772, but Humphrey Davy first noted its analgesic properties in 1800. Gardner Colton and Horace Wells are credited for having first used nitrous oxide as an anesthetic in humans in 1844 for a tooth extraction. Colton was responsible for promoting the use of nitrous oxide in the latter half of the 19th century. Chloroform was prepared in 1831. Although Holmes Coote first used it clinically as a general anesthetic in 1847, it wasn’t used in clinical practice until obstetrician James Simpson administered it to his patients to relieve labor pain. His most famous patient being Queen Victoria for which he was summoned to relieve the pain of childbirth. After the successful administration of the Chloroform on the 7th of April, 1853 for her labor Queen Victoria declared Anesthesia to be good and moral. This was very important to the future of Anesthesia because some at the time felt it immoral to remove all pain. It was widely believed the way to heaven is thru the pain we must endure in life.

Local & Regional Anesthesia History
On September 15th, 1884, Dr. Carl Koller, an ophthalmologist was credited with the origin of modern local anesthesia by demonstrating the use of topical cocaine for surgical anesthesia of the eye. Cocaine had been isolated from the coca plant leaves. Surgeon William Halsted demonstrated in 1884 that cocaine could be used for intradermal infiltration and nerve blocks. He also included the facial nerves, the brachial plexus, and the pudendal and posterior tibial nerves. August Bier administered the first spinal anesthetic in 1898 using 3 mL of 0.5% cocaine intrathecally. He was also the first to describe intravenous regional anesthesia in 1908. This procedure was later named the Bier Block after him. Procaine (Novocain) synthesis performed in 1904 by Alfred Einhorn, and within a year found clinical use as a local anesthetic by Heinrich Braun. Braun was also the first to
discover that by adding epinephrine he could prolong the duration of local anesthetics. Ferdinand Cathelin and Jean Sicard introduced caudal epidural anesthesia in 1901. In 1921 Fidel Pages first described Lumbar epidural anesthesia. Achille Dogliotti again discussed this in 1931. In time, additional local anesthetics were introduced clinically. This included dibucaine (1930), tetracaine (1932), lidocaine (1947), chloroprocaine (1955), mepivacine (1957), prilocaine (1960), bupivacaine (1963), and eitdocaine (1972). Ropivacaine, with the same duration of action as bupivacaine but perhaps less toxicity, is also available for clinical use.

Intravenous Anesthesia History
1855 - Invention of the hypodermic syringe and needle by Alexander Wood
1855 - IV anesthesia discovered. Early attempts included the use of chloral hydrate, chloroform and ether, and the combination of morphine and scopolamine
1903 - Fisher and von Mering synthesize barbiturates and diethylbarbituric acid (barbital) was the first barbiturate used for induction of anesthesia
1927 - Introduction of hexobarbital barbiturate induction becomes a popular technique
1932 - Volwiler and Tabern synthesized Thiopental
1934 - Thiopental first used clinically by John Lundy and Ralph Waters Thiopental remains the most common induction agent for anesthesia until Propofol
1957 - V.K. Stoelting made first clinical use of Methohexital
Methohexital is the only other barbiturate currently used at this time for induction
1957 - Synthesis of Chlordiazepoxide (Librium) allows the benzodiazepines to be used extensively for:
1969 - Lowenstein rekindled interest in opioid anesthesia by reintroducing the concept of high doses of narcotics as complete anesthetics. Morphine was initially employed, but fentanyl, Sufentanil, and Alfentanil were all subsequently used as sole agents. As experience grew with this

Opioid History
1805 - Morphine was isolated from opium by Sertürner and subsequently tried as an intravenous anesthetic. The morbidity and mortality initially associated with high doses of opioids in early reports caused many anesthetists to avoid opioids and favor pure inhalational anesthesia.
1939 - Interest in opioids in anesthesia returned following the synthesis of meperidine. The concept of balanced anesthesia was introduced by Lundy and others and evolved to consist of thiopental for induction, nitrous oxide for amnesia, meperidine (or any narcotic) for analgesia, and curare for muscle relaxation.

Premedication, Induction, Supplementation of anesthesia, Intravenous sedation. Benzodiazepines include:
- Diazepam (1959)
- Lorazepam (1971)
- Midazolam (1976)
1962 - Stevens synthesizes Ketamine
1964 - Etomidate synthesized
1965 - Corssen and Domino first use Ketamine clinically
1970 - Ketamine released to the market First intravenous agent associated with minimal cardiac and respiratory depression
1972 - Etomidate released to market
Enthusiasm over its relative lack of circulatory and respiratory effects has been tempered by reports of adrenal suppression after even a single dose
1989 – 2, 6 Di-isopropyl Phenol (Propofol) released to market. A major advance in outpatient anesthesia because of its short duration of action.
# Continuum of Depth of Sedation

<table>
<thead>
<tr>
<th></th>
<th>Minimal Sedation/Anxiolysis</th>
<th>Moderate Sedation/Analgesia “Conscious Sedation”</th>
<th>Deep Sedation/Analgesia</th>
<th>General Anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Responsiveness</strong></td>
<td>Normal response to verbal stimulation</td>
<td>Purposeful** response to verbal or tactile stimulation</td>
<td>Purposeful** response following repeated or painful stimulation</td>
<td>Un-arousable; even with painful stimulus</td>
</tr>
<tr>
<td><strong>Airway</strong></td>
<td>Unaffected</td>
<td>No intervention required</td>
<td>Intervention may be required</td>
<td>Intervention often required</td>
</tr>
<tr>
<td><strong>Spontaneous</strong></td>
<td>Unaffected</td>
<td>Adequate</td>
<td>May be inadequate</td>
<td>Frequently inadequate</td>
</tr>
<tr>
<td><strong>Ventilation</strong></td>
<td>Unaffected</td>
<td>Usually maintained</td>
<td>Usually maintained</td>
<td>May be impaired</td>
</tr>
<tr>
<td><strong>Cardiovascular</strong></td>
<td>Unaffected</td>
<td>Adequate</td>
<td>Usually maintained</td>
<td>Usually maintained</td>
</tr>
<tr>
<td><strong>Function</strong></td>
<td></td>
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<td></td>
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</tbody>
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**Committee of Origin:** Quality Management and Departmental Administration ASA House of Delegates

**Minimal Sedation (Anxiolysis):** Drug-induced state during which patients respond normally to verbal commands. Although cognitive function and physical coordination may be impaired, airway reflexes, and respiratory and cardiovascular functions are unaffected.

**Moderate Sedation/Analgesia (“Conscious Sedation”):** 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.

**Deep Sedation/Analgesia:** 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.

**General Anesthesia:** Drug-induced coma with loss of consciousness during which patients are not arousable and will not move to painful stimulation. The ability to independently maintain adequate 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 under deeper levels.

Because sedation is a continuum, it is not always possible to predict how an individual patient will respond. Hence, practitioners intending to produce a given level of sedation should be able to rescue patients whose level of sedation becomes deeper than initially intended. Individuals administering Moderate Sedation/Analgesia (“Conscious Sedation”) should be able to rescue patients who enter a state of Deep Sedation/Analgesia, while those administering Deep Sedation/Analgesia should be able to rescue patients who enter a state of General Anesthesia.

*** Rescue of a patient from a deeper level of sedation than intended is an intervention by a practitioner proficient in airway management and advanced life support. The qualified practitioner corrects adverse physiologic consequences of the deeper-than-intended level of sedation (such as hypoventilation, hypoxia and hypotension) and returns the patient to the originally intended level of sedation. It is not appropriate to continue the procedure at an unintended level of sedation.

** Reflex withdrawal from a painful stimulus is NOT considered a purposeful response.

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SEDATIVES AND ANALGESICS

Benzodiazepines

Benzodiazepines are used as sedatives, hypnotics, anxiolytics, anticonvulsants and muscle relaxants. Benzodiazepines are similar in pharmacological action but have different potencies. Some benzodiazepines work better in the treatment of particular conditions. The useful clinical effects of benzodiazepines are sedation, hypnosis, anti-anxiety, amnesia, muscle relaxation, and a potent anti-seizure effect. Their detrimental clinical effects are respiratory depression, rapid physical dependence, and an absence of analgesia.

**Clinical Effect:**

Cerebral:
- Antianxiety
- Amnesia – ante grade +/- retrograde
- Sedation

- Centrally medicated muscle relaxation
- Anti-Seizure effect
- No Analgesia!

Respiratory:
- Depress ventilation through blunting the brainstem CO2 respiratory drive center.
  Shallow breathing with an increased respirator rate is observed clinically.

**Mechanisms of Action:** Benzodiazepines are a class of agents that work on the central nervous system by modulating gamma-aminobutyric acid-A (GABA-A) receptors in the brain and especially the limbic system. It enhances the response to the inhibitory neurotransmitter GABA, by opening GABA-activated chloride channels and allowing chloride ions to enter the neuron, making the neuron negatively charged and resistant to excitation. This is important because excitation in the limbic system amplifies the anxious emotion making it very difficult for the patient to relax. The GABA(A) receptors are membrane protein complexes that are composed of α, β, and γ subunits. The membrane receptors are mainly expressed in the brain but can also be found in different tissue such as muscle and immune cells. γ-aminobutyric acid (GABA) is the endogenous ligand for the GABA(A) receptors inhibiting the signaling transduction mediated by these receptors by hyper-polarization (increase of chloride ion efflux). Other molecules are known to modulate GABA (A) receptor actions such as alcohol, steroids and barbiturates.

**Receptor Discussion:** The GABA receptor is the major molecular target for the action of many drugs in the brain. Some GABA receptor agonist include benzodiazepines, intravenous anesthetics (barbiturates and propofol) volatile anesthetics, and ethanol. GABA receptors have been identified electro physiologically and pharmacologically in all regions of the brain. The complex includes five major binding domains with binding sites localized in or near the Cl– channel. Benzodiazepines, barbiturates as well as anesthetic steroids (propofol) bind to sites on the GABA receptor. The domains modulate the receptor response to GABA stimulation. In addition, other drugs, including volatile anesthetics and ethanol have been reported to have an effect on this receptor. An integral part of this complex is the Cl– channel. The GABA specific binding site is directly responsible for opening the Cl– channel.
Benzodiazepine agonists represent the largest group of agents in this general GABA receptor class of depressant drugs. They show anticonvulsant, anxiolytic and sedative—hypnotic activity. Well known examples include diazepam and Xanax, which often are prescribed for their anti-anxiety effects. Halcion is known for its sedative and anxiolytic properties. The mechanism of action of benzodiazepine agonists is to enhance GABAergic transmission. The advantage of benzodiazepines is that they do not open the Cl- channels directly. Instead they act more subtly by potentiating the effect of GABA.

Barbiturates comprise another class of drugs commonly used therapeutically for anesthesia and control of epilepsy. Barbiturates bind to another site on the GABA receptor. This opens the Cl- channels directly and causes similar clinical effects. Phenobarbital and pentobarbital are two of the most commonly used barbiturates. Phenobarbital was used to treat patients with epilepsy since 1912. Pentobarbital is also an anticonvulsant and has useful sedative properties.

Different varieties of agonists bind to this site and elicit GABA-like responses. One of the most useful research agonists is the compound muscimol, a naturally occurring GABA receptor agonist isolated from the psychoactive mushroom Amanita muscaria. It is a potent and specific agonist at GABA receptors and has been a valuable tool for pharmacological and radio ligand binding studies.

**Biotransformation:** The benzodiazepines rely upon the liver for biotransformation into water-soluble glucuronide end products. *The phase I metabolites of diazepam are pharmacologically active.* Slow hepatic extraction and a large volume of distribution result in a long elimination half-life for diazepam (30 hours). Although lorazepam also has a low hepatic extraction ratio, its lower lipid solubility limits its volume of distribution, resulting in a shorter elimination half-life (15 hours). Nonetheless, the clinical duration of lorazepam is often quite prolonged owing to a very high receptor affinity. In contrast, midazolam shares diazepam’s volume of distribution, but its elimination half-life (2 hours) is the shortest of the group because of its high hepatic extraction ratio.

**Excretion:** The metabolites of benzodiazepine biotransformation are excreted chiefly in the urine. Enterohepatic circulation produces a secondary peak in diazepam plasma concentration 6 – 12 hours following administration.

**Effects on Organ Systems:**
**Cardiovascular:** The benzodiazepines display minimal cardiovascular depressant effects even at induction doses. Arterial blood pressure, cardiac output, and peripheral vascular resistance usually decline slightly, while heart rate sometimes rises. Midazolam tends to reduce blood pressure and peripheral vascular resistance more than does diazepam.

**Respiratory effects:**
- Depresses the ventilatory response to CO2 increases (hypercarbia)
- Depresses the ventilatory response to low O2 sat (hypoxia)
- Intravenous doses may cause apnea
- Potent SYNERGISTIC APNEA effect with all opiates

Discussion: Benzodiazepines depress the ventilator response to CO2. This depression is usually insignificant unless the drugs are administered intravenously or in association with other respiratory depressants. Although apnea may be less common than following...
barbiturate induction, even small intravenous doses of diazepam and midazolam have resulted in respiratory arrest. The steep dose-response curve, slightly prolonged onset (compared to thiopental or diazepam), and high potency of midazolam necessitate careful titration to avoid over dosage and apnea. Ventilation must be monitored in all patients receiving intravenous benzodiazepines, and resuscitating equipment must be immediately available.

_Cerebral:_ Benzodiazepines reduce cerebral oxygen consumption, cerebral blood flow, and intracranial pressure but not to the extent the barbiturates do. They are very effective in preventing and controlling grand mal seizures. Oral sedative doses often produce antegrade amnesia, a useful premedication property. The mild muscle-relaxant properties of these drugs are mediated at the spinal cord level, not at the neuromuscular junction. The antianxiety, amnesic, and sedative effects of benzodiazepine drugs seen at low doses progress to stupor and unconsciousness at induction doses. Compared to thiopental, induction with benzodiazepines is associated with a slower loss of consciousness and a longer recovery. Remember, benzodiazepines have no direct analgesic properties and pain may elicit an aggressive response.

**Adverse Reactions:** In general, benzodiazepines are safe and effective in the short term. All practitioners must be aware that cognitive impairments and paradoxical effects such as aggression or behavioral disinhibition occasionally occur. A minority of patients react reverse and contrary to what would normally be expected. For example, a state of panic may worsen considerably following intake of a benzodiazepine. Long-term use is controversial due to concerns about adverse psychological and physical effects, increased questioning of effectiveness, and, because benzodiazepines are prone to cause tolerance, physical dependence, and, upon cessation of use after long-term use, a withdrawal syndrome which may include seizures. Due to adverse effects associated with the long-term use of benzodiazepines, withdrawal and abstinence from benzodiazepines, in general, leads to improved physical and mental health. The elderly are at an increased risk of suffering from both short and long term adverse effects, including a 50% increase in the risk of dementia.

**Drug Interactions:**
- Cimetidine binds to cytochrome P-450 and reduces the metabolism of diazepam.
- Erythromycin inhibits midazolam metabolism.
- Benzodiazepines reduce the minimum alveolar concentration of volatile anesthetics as much as 30%.
- Ethanol potentiates the sedative effects of the benzodiazepines.
- Opiates: Synergistic respiratory depressant effect
## COMPARISON OF BENZODIAZEPINES

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<tbody>
<tr>
<td><strong>Long Acting</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chlordiazepoxide (Librium®)</td>
<td>Int. (po)</td>
<td>2-4(po)</td>
<td>5-30</td>
<td>3-100</td>
<td>10 mg</td>
</tr>
<tr>
<td>Diazepam¹</td>
<td>Rapid (po, IV)</td>
<td>1(po)</td>
<td>20-50</td>
<td>3-100</td>
<td>5 mg</td>
</tr>
<tr>
<td>Flurazepam</td>
<td>Rapid</td>
<td>0.5-2</td>
<td>inactive</td>
<td>47-100</td>
<td>30 mg</td>
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<tr>
<td><strong>Intermediate Acting</strong></td>
<td></td>
<td></td>
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<tr>
<td>Alprazolam¹</td>
<td>Int.</td>
<td>0.7-1.6</td>
<td>6-20</td>
<td>~4-10</td>
<td>0.5mg</td>
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<tr>
<td>Clonazepam¹</td>
<td>Int.</td>
<td>1-4</td>
<td>18-39</td>
<td>-</td>
<td>0.25mg</td>
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<tr>
<td>Lorazepam¹</td>
<td>Int. (po), Rapid (sl, IV)</td>
<td>1-1.5 (po)</td>
<td>10-20</td>
<td>~10-20</td>
<td>1mg</td>
</tr>
<tr>
<td>Oxazepam¹</td>
<td>Slow</td>
<td>2-3</td>
<td>3-21</td>
<td>-</td>
<td>15mg</td>
</tr>
<tr>
<td>Temazepam¹</td>
<td>Slow</td>
<td>0.75-1.5</td>
<td>10-20</td>
<td>-</td>
<td>30mg</td>
</tr>
<tr>
<td><strong>Short Acting</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Midazolam¹</td>
<td>Most Rapid IV</td>
<td>0.5-1 (IV)</td>
<td>1-4</td>
<td>~2-10</td>
<td>-</td>
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<tr>
<td>Triazolam</td>
<td>Int.</td>
<td>0.75-2</td>
<td>1.6-5.5</td>
<td>~2-10</td>
<td>0.5mg</td>
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### DOSING

<table>
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<tr>
<th>AGENT</th>
<th>USE</th>
<th>ROUTE / DOSE</th>
</tr>
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<tbody>
<tr>
<td>Diazepam (Valium)</td>
<td>Premedication Sedation Induction</td>
<td>Oral: .2 mg/kg Oral: .4 mg/kg IV: .05 mg/kg IV: .3 mg/kg</td>
</tr>
<tr>
<td>Midazolam (Versed)</td>
<td>Premedication Sedation Induction</td>
<td>Oral: .2 mg/kg Oral: .4 mg/kg IV: .03 mg/kg IV: .1 mg/kg</td>
</tr>
<tr>
<td>Triazolam (Halcion)</td>
<td>Sedation</td>
<td>Oral: Adult: .25 - .5 mg Oral: Geriatric: .125 mg</td>
</tr>
</tbody>
</table>

**REVERSAL Flumazenil:** Flumazenil is a benzodiazepine antagonist. It works by blocking receptors in the brain and central nervous system that benzodiazepines need to reach to be active, which helps reduce drowsiness and sedation.

<table>
<thead>
<tr>
<th>REVERSAL AGENT</th>
<th>INCREMENTAL DOSES</th>
<th>MAXIMUM DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flumazenil</td>
<td>.2 mg every minute until reaching the desired degree of reversal</td>
<td>1.0 mg</td>
</tr>
</tbody>
</table>
ORAL VERSED METABOLIC PATHWAY

BENZODIAZEPINE METABOLIC PATHWAY
Opioids

Opioids are among the world’s oldest known drugs; the therapeutic use of the opium poppy predates recorded history. The analgesic (painkiller) effects of opioids are due to decreased perception of pain, decreased reaction to pain as well as increased pain tolerance.

Clinical Effects:

Cerebral:
- Opiates produce a potent analgesic effect. Intravenous opioids have been the mainstay of pain control for more than a century.
- Opiates are very predictable when used as a primary or secondary sedative. Mild to moderate dose dependent sedation occurs with all opiates. Unlike barbiturates or benzodiazepines, relatively large doses of opioids are required to render patients unconscious.
- Regardless of the dose, opioids do not reliably produce amnesia.

Respiratory:
- Opioids depress ventilation, particularly respiratory rate. Resting PaCO2 increases and the response to a CO2 challenge is blunted, resulting in a shift of the CO2 response curve downward and to the right. These effects are mediated through the respiratory centers in the brainstem. The apneic threshold, the highest PaCO2 at which a patient remains apneic, is elevated, and hypoxic drive is decreased.
- Morphine and meperidine can cause histamine-induced bronchospasm in susceptible patients.
- Opioids (particularly fentanyl, Sufentanil, and Alfentanil) can induce chest wall rigidity severe enough to prevent adequate ventilation. This centrally mediated muscle contraction is most frequent after large drug boluses and is effectively treated with muscle relaxants.
- Opioids can effectively blunt the broncho-constrictive response to airway stimulation such as that occurring during intubation.

The human body naturally produces its own opiate-like substances and uses them as neurotransmitters. These substances include endorphins, enkephalins, and dynorphin, often collectively known as endogenous opioids. Endogenous opioids modulate our reactions to painful stimuli. They also regulate vital functions such as hunger and thirst and are involved in mood control, immune response, and other processes. The reason that medicine based opiates affect us so powerfully is that these exogenous substances bind to the same receptors as our endogenous opioids.

Medicine based opioids bind to specific receptors located throughout the central nervous system and other tissues. Four major types of opioid receptor have been identified: mu, kappa, delta, and sigma. While opioids provide some degree of sedation, they are most effective at producing analgesia. The pharmacodynamic properties of specific opioids depend upon which receptor is bound, the binding affinity, and whether the receptor is activated.
Opioid Receptors

<table>
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<th>RECEPTOR</th>
<th>CLINICAL EFFECT</th>
<th>OPIATE</th>
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<tbody>
<tr>
<td>Mu</td>
<td>Respiratory Depression</td>
<td>Morphine Hydrocodone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fentanyl Oxycodone</td>
</tr>
<tr>
<td>Kappa</td>
<td>Sedation</td>
<td>Morphine Hydrocodone</td>
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<tr>
<td></td>
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<td>Fentanyl Oxycodone</td>
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<tr>
<td>Delta</td>
<td>Analgesia</td>
<td>Morphine Hydrocodone</td>
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<td></td>
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<td>Fentanyl Oxycodone</td>
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<td>Sigma</td>
<td>Dysphoria</td>
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<td>Hydromorphone</td>
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</tbody>
</table>

**Mechanisms of Action:** Opioids bind to specific receptors located throughout the central nervous system and other tissues. Four major types of opioid receptor have been identified: mu, kappa, delta, and sigma. While opioids provide some degree of sedation, they are most effective at producing analgesia. The pharmacodynamic properties of specific opioids depend upon which receptor is bound, the binding affinity, and whether the receptor is activated. See Table below. Although both opioid agonists and antagonists bind to opioid receptors, only agonists are capable of receptor activation. Agonist-antagonists (eg. nalbuphine, nalorphine, butorphanol, and pentazocine) are drugs that have opposite actions at different receptor types.

Opioid-receptor activation inhibits the presynaptic release and postsynaptic response to excitatory neurotransmitters (eg, acetylcholine, substance P) from nociceptive neurons. The cellular mechanism for this neuromodulation may involve alterations in potassium and calcium ion conductance. Transmission of pain impulses can be interrupted at the level of the dorsal horn of the spinal cord with intrathecal or epidural administration of opioids. Modulation of a descending inhibitory pathway from the periaqueductal gray through the nucleus raphe magnus to the dorsal horn of the spinal cord may also play a role in opioid analgesia. Although opioids exert their greatest effect within the central nervous system, opioid receptors have also been isolated from somatic and sympathetic peripheral nerves.

**Effects on Organ Systems:**

**Cardiovascular:** In general, opioids do not seriously impair cardiovascular function. Meperidine tends to increase heart rate (it is structurally similar to atropine), while high doses of morphine, fentanyl, Sufentanil, and Alfentanil are associates with a vagusmediated bradycardia. With the exception of Meperidine, the opioids do not depress cardiac contractility. Meperidine and morphine evoke histamine release in some individuals that can lead to profound drops in arterial blood pressure and systemic vascular resistance. The effects of histamine release can be minimized in susceptible patients by slow opioid infusion, adequate intravascular volume, or pretreatment with H1 and H2 histamine antagonists.

**Respiratory:** Opioids depress ventilation, particularly respiratory rate. Resting PaCO2 increases and the response to a CO2 challenge is blunted, resulting in a shift of the CO2 response curve downward and to the right. These effects are mediated through the respiratory centers in the brainstem. The apneic threshold – the highest PaCO2 at
which a patient remains apneic – is elevated, and hypoxic drive is decreased. Morphine and meperidine can cause histamine-induced bronchospasm in susceptible patients. Opioids (particularly fentanyl, Sufentanil, and Alfentanil) can induce chest wall rigidity severe enough to prevent adequate ventilation. This centrally mediated muscle contraction is most frequent after large drug boluses and is effectively treated with muscle relaxants. Opioids can effectively blunt the bronchoconstrictive response to airway stimulation such as that occurring during intubation.

*Cerebral*: Potent analgesia: Intravenous opioids have been the mainstay of pain control for more than a century. Mild to moderate dose dependent sedation occurs with all opiates. Unlike barbiturates or benzodiazepines, relatively large doses of opioids are required to render patients unconscious. Stimulation of the medullary chemoreceptor trigger zone is responsible for a high incidence of nausea and vomiting. Regardless of the dose, opioids do not reliably produce amnesia. Physical dependence is a significant problem associated with repeated opioid administration.

**Dosing Profile of Opioids:**

<table>
<thead>
<tr>
<th>OPIOID</th>
<th>ROUTE</th>
<th>DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>IM</td>
<td>.2 mg/Kg</td>
</tr>
<tr>
<td>Morphine</td>
<td>IV</td>
<td>.05 mg/Kg</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Intranasal</td>
<td>.25 mcg/Kg</td>
</tr>
<tr>
<td></td>
<td>IM</td>
<td>1 mcg/Kg</td>
</tr>
<tr>
<td></td>
<td>IV</td>
<td>.25 mcg/Kg/increments</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>Oral</td>
<td>5-10 mg PO</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>Oral</td>
<td>5-10 mg PO</td>
</tr>
<tr>
<td>Morphine</td>
<td>Oral</td>
<td>3-5 mg PO</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>Oral</td>
<td>2-4 mg PO</td>
</tr>
</tbody>
</table>

**Adverse reactions/ Side effects:**
- Respiratory depression
- Constipation
- Opioids can cause cough suppression
- Opioid dependence can develop with ongoing administration, leading to a withdrawal syndrome with abrupt discontinuation

**Drug Interactions:**
- The combination of opioids – particularly meperidine – and monoamie oxidase inhibitory may result in respiratory arrest, hypertension or hypotension, coma, and hyperpyrexia
- The cause of this dramatic interaction is not understood.
- Barbiturates, benzodiazepines, and other central nervous system depressants can have synergistic cardiovascular, respiratory, and sedative effects with opioids.
- The biotransformation of Alfentanil, but not Sufentanil, may be impaired following a 7-day course of erythromycin, leading to prolonged sedation and respiratory depression.
**Emergency Reversal:**

*Narcan* (naloxone) is indicated for the complete or partial reversal of opioid depression. Narcan (naloxone) prevents or reverses the effects of opioids including respiratory depression and sedation. Narcan is essentially a pure opioid antagonist. It blocks the action of opioids at the receptor - opioid interface through binding.

**Emergency Reversal Dosing**

<table>
<thead>
<tr>
<th>REVERSAL AGENT</th>
<th>INCREMENTAL DOSES</th>
<th>MAX DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naloxone</td>
<td>IV: .04 mg every 3 minutes until adequate ventilation and alertness are achieved</td>
<td>.4 mg</td>
</tr>
<tr>
<td></td>
<td>IM: .2 mg IM</td>
<td></td>
</tr>
</tbody>
</table>
# DOSING MATRIX

## KEEP DOSING SIMPLE - DOSING PROTOCOL MATRIX

<table>
<thead>
<tr>
<th>ADULT HALCION PROTOCOL</th>
<th>INITIAL ADULT DOSE</th>
<th>TITRATION DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADULT HALCION PROTOCOL</td>
<td>.125 mg Halcion</td>
<td>.125 mg Halcion</td>
</tr>
<tr>
<td>ADULT HALCION PROTOCOL</td>
<td>.25 mg Halcion</td>
<td>.25 mg Halcion</td>
</tr>
<tr>
<td>ADULT HALCION PROTOCOL</td>
<td>.5 mg Halcion</td>
<td>.25 mg Halcion</td>
</tr>
</tbody>
</table>

## ADULT COMBINATION PROTOCOL

<table>
<thead>
<tr>
<th>INITIAL ADULT DOSE</th>
<th>TITRATION DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 mg Hydrocodone</td>
<td>.125 mg Halcion</td>
</tr>
<tr>
<td>5 mg Hydrocodone</td>
<td>.25 mg Halcion</td>
</tr>
<tr>
<td>5 mg Hydrocodone</td>
<td>.25 mg Halcion</td>
</tr>
</tbody>
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## COMBINATION PROTOCOLS

<table>
<thead>
<tr>
<th>5 mg Hydrocodone</th>
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<td>.25 mg Halcion</td>
</tr>
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</table>

## TITRATION DECISIONS

<table>
<thead>
<tr>
<th>IS IT A PAIN ISSUE OR A SEDATION ISSUE?</th>
<th>PAIN ISSUE - CONSIDER:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IMPROVING THE BLOCK</td>
</tr>
<tr>
<td></td>
<td>USING N20</td>
</tr>
<tr>
<td></td>
<td>FULL BLADDER</td>
</tr>
<tr>
<td></td>
<td>PATIENT POSITIONING</td>
</tr>
<tr>
<td>HOW MUCH TIME IS LEFT IN THE CASE?</td>
<td></td>
</tr>
<tr>
<td>1. 15 - 30 min - Consider using bedside manner</td>
<td></td>
</tr>
<tr>
<td>2. Greater than 45 min - Consider titration</td>
<td></td>
</tr>
<tr>
<td>HOW MUCH STIMULATION IS THE DENTAL WORK GOING TO CAUSE?</td>
<td>Consider using N20</td>
</tr>
<tr>
<td>WHAT IS THE TOTAL DOSE FOR THE CASE?</td>
<td></td>
</tr>
<tr>
<td>1 mg Halcion - 2 hours</td>
<td></td>
</tr>
<tr>
<td>2 mg Halcion - 4 hours</td>
<td></td>
</tr>
</tbody>
</table>

## PEDIATRIC PROTOCOLS - NEVER TITRATE

<table>
<thead>
<tr>
<th>PEDIATRIC PROTOCOL</th>
<th>ORAL DOSE</th>
<th>MAX ORAL DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>MIDAZOLAM</td>
<td>.25 mg/kg to .75 mg/kg</td>
<td>25 mg ORAL MIDAZOLAM</td>
</tr>
<tr>
<td>MIDAZOLAM DEEROL</td>
<td>.25 mg/kg Midazolam 1 mg/kg Demerol</td>
<td>100 mg ORAL DEMEROL</td>
</tr>
</tbody>
</table>
PEDIATRIC ANATOMY & PHYSIOLOGY

Children pose significant challenges to sedate. Their anatomy and physiology define the risks involved with sedation. **This section is reviewed in all courses to make dentists aware of the extra training needed to sedate children!**

1. Children by definition have a difficult airway due to their large tongue, floppy u shaped epiglottis, and large occipital growth.
2. Children desaturate very quickly because they have little respiratory reserve.
3. Children desaturate very quickly because their metabolic consumption of oxygen is 4 X's that of an adult.
4. Children have a thick and non-compliant heart. This makes the blood pressure heart rate dependent. When their saturation drops, their heart rate drops. This poses a very dangerous physiologic problem.

Children are not small adults. Pediatric patients vary considerably and include the following groups:
- **Neonates** – a baby within 44 weeks of age from the date of conception
- **Infants** – a child of up to 12 months of age
- **Child** – 1 to 12 years
- **Adolescent** – 13 to 16 years

The differences between pediatric and adult anesthetic practice are reduced as the patients become older.

USEFUL PEDIATRIC RESPIRATORY EQUATIONS:
- \( RR = 24 - \frac{\text{age}}{2} \)
- Spontaneous ventilation \( TV = 6-8 \text{ ml/kg} \)

PEDIATRIC ANATOMY - OBLIGATE NASAL BREATHERS:
- Proportionately larger head
- Proportionately larger tongue
- Narrow nasal passages
- Anterior and cephalad larynx
- Long epiglottis
- Short trachea and neck

In children > 5 years of age, the cricoid cartilage is the narrowest point in the airway. The glottis is the narrowest point of the airway in adults. Therefore, edema has a proportionately greater effect in a child's airway. In older children, prominent adenoidal and tonsillar tissue can obstruct visualization of the larynx.
The respiratory mechanism of the pediatric patient varies from the adult in both anatomy and physiology. As children grow, the airway enlarges and moves more caudally as the c-spine elongates. The pediatric airway overall has poorly developed cartilaginous integrity allowing for more laxity throughout the airway. Another important distinction is the narrowest point in the airway in adults is at the cords versus below the cords for children. An important aspect of the narrow airway in children is that resistance is significantly increased. The formula to consider is:

- \[ R \sim \frac{8l}{r^4} \]
- \( R \) – resistance, \( l \) – length, \( r \) – radius
Small changes in the airway radius will therefore increase the resistance to the fourth power. Therefore, a small amount of post-extubation sub-glottic edema will significantly increase the work of breathing for an infant. Children also have a smaller functional residual capacity (FRC) defined as the residual volume plus the expiratory reserve volume. Physiologically, FRC occurs when the outward pull of the chest wall equals the inward collapse of the lungs.

FRC essentially acts as a respiratory reserve. When patients begin to develop respiratory distress, an increased FRC equates to a longer period of time prior to respiratory failure. The reduced FRC is important in two particular circumstances. First, it can be decreased by up to 30% in a supine patient as compared to a sitting patient. As the abdominal contents push up on the diaphragm in a supine patient, the FRC is affected. This situation is amplified in pediatric patients because of a compliant chest wall, small thoracic cage, and large abdominal contents. Second, while pre-oxygenating a patient prior to intubation the reduced FRC decreases the amount of time allowed to establish an endotracheal tube prior to desaturation. There are also many physiologic differences in respiratory mechanisms between children and adults. Children have a more complaint trachea, larynx, and bronchi due to poor cartilaginous integrity. This in turn allows for dynamic airway compression, i.e. a greater negative inspiratory force “sucks in” the floppy airway and decreases airway diameter. This in turn increases the work of breathing by increasing the negative inspiratory pressure generated.

A vicious cycle is created which may eventually lead to respiratory failure:
- Subglottic stenosis ⇒ ⦿ negative inspiratory force
- ⦿ negative inspiratory force ⇒ airway collapse
- Airway collapse ⇒ ⦿ subglottic stenosis
- ⦿ subglottic stenosis ⇒ ⦿ negative inspiratory force
- ⦿ negative inspiratory force ⇒ ⦿ work of breathing
- ⦿ work of breathing ⇒⇒ respiratory failure.

<table>
<thead>
<tr>
<th>Anatomy</th>
<th>PEDIATRIC</th>
<th>ADULT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tongue</td>
<td>Large</td>
<td>Normal</td>
</tr>
<tr>
<td>Epiglottis Shape</td>
<td>Floppy omega shaped</td>
<td>Firm flatter</td>
</tr>
<tr>
<td>Epiglottis Level</td>
<td>Level of C3 - C4</td>
<td>Level of C5 - C6</td>
</tr>
<tr>
<td>Trachea</td>
<td>Smaller shorter</td>
<td>Wider longer</td>
</tr>
<tr>
<td>Larynx Shape</td>
<td>Funnel shaped</td>
<td>Column</td>
</tr>
<tr>
<td>Larynx Position</td>
<td>Angles posteriorly away from glottis</td>
<td>Straight up and down</td>
</tr>
<tr>
<td>Narrowest Point</td>
<td>Sub-glottic region</td>
<td>At level of Vocal cords</td>
</tr>
<tr>
<td>Lung Volume</td>
<td>250ml at birth</td>
<td>6000 ml as adult</td>
</tr>
</tbody>
</table>
Pediatric patients also have more compliant chest walls also increasing the work of breathing – i.e. the outward pull of the chest is greater. Infants are dependent on functional diaphragms for adequate ventilation. The accessory muscles contribute less to the overall work of breathing in infants as compared to older children and adults. Therefore, a non-functional diaphragm often leads to respiratory failure. Diaphragmatic fatigue is one amongst several potential causes of respiratory failure and apnea in young patients with RSV bronchilitis. Considerable structural changes in the chest wall may change infant and childhood predisposition to respiratory failure, lung injury, and ventilation-associated lung injury. The orientation of the ribs is horizontal in the infant; by 10 years of age, the orientation is downward. Finally, the respiratory muscles themselves have a significant oxygen and metabolite requirement in children. In pediatric patients the work of breathing can account for up to 40% of the cardiac output, particularly in stressed conditions.

This cursory discussion of the pediatric respiratory anatomy and physiology allows one to appreciate the significant differences between children and adults. Therefore, the child with respiratory distress / failure should be approached and treated with urgency, vigilance, and caution. It is important to recognize that the pediatric metabolism is 2 - 4 X’s that of an adult. During Respiratory Distress, the increased metabolism coupled with a low lung reserve will cause desaturation to occur much quicker than in an adult.

In infants the myocardium is less contractile causing the ventricles to be less compliant and less able to generate tension during contraction. This limits the size of the stroke volume. Cardiac output is therefore rate dependent. The infant behaves as with a fixed cardiac output state. Vagal parasympathetic tone is the most dominant, which makes neonates and infants more prone to bradycardias.

- SIMPLY STATED: As the blood pressure drops with bradycardia, resuscitative efforts need to rapidly be implemented!
- PEARL: Bradycardia associated with hypoxia should be treated with oxygen and ventilation initially.

### NORMAL PEDIATRIC CV VITAL SIGNS

<table>
<thead>
<tr>
<th>Age</th>
<th>Average</th>
<th>Range</th>
<th>Mean SBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm</td>
<td>130</td>
<td>120-170</td>
<td>40-55</td>
</tr>
<tr>
<td>New-born</td>
<td>120</td>
<td>100-170</td>
<td>50-90</td>
</tr>
<tr>
<td>1-11 months</td>
<td>120</td>
<td>80-160</td>
<td>85-105</td>
</tr>
<tr>
<td>2 years</td>
<td>115</td>
<td>80-130</td>
<td>95-105</td>
</tr>
<tr>
<td>4 years</td>
<td>110</td>
<td>80-120</td>
<td>95-110</td>
</tr>
<tr>
<td>6 years</td>
<td>100</td>
<td>75-115</td>
<td>95-110</td>
</tr>
<tr>
<td>8 years</td>
<td>90</td>
<td>70-110</td>
<td>95-110</td>
</tr>
<tr>
<td>10 years</td>
<td>90</td>
<td>70-110</td>
<td>100-120</td>
</tr>
<tr>
<td>14 years</td>
<td>80</td>
<td>60-100</td>
<td>110-130</td>
</tr>
<tr>
<td>boy</td>
<td>85</td>
<td>65-105</td>
<td>110-130</td>
</tr>
<tr>
<td>girl</td>
<td>85</td>
<td>65-105</td>
<td>110-130</td>
</tr>
<tr>
<td>16 years</td>
<td>75</td>
<td>55-95</td>
<td>110-130</td>
</tr>
<tr>
<td>boy</td>
<td>80</td>
<td>60-100</td>
<td>110-130</td>
</tr>
<tr>
<td>girl</td>
<td>80</td>
<td>60-100</td>
<td>110-130</td>
</tr>
</tbody>
</table>
ANTI-NAUSEA MEDICINE

Ondansetron & Granisetron

Mechanism of Action:
Ondansetron and granisetron selectively block serotonin 5-HT₃ receptors, with little or no effect on dopamine receptors. The 5-HT₃ receptors, which are located peripherally (abdominal vagal afferents) and centrally (chemoreceptor trigger zone of the area postrami and the nucleus tractus solitaries), appear to play an important role in the initiation of the vomiting reflex. Unlike metoclopramide, these agents do not affect gastrointestinal motility or lower esophageal sphincter tone.

Clinical Uses:
Ondansetron has been proven to be an effective antiemetic in the postoperative period. In some studies, it has provided superior prophylaxis compared to metoclopramide or droperidol. Because of its expense, however, it should not be used as a routine prophylaxis. Rather, it should be considered in patients with a prior history of postoperative nausea; those who are undergoing procedures at high risk for nausea (eg, laparoscopy); those in whom nausea and vomiting must be avoided (eg, neurosurgery); and those experiencing nausea and vomiting to prevent further episodes. At this time, granisetron has been approved only for prevention of chemotherapy-induced nausea and vomiting.

Side Effects:
Ondansetron and granisetron are essentially devoid of serious side effects, even at amounts several times the recommended dose. They do not appear to cause sedation, extrapyramidal signs, or respiratory depression. Transient mild changes in blood pressure have been reported with granisetron, but not ondansetron. Intravenous administration can be associated with pain on injection.

Dosage:

a. The recommended adult intravenous dose of ondansetron for prevention of perioperative nausea and vomiting is 4 mg prior to sedation.

b. Postoperative nausea and vomiting can also be treated with a 4 mg dose, repeated as needed every 4-8 hours. Ondansetron undergoes extensive metabolism in the liver via hydroxylation and conjunction by cytochrome O-450 enzymes.

c. Liver failure impairs clearance severalfold, and the dose should be reduced accordingly. Granisetron is more potent and longer acting than ondansetron, but is also dependent on the liver for clearance.

d. A granisetron dosage for postoperative nausea and vomiting is 10 µg.kg. A single daily dose of 10 µg.kg is being recommended 30 minutes before initiation of chemotherapy.

Drug Interactions:
No significant drug interactions with ondansetron or granisetron have been reported.
NITROUS OXIDE

Physical Properties: Nitrous oxide (N₂O: Laughing Gas) is the only inorganic anesthetic gas in clinical use. It is colorless and essentially odorless. Although nonexplosive and nonflammable, nitrous oxide is as capable as oxygen of supporting combustion. Unlike the potent volatile agents, nitrous oxide is a gas at room temperature and ambient pressure. It can be kept as a liquid under pressure, however, because its critical temperature lies above room temperature. Nitrous oxide is a relatively inexpensive anesthetic.

Effects on Organ Systems:
Cardiovascular: The circulatory effects of nitrous oxide are explained by its tendency to stimulate the sympathetic nervous system. Even though nitrous oxide directly depresses myocardial contractility in vitro, arterial blood pressure, cardiac output, and heart rate are essentially unchanged or slightly elevated in vivo because of its stimulation of catecholamines. Myocardial depression may be unmasked in patients with coronary artery disease or severe hypovolemia. The resulting drop in arterial blood pressure may occasionally lead to myocardial ischemia. Constriction of pulmonary vascular smooth muscle increases pulmonary vascular resistance, which results in an elevation of right atrial pressure. Despite vasoconstriction of cutaneous vessels, peripheral vascular resistance is not significantly altered. Because nitrous oxide increases endogenous catecholamine levels, it may be associated with a higher incidence of epinephrine-induced dysrhythmias.

Respiratory: Nitrous oxide increases respiratory rate and decreases tidal volume as a result of central nervous system stimulation and, perhaps, activation of pulmonary stretch receptors. The net effect is a minimal change in minute ventilation and resting arterial CO₂ levels. Hypoxic drive, the ventilatory response to arterial hypoxia that is mediated by peripheral chemoreceptors in the carotid bodies, is markedly depressed by even small amounts of nitrous oxide. This has serious implications in the recovery room, where patients with low arterial oxygen tensions may go unrecognized.

Cerebral: By increasing cerebral blood flow, nitrous oxide produces a mild elevation of intracranial pressure. Nitrous oxide also increases cerebral oxygen consumption (CMRO₂). Levels of nitrous oxide below MAC provide analgesia in dental surgery and other minor procedures.

Gastrointestinal: Some studies have implicated nitrous oxide as a cause of postoperative nausea and vomiting, presumably as a result of activation of chemoreceptor trigger zone and the vomiting center in the medulla. Other studies have failed to demonstrate any association between nitrous oxide and emesis.

Biotransformation & Toxicity: During emergence, almost all nitrous oxide is eliminated by exhalation. A small amount diffuses out through the skin. Biotransformation is limited to the less than 0.01% that undergoes reductive metabolism in the gastrointestinal tract by anaerobic bacteria. By irreversibly oxidizing, the cobalt atom in vitamin B₁₂, nitrous oxide inhibits enzymes that are vitamin B₁₂ dependent. These enzymes include methionine synthetase, which is necessary for myelin formation, and thymidylate synthase, which is necessary for DNA
synthesis. Prolonged exposure to anesthetic concentrations of nitrous oxide can result in bone marrow depression and even neurologic deficiencies. Because of possible teratogenic effects, nitrous oxide is often avoided in pregnant patients. Nitrous oxide may also alter the immunologic response to infection by affecting chemotaxis and motility of polymorphonuclear leukocytes.

Contraindications: Although nitrous oxide is insoluble in comparison with other inhalational agents, it is 35 times more soluble than nitrogen in blood. Thus, it tends to diffuse into air-containing cavities more rapidly than nitrogen is absorbed by the bloodstream. For instance, if a patient with a 100-mL pneumothorax inhales 50% nitrous oxide, the gas content of the pneumothorax will tend to approach that of the bloodstream. Since nitrous oxide will diffuse into the cavity more rapidly than the air (principally nitrogen) diffuses out, the pneumothorax expands until it contains 100 mL of air and 100 mL of nitrous oxide. If the walls surrounding the cavity are rigid, pressure rises instead of volume. Examples of conditions where nitrous oxide might be hazardous included air embolism, pneumothorax, acute intestinal obstruction, intracranial air, pulmonary air cysts, intraocular air bubbles, and tympanic membrane grafting. Nitrous oxide will even diffuse into endotracheal tube cuffs, increasing the pressure against the tracheal mucosa.

Diffusion Hypoxia
Because of the effect of nitrous oxide on the pulmonary vasculature, it should be avoided in patients with pulmonary hypertension. Obviously, nitrous oxide is of limited value in patients requiring high inspired oxygen concentrations.

Drug Interactions: Because the relatively high MAC of nitrous oxide prevents its use as a complete general anesthetic, it is frequently used in combination with the more potent volatile agents. The addition of nitrous oxide decreases the requirements of these other agents. While nitrous oxide should not be considered a benign carrier gas, it does attenuate the circulatory and respiratory effects of volatile anesthetics in adults. Nitrous oxide potentiates neuromuscular blockade, but less so than the volatile agents. The concentration of nitrous oxide flowing through a vaporizer can influence the concentration of volatile anesthetic delivered. For example, decreasing nitrous oxide concentration increases the concentration of volatile agent despite a constant vaporizer setting. This disparity is due to the relative solubility’s of nitrous oxide and oxygen in liquid volatile anesthetics.
EMERGENCY MEDICINE

**Epinephrine**: Direct stimulation of β1 receptors by epinephrine raises cardiac output and myocardial oxygen demand by increasing contractility and heart rate (increased rate of spontaneous phase IV depolarization). Alpha stimulation decreases splanchnic and renal blood flow but increases coronary and cerebral perfusion pressure. Systolic blood pressure rises, although β2-mediated vasodilation in skeletal muscle may lower diastolic pressure. Beta2 stimulation also relaxes bronchial smooth muscle. Epinephrine administration is the principal pharmacologic treatment for anaphylaxis and ventricular fibrillation.

**Phenylephrine** is a noncatecholamine with predominantly direct α1 agonist activity. Its primary effect is peripheral vasoconstriction with a concomitant rise in systemic vascular resistance and arterial blood pressure. Reflex bradycardia can reduce cardiac output.

**Atropine’s** effects are due to binding to muscarinic acetylcholine receptors (M2). It is an muscarinic receptor antagonist agent. Muscarinic receptor antagonists bind to muscarinic receptors thereby preventing ACh from binding to and activating the receptor. By blocking the actions of ACh, muscarinic receptor antagonists very effectively block the effects of vagal nerve activity on the heart. By doing so, they increase heart rate. The vagus (parasympathetic) nerves that innervate the heart release acetylcholine (ACh) as their primary neurotransmitter. ACh binds to muscarinic receptors (M2) that are found principally on cells comprising the sinoatrial (SA) node. Muscarinic receptors are coupled to the mechanisms that stimulate the SA node to increase heart rate. In the resting state, there is a large degree of vagal tone on the heart (the vagus nerve is releasing ACh to activate M2 receptors that slow the heart rate). High vagal tone is responsible for low resting heart rates.

**Narcan** (naloxone) is indicated for the complete or partial reversal of opioid depression. Narcan (naloxone) prevents or reverses the effects of opioids including respiratory depression and sedation. Narcan is essentially a pure opioid antagonist. It blocks the action of opioids at the receptor - opioid interface through binding.

### Narcan Reversal Dosing

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Flumazenil: Flumazenil is a benzodiazepine antagonist. It works by blocking receptors in the brain and central nervous system that benzodiazepines need to reach to be active, which helps reduce drowsiness and sedation.

<table>
<thead>
<tr>
<th>REVERSAL AGENT</th>
<th>INCREMENTAL DOSES</th>
<th>MAXIMUM DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flumazenil</td>
<td>.2 mg every minute until reaching the desired degree of reversal</td>
<td>1.0 mg</td>
</tr>
</tbody>
</table>
RESPIRATORY SYSTEM

Useful definitions:

**pharynx** - The passageway between the mouth and the esophagus and trachea. Food passes from the pharynx to the esophagus, and air passes from the pharynx to the trachea.

**epiglottis** - A flap of tissue that closes off the trachea during swallowing.

**larynx** - A hollow structure at the beginning of the trachea. The vocal cords extend across the opening of the larynx.

**bronchi** - Tubes that carry air from the trachea to the lungs (sing.: bronchus).

**alveoli** - Tiny, thin-walled, inflatable sacs in the lungs where oxygen and carbon dioxide are exchanged.

**capillaries** - Small, thin-walled blood vessels that allow oxygen to diffuse from the blood into the cells and carbon dioxide to diffuse from the cells into the blood.

**ventilation** - The mechanics of breathing in and out through the use of the diaphragm and muscles in the wall of the thoracic cavity.

**metabolism** - The sum of all chemical reactions (energy exchanges) in cells.

**oxygenation** - The act or process of adding oxygen.

**apnea** - A disorder in which breathing stops for periods longer than 10 seconds during sleep; can be caused by failure of the automatic respiratory center to respond to elevated blood levels of carbon dioxide.

**hypoxia** - Insufficient oxygen reaching the blood. or Oxygen saturation levels below 85% in a patient.

I. Respiratory Anatomy and the Mechanics of Ventilation

The respiratory system includes the lungs, pathways connecting them to the outside environment, and structures in the chest involved with moving air in and out of the lungs. Air enters the body through the nose, is warmed, filtered, and passed through the nasal cavity. Air passes the pharynx (which has the epiglottis that prevents food from entering the trachea). The upper part of the trachea contains the larynx. The vocal cords are two bands of tissue that extend across the opening of the larynx. After passing the larynx, the air moves into the bronchi that carry air in and out of the lungs.

Bronchi are reinforced to prevent their collapse and are lined with ciliated epithelium and mucus-producing cells. Bronchi branch into smaller and smaller tubes known as bronchioles. Bronchioles terminate in grape-like sac clusters known as alveoli. Alveoli are surrounded by a network of thin-walled capillaries. Only about 0.2 µm separate the alveoli from the capillaries due to the extremely thin walls of both structures. Once air reaches the alveoli, oxygen diffuses from the alveoli into the surrounding capillaries. Room air is composed of 20% oxygen and 80% nitrogen. The atmospheric pressure and the 20% oxygen concentration create the major driving force that allows oxygen to enter the capillary blood stream.
The lungs are large, lobed, paired organs in the chest (also known as the thoracic cavity). Thin sheets of epithelium (pleura) separate the inside of the chest cavity from the outer surface of the lungs. The bottom of the thoracic cavity is formed by the diaphragm. This complex anatomical system is dependent on productive ventilation to function properly. Ventilation is the mechanics of breathing in and out. When you inhale, muscles in the chest wall contract, lifting the ribs and pulling them outward. The diaphragm at this time moves downward enlarging the chest cavity. Reduced air pressure in the lungs causes air to enter the lungs. Exhaling reverses these steps.

II. Oxygenation

It is important to understand why the human body is oxygen dependent. Each organ system requires energy in order to properly function. Oxygen is a crucial molecule in every organ's metabolic process. In general, the metabolic process involves cellular respiration, the Kreb's Cycle, and the Electron Transport System. Cellular respiration involves the breakdown of organic molecules to produce ATP. A sufficient supply of oxygen is required for the aerobic respiratory machinery of Kreb's Cycle and the Electron Transport System to efficiently convert stored organic energy into energy trapped in ATP. Organ's use ATP as their energy source to function. Carbon dioxide is also generated by cellular metabolism and must be removed from the cell. There must be an exchange of gases: carbon dioxide leaving the cell, oxygen entering. The respiratory system is involved in facilitating this exchange and red blood cells are involved in the transport of gases to and from exchange areas.
In order for organs to maintain proper function three steps need to occur. First, successful diffusion of oxygen from the alveoli into the blood occurs. Second, transport of oxygen to the tissues and cells of the body takes place. Third, diffusion of oxygen from the blood into cells completes the process. Diffusion is the movement of materials from a higher to a lower concentration. The differences between oxygen and carbon dioxide concentrations are measured by partial pressures. The greater the difference in partial pressure the greater the rate of diffusion. An organized mathematical way to think about partial pressures and oxygenation is outlined on the following page.
\[ p_{aO_2} = F_{1O_2}(P_{ATM} - p_{H_2O}) - p_{aCO_2} / RER \]

<table>
<thead>
<tr>
<th>Unit</th>
<th>Description</th>
<th>Sample value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( p_{aO_2} )</td>
<td>The alveolar partial pressure of oxygen (( pO_2 ))</td>
<td>107 mmHg (14.2 kPa)</td>
</tr>
<tr>
<td>( F_{1O_2} )</td>
<td>The fraction of inspired gas that is oxygen (expressed as a decimal).</td>
<td>0.21</td>
</tr>
<tr>
<td>( P_{ATM} )</td>
<td>The prevailing atmospheric pressure</td>
<td>760 mmHg (101 kPa)</td>
</tr>
<tr>
<td>( p_{H_2O} )</td>
<td>The saturated vapor pressure of water at body temperature and the prevailing atmospheric pressure</td>
<td>47 mmHg (6.25 kPa)</td>
</tr>
<tr>
<td>( p_{aCO_2} )</td>
<td>The arterial partial pressure of carbon dioxide (( pCO_2 ))</td>
<td>40 mmHg (4.79 kPa)</td>
</tr>
<tr>
<td>RER</td>
<td>The respiratory exchange ratio</td>
<td>0.8</td>
</tr>
</tbody>
</table>

The alveolar partial pressure of oxygenation equation demonstrates that oxygenation clinically is dependent on:

A. The oxygen concentration delivered
   - Room Air = 20%
   - Nasal Cannula = 30%

B. Ventilation
   - Partial or Complete Obstruction
   - Apnea
C. Elevation

- Atmospheric Pressure: As altitude increases, atmospheric pressure decreases. Above 10,000 feet, decreased oxygen pressures cause loading of oxygen into hemoglobin to drop off, leading to lowered oxygen levels in the blood. The result can be mountain sickness (nausea and loss of appetite). Mountain sickness does not result from oxygen starvation but rather from the loss of carbon dioxide due to increased breathing in order to obtain more oxygen.

D. Lung Disease:

However, delivery of oxygen is dependent on blood flow and the blood's oxygen content.

- Functional Blood Flow = Blood Pressure
- Oxygen Content: \( \text{O}_2 = \text{Hgb} \times \text{Sat} + 0.003(P_0) \)

In summary, increasing the delivered oxygen concentration, ensuring that the patient does not obstruct their airway, avoiding apnea, avoiding patients with lung disease, maintaining an appropriate saturation, and maintaining an appropriate blood pressure, all maximize effective oxygenation and delivery to vital organs.

III. Ventilation: Ventilation does provide oxygen flow to the lungs. However, its removal of carbon dioxide is equally as important to our body. Carbon dioxide follows a reverse path in the process of diffusion explained above. Carbon dioxide concentration in metabolically active cells is much greater than in capillaries, so carbon dioxide diffuses from the cells into the capillaries. Water in the blood combines with carbon dioxide to form bicarbonate. This removes the carbon dioxide from the blood so diffusion of even more carbon dioxide from the cells into the capillaries continues yet still manages to "package" the carbon dioxide for eventual passage out of the body. In the alveoli capillaries, bicarbonate combines with a hydrogen ion (proton) to form carbonic acid, which breaks down into carbon dioxide and water. The carbon dioxide then diffuses into the alveoli and out of the body with the next exhalation.

Clinically, ventilation is dependent on:

A. Respiratory Drive
B. An Unobstructed Airway
C. Minute Ventilation: \( \text{Minute Ventilation} = \text{Respiratory Rate} \times \text{Tidal Volume} \). Respiratory rate is defined as how many times a patient breathes per minute. Tidal Volume is defined as the volume of air a patient takes in with each breath. The product of each defines minute ventilation. Ventilation is a key component in both delivering oxygen to the lungs and removing carbon dioxide from the lungs. All oral sedatives depress ventilation to some degree.

IV. Respiratory Drive: The body's respiratory drive is what generates the body's impulse to breath. Muscular contraction and relaxation controls the rate of expansion and constriction of the lungs. These muscles are stimulated by nerves that carry messages from the part of the brain that controls breathing, the medulla. Two systems control breathing: an automatic response and a voluntary response. Both are involved in holding your breath. Although the automatic breathing regulation system allows you to breathe while you sleep, it sometimes malfunctions. Apnea involves stoppage of breathing for as long as 10 seconds. In some individuals, apnea occurs as often as 300 times per night. This failure to respond to elevated blood levels of carbon dioxide may result from high doses sedatives, sleep apnea, viral infections of the brain, or tumors.
malfunction of the breathing centers in newborns may result in SIDS (sudden infant death syndrome). The automatic response (innate drive to breath) is composed of the following mechanisms

- **A. Carotid bodies** - Neural tissue located on the carotid arteries that senses the oxygen level in the blood stream. As the oxygen level drops, the carotid body transmits a very potent signal to breath through the central nervous system. This mechanism is known as the HYPOXIC DRIVE.

- **B. CO2 receptors in the brainstem** - The CO2 receptors in the brainstem sense the amount of CO2 in the blood stream. As sedation medicine blunts this mechanism ventilation slows. CO2 levels in the blood stream then rise. (Not as much CO2 is blown off by the respiratory system). Eventually, CO2 levels rise to the point that the respiratory drive again is generated.

- **C. Receptors in the lungs** - As the lungs expand, the j receptors remain inactive. When the rhythmic expansion of the lungs is diminished, the receptors generate a signal that is perceived as “breath holding discomfort.”

- **D. The Reticular Activating System of the midbrain** - This system sets the global respiratory rate when the body is at rest.

The clinical application of respiratory drive physiology is very important when patients are exposed to sedatives. Sedatives depress the respiratory drive -> ventilation slows -> the patient is more susceptible to becoming hypoxic.

**IV. Respiratory Reserve**: Respiratory reserve is the lung volume that does not interact with capillaries. This volume is a reservoir for the active lung volume that has oxygen taken away by capillaries. The oxygen travels from inactive lung chambers to active lung chambers through pores. Therefor, when ventilation is partially obstructed, completely obstructed, or the patient stops breathing, the reserve lung volume provides oxygen for the body.

<table>
<thead>
<tr>
<th>MEASUREMENT</th>
<th>DEFINITION</th>
<th>AVERAGE VALUES (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Volume (TV)</td>
<td>Each normal breath</td>
<td>500</td>
</tr>
<tr>
<td>Inspiratory reserve volume (IRV)</td>
<td>Maximum volume that can be inspired above TV</td>
<td>3000</td>
</tr>
<tr>
<td>Expiratory reserve volume (ERV)</td>
<td>Maximum volume that can be expired below TV</td>
<td>1100</td>
</tr>
<tr>
<td>Residual volume (RV)</td>
<td>Volume remaining after maximal exhalation</td>
<td>1200</td>
</tr>
<tr>
<td>Total lung capacity (TLC)</td>
<td>RV + ERV + TV + IRV</td>
<td>5800</td>
</tr>
<tr>
<td>Functional residual capacity (FRC)</td>
<td>RV + ERV</td>
<td>2300</td>
</tr>
</tbody>
</table>

**V. Hypoxia**: Hypoxia is insufficient oxygen reaching the blood or oxygen saturation levels below 85% in a patient. Hypoxia is the most expensive mismanaged morbidity in dental offices that provide sedation. However, the causes of hypoxia are predictable and usually quickly reversed with basic airway management. To practice superior sedation dentistry, memorize the four physiologic causes of hypoxia. 1) HYPOVENTILATION 2) V/Q MISMATCH 3) SHUNT 4) DIFFUSION BARRIER

**V. Transport of O2 and CO2**: The average adult has about five liters of blood living inside of their body, coursing through their vessels, delivering essential elements, and removing harmful wastes. Without blood, the human body would stop working.

© RMS–PLLC
Blood is the fluid of life, transporting oxygen from the lungs to body tissue and carbon dioxide from body tissue to the lungs. Blood is the fluid of growth, transporting nourishment from digestion and hormones from glands throughout the body. Blood is the fluid of health, transporting disease fighting substances to the tissue and waste to the kidneys. Red blood cells are red only because they contain a protein chemical called hemoglobin which is bright red in color. Hemoglobin contains the element Iron, making it an excellent vehicle for transporting oxygen and carbon dioxide. As blood passes through the lungs, oxygen molecules attach to the hemoglobin. As the blood passes through the body’s tissue, the hemoglobin releases the oxygen to the cells. The empty hemoglobin molecules then bond with the tissue’s carbon dioxide or other waste gases, transporting it away. In order for transport of oxygen and carbon dioxide to occur, functional blood pressures with intact blood vessels circulate blood utilizing the heart as the primary pump. The circulatory system will be outlined in the cardiovascular physiology section.
I. BLOOD: The average adult has about five liters of blood living inside of their body, coursing through their vessels, delivering essential elements, and removing harmful wastes. Without blood, the human body would stop working. Blood is the fluid of life, transporting oxygen from the lungs to body tissue and carbon dioxide from body tissue to the lungs. Blood transports nourishment from digestion and hormones from glands throughout the body. Blood also transports disease fighting substances to the tissue and waste to the kidneys.

Red blood cells are red only because they contain a protein chemical called hemoglobin which is bright red in color. Hemoglobin contains the element Iron, making it an excellent vehicle for transporting oxygen and carbon dioxide. As blood passes through the lungs, oxygen molecules attach to the hemoglobin. As the blood passes through the body's tissue, the hemoglobin releases the oxygen to the cells. The empty hemoglobin molecules then bond with the tissue's carbon dioxide or other waste gases, transporting it away.

II. CIRCULATORY SYSTEM: On average, your body has about 5 liters of blood continually traveling through it by way of the circulatory system. The heart, the lungs, and the blood vessels work together to form the circle part of the circulatory system. The pumping of the heart forces the blood on its journey. The body's circulatory system really has three distinct parts: pulmonary circulation (the lungs), coronary circulation (the heart), and systemic circulation (body's other organs). Each part must be working independently in order for them to all work together. Pulmonary circulation is the movement of blood from the heart, to the lungs, and back to the heart again. Systemic circulation supplies nourishment to all of the tissue located throughout your body, with the exception of the heart and lungs because they have their own systems. While the circulatory system is busy providing oxygen and nourishment to every cell of the body, let's not forget that the heart, which works hardest of all, needs nourishment, too. Coronary circulation refers to the movement of blood through the tissues of the heart. The circulation of blood through the heart is just one part of the overall circulatory system.

III. THE HEART: The human heart is primarily a shell. There are four cavities, or open spaces, inside the heart that fill with blood. Two of these cavities are called atria. The other two are called ventricles. The two atria form the curved top of the heart. The ventricles meet at the bottom of the heart to form a pointed base which points toward the left side of your chest. The left ventricle contracts most forcefully, so you can best feel your heart pumping on the left side of your chest. The left side of the heart houses one atrium and one ventricle. The right side of the heart houses the others. A wall, called the septum, separates the right and left sides of the heart. A valve connects each atrium to the ventricle below it. The mitral valve connects the left atrium with the left ventricle. The tricuspid valve connects the right atrium with the right ventricle. The top of the heart connects to a few large blood vessels. The largest of these is the aorta, or main artery, which carries nutrient-rich blood away from the heart. Another important vessel is the pulmonary artery which connects the heart with the lungs as part of the pulmonary circulation system. The two
largest veins that carry blood into the heart are the superior vena cava and the inferior vena cava. They are called "vena cava" because they are the "heart's veins." The superior is located near the top of the heart. The inferior is located beneath the superior. The heart's structure makes it an efficient, never-ceasing pump.

The heart can be functionally divided into right and left pumps, each consisting of an atrium and a ventricle. The atria serve as both conduits and priming pumps, while the ventricles act as the major pumping chambers. The right ventricle receives systemic venous (deoxygenated) blood and pumps it into the pulmonary circulation, while the left ventricle receives pulmonary venous (oxygenated) blood and pumps it into the systemic circulation. Four valves normally ensure unidirectional flow through each chamber. The normal pumping action of the heart is the result of a complex series of electrical and mechanical events. The heart consists of specialized striated muscle in a connective tissue skeleton. Cardiac muscle can be divided into atrial, ventricular, and specialized pacemaker and conducting cells. The self-excitatory nature of cardiac muscle cells and their unique organization allow the heart to function as a highly efficient pump. Serial low-resistance connections (intercalated disks) between individual myocardial cells allow the rapid and orderly spread of electrical activity in each pumping chamber. Electrical activity readily spreads from one atrium to another and from one ventricle to another via specialized conduction pathways.

The absence of direct connections between the atria and ventricles except through the atrioventricular (AV) node delays conduction and enables atrial contraction to prime the ventricle.

IV. BLOOD VESSELS: In a general sense, a vessel is defined as a hollow utensil for carrying something: a cup, a bucket, a tube. Blood vessels, then, are hollow utensils for carrying blood. Located throughout your body, your blood vessels are hollow tubes that circulate your blood. There are three varieties of blood vessels: arteries, veins, and capillaries. During blood circulation, the arteries carry blood away from the heart. The capillaries connect the arteries to veins. Finally, the veins carry the blood back to the heart.

ARTERIES: The heart pumps blood out through one main artery called the aorta. The main artery then divides and branches out into many smaller arteries so that each organ of your body has its own system of arteries supplying it with fresh, oxygen-rich blood.

Arteries are tough on the outside and smooth on the inside. An artery actually has three layers: an outer layer of tissue, a muscular middle, and an inner layer of epithelial cells. The muscle in the middle is elastic and very strong. The inner layer is very smooth so that the blood can flow easily with no obstacles in its path. The muscular wall of the artery helps the heart pump the blood. When the heart beats, the artery expands as it fills with blood. When the heart relaxes, the artery contracts, exerting a force that is strong enough to push the blood along. This rhythm between the heart and the artery results in an efficient circulation system. You can actually feel your artery expand and contract. Since the artery keeps pace with the heart, we can measure heart rate by counting the contractions of the artery. That's how we take our pulse.

The arteries deliver the oxygen-rich blood to the capillaries where the actual exchange of oxygen and carbon dioxide occurs. The capillaries then
deliver the waste-rich blood to the veins for transport back to the lungs and heart.

**CAPILLARIES**

Unlike the arteries and veins, capillaries are very thin and fragile. The capillaries are actually only one epithelial cell thick. They are so thin that blood cells can only pass through them in single file. The exchange of oxygen and carbon dioxide takes place through the thin capillary wall. The red blood cells inside the capillary release their oxygen which passes through the wall and into the surrounding tissue. The tissue releases its waste products, like carbon dioxide, which passes through the wall and into the red blood cells. Arteries and veins run parallel throughout the body with a web-like network of capillaries, embedded in tissue, connecting them. The arteries pass their oxygen-rich blood to the capillaries which allow the exchange of gases within the tissue. The capillaries then pass their waste-rich blood to the veins for transport back to the heart.

**VEINS:** The veins bring waste-rich blood back to the heart, entering the right atrium throughout two large veins called vena cavae. The right atrium fills with the waste-rich blood and then contracts, pushing the blood through a one-way valve into the right ventricle. The right ventricle fills and then contracts, pushing the blood into the pulmonary artery which leads to the lungs. In the lung capillaries, the exchange of carbon dioxide and oxygen takes place. The fresh, oxygen-rich blood enters the pulmonary veins and then returns to the heart, re-entering through the left atrium. The oxygen-rich blood then passes through a one-way valve into the left ventricle where it will exit the heart through the main artery, called the aorta. The left ventricle’s contraction forces the blood into the aorta and the blood begins its journey throughout the body.

The one-way valves are important for preventing any backward flow of blood. The circulatory system is a network of one-way streets. If blood started flowing the wrong way, the blood gases (oxygen and carbon dioxide) might mix, causing a serious threat to your body.

Besides circulating blood, the blood vessels provide two important means of measuring vital health statistics: pulse and blood pressure. We measure heart rate, or pulse, by touching an artery. The rhythmic contraction of the artery keeps pace with the beat of the heart. Since an artery is near the surface of the skin, while the heart is deeply protected, we can easily touch the artery and get an accurate measure of the heart’s pulse.

When we measure blood pressure, we use the blood flowing through the arteries because it has a higher pressure than the blood in the veins. Your blood pressure is measured using two numbers. The first number, which is higher, is taken when the heart beats during the systole phase. The second number is taken when the heart relaxes during the diastole phase. Those two numbers stand for millimeters. A column of mercury rises and falls with the beat of the heart. The height of the column is measured in millimeters. Normal blood pressure ranges from 110 to 150
millimeters (as the heart beats) over 60 to 80 millimeters (as the heart relaxes). It is normal for your blood pressure to increase when you are exercising and to decrease when you are sleeping. If your blood pressure stays too high or too low, however, you may be at risk of heart disease.

V. CARDIAC PHYSIOLOGY:
Ventricular systolic function, Heart Rate, and Stroke Volume are the major determinants of ventricular performance. Discussions of ventricular function usually refer to the left ventricle, but the same concepts apply to the right ventricle. Although the ventricles are often thought of as functioning separately, their interdependence has clearly been demonstrated. Moreover, factors affecting systolic and diastolic functions can be differentiated: Systolic function involves ventricular ejection, while diastolic function is related to ventricular filling. Ventricular systolic function is most often equated with cardiac output, which can be defined as the volume of blood pumped by the heart per minute. Because the two ventricles function in series, their outputs are normally equal.

Cardiac output (CO) is expressed by the following equation:

- \( CO = SV \times HR \) Where \( SV \) is the stroke volume (the volume pumped per contraction) and \( HR \) is heart rate.
- To compensate for variations in body size, \( CO \) is often expressed in terms of total body surface area: \( CI = \frac{CO}{BSA} \) Where \( CI \) is the cardiac index and \( BSA \) is total body surface area. \( BSA \) is usually obtained from normograms based on height and weight.

Heart Rate: Cardiac output is generally directly proportional to heart rate. Heart rate is an intrinsic function of the SA node (spontaneous depolarization) but is modified by autonomic, humeral, and local factors. The normal intrinsic rate of the SA node in young adults is about 90-100 beats/minute, but it decreases with age according to the following formula:

- Normal intrinsic heart rate = 118 beats/min – (0.57 x age)
- Enhanced vagal activity slows the heart rate via stimulation of muscarinic (M2) cholinergic receptors, while enhanced sympathetic activity increases heart rate mainly through activation of \( \beta_1 \) – adrenergic receptors and, to lesser extent, \( \beta_2 \) – adrenergic receptor

Stroke Volume: Stroke volume is normally determined by three major factors: preload, afterload, and contractility. This analysis is analogous to laboratory observations on skeletal muscle preparations. Preload is muscle length prior to contraction, while afterload is the tension against which the muscle must contract. Contractility is an intrinsic property of the muscle that is related to the force of contraction but independent of both preload and afterload. Since the heart is a three-dimensional multi chambered pump, both ventricular geometric form and valvular dysfunction can also affect stroke volume.

- Preload: Ventricular preload is end-diastolic volume, which is generally dependent on ventricular filling. The relationship between cardiac output and left ventricular end-diastolic volume is known as Starling’s law of the heart.
- Afterload: Afterload for the intact heart is commonly equated with either ventricular wall tension during systole or arterial impedance to ejection.
- Contractility: Cardiac contractility (inotropism) is the intrinsic ability of the myocardium to pump in the absence of changes in preload or afterload.

VI. CONDUCTION:
Electrocardiography (ECG or EKG) is the recording of the electrical activity of the heart. Traditionally this is in the form of a transthoracic (across the thorax or chest) interpretation of the electrical activity of the heart over a period of time, as detected by electrodes attached to the surface of the skin and
recorded or displayed by a device external to the body. The recording produced by this noninvasive procedure is termed an electrocardiogram (also ECG or EKG).

An ECG is used to measure the heart’s electrical conduction system. It picks up electrical impulses generated by the polarization and depolarization of cardiac tissue and translates into a waveform. The waveform is then used to measure the rate and regularity of heartbeats, as well as the size and position of the chambers, the presence of any damage to the heart, and the effects of drugs or devices used to regulate the heart.

MONITORS

One of the primary responsibilities of the dentist during oral sedation is to be the guardian for the sedated patient during the procedure. Optimal vigilance requires an understanding of the monitors. An in depth study of how all of the required monitors work will be taught at the course. It is important to memorize the below information. This will allow a systematic and efficient skill set when evaluating the patient’s physiologic profile during sedation.

Utilize the monitors with a system:

(I) VENTILATION → (II) OXYGENATION → (III) CIRCULATION

The components of each are as follows:

I. OXYGENATION→Clinical: Skin, Lips, Gums, Blood→Monitors: Pulse Ox

II. VENTILATION→Clinical: Chest, Abdomen, Breath Sounds, Verbalization→Monitors: Capnogram, Precordial Stethoscope, Stethoscope

III. CIRCULATION→Clinical: Palpable Pulse, Capillary Refill Time→Monitors: EKG, NIBP

I. Oxygenation (Pulse Oximetry) Oxygenation refers to the concentration of oxygen in the blood. It can be determined rapidly through the use of a basic clinical exam.

• Skin color
• Flush lips
• Conjunctiva
• Capillary refill

Pulse oximetry is a procedure used to measure the oxygen level (or oxygen saturation) in the blood. It is considered to be a noninvasive, painless, general indicator of oxygen delivery to the peripheral tissues (such as the finger, earlobe, or nose). Oxygen in the air is breathed into the lungs. The oxygen then passes into the blood where the majority of the oxygen attaches to hemoglobin (a protein located inside the red blood cell) for transport in the bloodstream. The oxygenated blood circulates to the tissues.

Pulse oximetry technology utilizes the light absorptive characteristics of hemoglobin and the pulsating nature of blood flow in the arteries to aid in determining the oxygenation status in the body. First, there is a color difference between arterial hemoglobin
saturated with oxygen, which is bright red, and venous hemoglobin without oxygen, which is darker. Second, with each pulsation or heartbeat there is a slight increase in the volume of blood flowing through the arteries. Because of the increase of blood volume, albeit small, there is an associated increase in oxygen-rich hemoglobin. This represents the maximum amount of oxygen-rich hemoglobin pulsating through the blood vessels. A clip-like device called a probe is placed on a body part, such as a finger or ear lobe, to measure the blood that is still carrying or is saturated with oxygen. The probe houses a light source, a light detector, and a microprocessor, which compares and calculates the differences in the oxygen-rich versus oxygen-poor hemoglobin.

The four pieces of valuable information that can be determined from pulse oximetry:
1. Oxygen Saturation - primary information
2. HR (Heart Rate) - measured through capillary pulsations
3. Rhythm (Approximate) - if the pulse oximetry beats are regular, it is unlikely that an arrhythmia is present
4. Perfusion (Approximate) - because the device senses capillary pulsations, this means some degree of tissue perfusion is occurring POINT: a blood pressure is being generated by the heart!

Pulse oximeters are mandatory sedation monitors for dental sedation. Oximetry depends upon the observation that oxygenated and reduced hemoglobin differ in their absorption of red and infrared light. Oxyhemoglobin absorbs more infrared light 990nm. Deoxyhemoglobin absorbs more red light 660nm. The change in light absorption during arterial pulsations is the basis for oximetry determinations.

II. Ventilation (Capnography / Plethoragraph / Precordial Stethoscope)
Clinical Exam: Misting - Chest excursion - Audible sounds

Capnography: This monitor determines the presence of end tidal CO2 to confirm ventilation. Aspiration technique: Aspiration capnographs continuously suction gas from the mouth into a sample cell within the monitor. End tidal CO2 monitoring is now the standard of care for moderate sedation. Capnography provides the following information: (1) Confirmation of ventilation and (2) Respiratory rate

Plethoragraph: This monitor determines rhythmic chest movement via the EKG leads. The plethoragraph determines a chest excursion rate BUT DOES NOT verify ventilation.
**Precordial Stethoscope:** The precordial stethoscope is a mainstay tool that assists in detecting airflow through the large airways. This is yet another way to evaluate ventilation.

**Stethoscope:** The stethoscope allows the practitioner to listen for the heart rate, rhythm, and pathological sounds.

**Circulation (NIBP / EKG / Stethoscope)**

Clinical assessment
- Radial Pulse: HR, Rhythm, Perfusion Pressure
- Brachial Pulse: HR, Rhythm, Perfusion Pressure

Noninvasive arterial blood pressure monitoring detects blood pressure via Oscillometry. Oscillometry: Arterial pressures cause oscillations in cuff pressure. These oscillations are small if the cuff pressure is inflated above the SBP. When the cuff pressure decreases to the SBP, the oscillations markedly increase. At this time, the SBP is detected.

Definitions:
- Systolic arterial blood pressure (SBP) is the peak pressure generated during systolic contraction
- Systolic Arterial Blood Pressure (SBP) is the peak pressure generated during systolic contraction
- Diastolic Arterial Blood Pressure (DBP) is the trough pressure during diastolic relaxation
- Mean Arterial Pressure (MAP) is the time weighted average of arterial pressures during a pulse cycle
  \[ MAP = \frac{(SBP) + 2(DBP)}{3} \]
- NIBP monitoring gives the following information: SBP, DBP, MAP, HR
Electrocardiography:
The EKG is a recording of the electrical potentials generated by myocardial cells. It helps detect dysrhythmias, myocardial ischemia, conduction issues and electrolyte disturbances. Patient movement electric surges and faulty electrodes can yield false signs. The three lead system is the most common used for sedation.
AIRWAY MANAGEMENT

Practically, when an airway presents with partial obstruction, the doctor performs many tasks at the same time.
1. Reposition the airway (this simultaneously stimulates the patient) and change the gas flow to deliver 100% oxygen.
2. Determine if the patient is making an effort to breathe.
3. If the airway management is insufficient with these efforts, call for help and immediately use airway support equipment.
   • Oral airway
   • Ambu Bag
   • Consider use of an LMA

If the patient’s airway is successfully managed and there is no respiratory effort, it is appropriate to reverse the benzodiazepine at this time. The primary duty of the practitioner is to establish and maintain a safe airway FIRST.
   • Narcan is used to reverse opioids.
   • Flumazenil is used to reverse benzodiazepines.

AIRWAY EQUIPMENT AND PROTOCOLS

Equipment
1. Oral & Nasal airways
   • Loss of upper airway muscle tone in sedated patients allows the tongue and epiglottis to fall back against the posterior wall of the pharynx.
   • Oral and nasal airways create an air passage between the tongue and the posterior wall.
   • Awake or lightly anesthetized patients may cough or even develop laryngospasm if placed without evaluation.

2. Face Mask
   • A facemask is designed to deliver a higher concentration of oxygen than room air.

<table>
<thead>
<tr>
<th></th>
<th>FiO2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Room air</td>
<td>20%</td>
</tr>
<tr>
<td>Nasal cadula</td>
<td>~30%</td>
</tr>
<tr>
<td>Face mask</td>
<td>~50% - 70% *dependent on mask design.</td>
</tr>
</tbody>
</table>

• Effective positive pressure ventilation with an ambu bag requires both a gas-tight mask fit and a patent airway.
• Technique: TWO HANDS
3. Laryngeal mask
   • Large bore tube connected to an elliptical cuff.
   • Laryngeal mask provides a conduit for ventilation and oxygenation but does not protect against gastric regurgitation.
   • The depth of sedation to insert this is moderate to deep.

4. Endotracheal Tube
   • Endotracheal tubes deliver gasses directly to the trachea
   • Temporary paralysis or a state of profoundly deep sedation with narcotics is required to prevent vocal cord spasm.
   • A cuffed tube has an inflatable cuff that is placed below the glottis.

5. ALWAYS: Prior to any sedation case ALWAYS assess the likelihood of:
   • Difficult intubation
   • Difficult ventilation

6. Emergent airway:
   • Loss of consciousness with aspiration potential
   • Hypoxia with no signs of a respiratory drive
   • Unable to mask ventilate
     a. IMMEDIATELY adjust the Mask Ventilation Technique with %100 O2
        1) Positioning
        2) Oral Airway
        3) Nasal Airway
        4) LMA
     b. If adjustments are unsuccessful, assess, choose, and preform the technique required for establishing an airway:
        1) LMA
        2) Non-surgical vs. Surgical intubation
        3) Awake vs. Induction
     c. Diagnose and treat the cause of:
        1) Hypoxia
        2) Loss of Respiratory Drive
        3) Obstruction
HYPOXIA
AIRWAY ALGORHYTHM

DOES THE PATIENT SHOW AN EFFORT TO BREATHE?

YES

IS THE AIRWAY OBSTRUCTED?

DIAGNOSIS OPTIONS:
1. LISTEN TO PRECORDIAL STETHASCOPE
2. OBSERVE CAPNOGRAM
3. FEEL FOR A PRODUCTIVE BREATH
4. ASK THE PATIENT TO TALK

YES

MOST LIKELY CAUSES OF OBSTRUCTION

PATIENTS AIRWAY ANATOMY DUE TO THE DEPTH OF SEDATION

FOREIGN BODY

LARYNGOSPASM

NO

APNEA

MOST LIKELY CAUSE: SEDATIVES
TREATMENT:
1. POSITIVE PRESSURE VENTILATION WITH 100% O2 + AMBU BAG
2. ONCE AIRWAY IS MANAGED, CONSIDER REVERSAL

NO

CONSIDER THE FOLLOWING

MODERATE TO SEVERE HYPOVENTILATION

BRONCHOSPASM

ASTHMA / COPD

ASPIRATION

ANAPHYLAXIS
OFFICE SETUP

Basic Office Setup
1. 911 designator
2. ACLS certified staff and ACLS appointee
3. Ancillary staff
4. Monitoring Layout
5. Emergency Equipment Layout
6. Location of ACLS handbook and Emergency protocols
7. 911 entrance and exit
8. Reserve positive pressure oxygen delivery
9. Lighting
10. Recovery area and standards
11. Medication storage area - Federal Regulations
12. Gas storage area
13. Sterilization area
14. Designated supplies/equipment room

Facility and Equipment Standards:
1) An operatory large enough to adequately accommodate the patient and permit a team consisting of at least three individuals to freely move about the patient.
2) A table or dental chair which permits the patient to be positioned so the attending team can maintain the airway, quickly alter patient position in an emergency, and provide a firm platform for the management of CPR.
3) A lighting system which is adequate to permit evaluations of the patient's skin and mucosal color and a backup lighting system which is battery powered and of sufficient intensity to permit completion of any treatment which may be underway at the time of a general power failure.
4) An appropriate functional suctioning device that permits aspiration of the oral and pharyngeal cavities. A backup suction device that can function at the time of general power failure must also be available.
5) A positive-pressure oxygen delivery system capable of administering greater than 90% oxygen at 10 liter/minute flow for at least sixty minutes (650 liter “E” cylinder), even in the event of a general power failure.
6) Inhalation sedation equipment, if used in conjunction with oral sedation, must have the capacity for delivering 100%, and never less than 25% oxygen concentration and have a fail safe system. The equipment must be maintained and checked for accuracy at least annually.

Ancillary Equipment:
1) Age appropriate oral airways capable of accommodating patients of all sizes
2) Age-appropriate sphygmomanometer with cuffs of appropriate size for patients of all sizes
3) Precordial/pretracheal stethoscope
4) Pulse oximeter
Records Maintenance:
1) Adequate medical history and physical evaluation updated prior to each administration of oral conscious sedation including but not limited to:
   • Visual examination of the airway
   • Age/sex/weight/physical status
   • Rationale for sedation
   • Written informed consent

1) Conscious sedation records shall include:
   • Baseline vital signs
   • Intermittent quantitative monitoring
   • Recording of oxygen saturation
   • Heart and respiratory rates
   • Blood pressure
   • Name, dose and time of administration of all drugs administered including local and inhalation anesthetics
   • Length of procedure
   • Any complications of sedation
   • Statement of the patient's condition at time of discharge

Emergency Cart:
1) An emergency cart or kit shall be available and readily accessible and shall include the necessary and appropriate drugs and size appropriate equipment to resuscitate a non-breathing and unconscious patient and provide continuous support while the patient is transported to a medical facility.
2) There must be documentation that all emergency equipment and drugs are checked and maintained on a prudent and regularly scheduled basis.
3) Emergency drugs of the following types shall be available:
   • Epinephrine
   • Bronchodilator
   • Appropriate drug antagonists
   • Antihistaminic
   • Anticholinergic
   • Anticonvulsant
   • Oxygen
   • Dextrose or other antihypoglycemic

Kansas Dental Practices Act Statutes, Regulations, And Related Laws Pertaining To Dentists 71-5-10.
Level I permit: enteral conscious sedation or combination inhalation-enteral conscious sedation. p 62-63

Before administering enteral conscious sedation or combination inhalation-enteral conscious sedation, you must perform the following:
1. Review the patient’s medical history and current medications
2. Patients with a severe systemic disease, consult with the patient’s primary care physician or any consulting medical specialist regarding the potential risks
3. Document that the patient or guardian received written preoperative instructions, including dietary instructions that are based on the sedation technique to be used and the patient’s physical status, and that the patient or guardian reported that the patient complied with the instructions
4. Obtain from the patient or guardian a signed informed consent form
5. Evaluate the inhalation equipment for proper operation
6. Determine that an adequate oxygen supply is available and can be delivered to the patient if an emergency occurs
7. Obtain the patient’s vital signs and perform a patient assessment
8. Confirm the time when the patient last took any solid or liquid by mouth
During the administration of enteral conscious sedation or combination inhalation-ental conscious sedation, each treating dentist shall ensure that both of the following conditions are met:

1. At least one additional staff person who has either a current “basic cardiac life support for the health care provider” certificate from the American heart association or a current certificate deemed equivalent by the board from a provider approved by the board is present.
2. The following equipment is available and in working order:
   a. A pulse oximeter
   b. A drug kit that includes an agent to reverse the effects of the sedation agent administered, if an agent to reverse the effects of the sedation agent is commercially available
   c. A bag-valve mask with patient-appropriate masks that have all connections necessary to attach the bag-valve mask to a 100 percent oxygen source or a separate positive-pressure oxygen source
   d. Oropharyngeal airways in patient-appropriate sizes.

Whenever enteral conscious sedation or combination inhalation-ental conscious sedation is administered, each treating dentist shall cause the following records to be contemporaneously created. These records shall be maintained, for at least 10 years, as part of each patient’s record:

1. The date, the type of procedure, the personnel present, and the patient’s name, address, and date of birth
2. Documentation of the sedative agents administered, the approximate time when the sedative agents were administered, the amount of each agent administered, and the patient’s blood pressure, heart rate, and oxygen saturation readings at the start of sedation and at the end of the surgical or operative procedure and at 15-minute intervals throughout the procedure
3. An indication of the extent to which the effects of the sedation had abated at the time of the patient’s release
4. The gases used, with flow rates expressed in liters per minute or relative percentages, and the amount of time during which each gas was administered
5. The full name of the person to whom the patient was released
6. A record of all prescriptions written or ordered for the patient
7. Each type of monitor used

During the administration of enteral conscious sedation or combination inhalation-ental conscious sedation and the recovery phase, the treating dentist shall ensure that all of the following conditions are met:

1. The patient is continuously observed
2. The patient is continuously monitored with a pulse oximeter
3. The patient’s respiration is continuously confirmed
4. The patient’s blood pressure, heart rate, and oxygen saturation reading are recorded at least every 15 minutes
5. The patient’s ability to appropriately respond to physical stimulation or verbal command is documented every 15 minutes.
Following the administration of enteral conscious sedation or combination inhalation-enteral conscious sedation and during the recovery phase, each treating dentist shall ensure that all of the following conditions are met:

1. Oxygen and suction equipment are immediately available in the recovery area
2. The patient is continuously supervised until oxygenation, ventilation, and circulation are stable and until the patient is appropriately responsive for discharge from the facility
3. Written and verbal postoperative instructions, including an emergency telephone number to contact the treating dentist, are provided to the patient, guardian, or any escort present at the time of discharge
4. The patient meets the discharge criteria established by the treating dentist, including having stable vital signs, before leaving the office

Whenever enteral conscious sedation or combination inhalation-enteral conscious sedation is administered, each treating dentist shall cause the following information to be entered into a sedation log:

1. The name of each patient
2. The date of administration of each sedative agent
3. The name, strength, and dose of each sedative agent. Each entry shall be maintained for at least 10 years.

PRESCREENING

Prescreening is performed with a format that ensures medical issues are not missed. A patient may state that they have no medical problems. However, when their medication list is reviewed, they reveal use of anti hypertensives. From the patient’s perspective, the medicine controls their blood pressure so they are "fixed." This is just one of many examples that demonstrate how a format can eventually expose the patients true medical profile.

- The question "Do you have any medical problems?" did not reveal HTN.
- The medication list revealed HTN
PRESCREENING FORMAT

1. Can I manage the patients airway?
   a. Class I-III airways: YES
   b. Class IV airway: NO
2. Name, DOB
3. Sedation Indication
4. NPO status
5. Weight in kg
6. Allergies
7. Medication list
8. Consult / Studies
9. Medical problems
10. Surgery History
11. Recent Hospitalizations
12. Recent ER / Urgent Care visits
13. Last visit to primary care MD

If you stick to this format, rarely will any major medical problems be missed. The prescreening matrix below lists major disease states by the organ system affected. In general, a series of questions can be applied to any of the disease states to determine if they are medically stable. For dental sedation, any disease state needs to be MEDICALLY OPTIMIZED prior to sedation exposure.

HELPFUL QUESTIONS

1. When is the last time you visited you doctor for ________?
2. Have you had medication changes in the past three months for your ________?
3. Have you been to an ER/Hospital/Urgent care in the last 6 months for your ____?
4. Can you exert yourself like you used to be able to?
   • Walking your dog?
   • Pushing a shopping cart?
   • Walking up stairs?
   • Physical hobbies?

PRESCREENING

<table>
<thead>
<tr>
<th>Cardiovascular</th>
<th>Musculoskeletal</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Congestive heart failure</td>
<td>• Patients with any disease state that require assistance walking or are wheel chair bound.</td>
</tr>
<tr>
<td>• New onset arrhythmia</td>
<td>• Post-surgery patients</td>
</tr>
<tr>
<td>• Medically optimized arrhythmia</td>
<td></td>
</tr>
<tr>
<td>• Myocardial infarction less than</td>
<td></td>
</tr>
<tr>
<td>3 months</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiratory</th>
<th>GI</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Sleep apnea</td>
<td>• Acute pancreatitis</td>
</tr>
<tr>
<td>• COPD</td>
<td>• Liver disease</td>
</tr>
<tr>
<td>• Asthma</td>
<td></td>
</tr>
<tr>
<td>• Recent cough or cold</td>
<td></td>
</tr>
<tr>
<td>• Pneumonia</td>
<td></td>
</tr>
</tbody>
</table>
## Neurologic
- Age greater than 70
- Cognitive impairment
- Recent stroke less than 3 months
- Dementia, Alzheimer’s, Parkinson’s

## Renal
- End stage renal disease
- Acute renal failure

### GENERAL RULES
1. No sedation for Moderate - Severe Sleep Apnea
2. No sedation for Severe Asthma
3. CAD / Stroke - must wait 6 months post incident
4. Pneumonia - must wait two weeks after last Abx dose has been taken

### Functions of the Prescreening Process
The prescreening process in dental and oral surgery suites is very important when sedation is introduced. The patient now is in a cognitive state that prevents reliable communication about their pre-existing disease states. For example, sedated patients do not communicate that they may become hypoglycemic. This manual will review general prescreening that is followed by a thorough review of cardiac prescreening.

### GENERAL PRESCREENING

#### The prescreening goals:
1. Assess patients' medical status as soon as they are scheduled for surgical sedation.
2. If needed, obtain diagnostic tests to medically screen patients.
3. Identify potential patients at risk.
4. Increase patient confidence in how to prepare, what to expect and what they will physically encounter during their surgical procedure and how that may affect their care after surgery.
5. Inform patients about the risks and benefits of different types of sedation.
   a. Provide patient-specific instructions (meds, diet) for the day of surgery.
   b. Initiate post-op care expectations for patients and their families.
Health Conditions Indicating Need for an "In Person" Prescreening Visit:

A. General Health Status

- Inhibited ability to engage in normal daily activity
- Requires continuous monitoring at home
- Recent admission (2 months) for acute condition or exacerbation of a chronic condition
- Past or family history of problem with sedation or anesthesia
- Personal or family history of malignant hyperthermia
- Morbid obesity (BMI > 35)

B. Cardiovascular

- History of angina, coronary artery disease, myocardial infarction
- Symptomatic arrhythmias
- Poorly controlled hypertension (diastolic > 110; systolic > 160)
- History of congestive heart failure

C. Respiratory

- Asthma, COPD requiring medication or with acute exacerbation and progression within the past six months
- History of major airway surgery or unusual airway anatomy
- Upper or lower airway tumor or obstruction
- Home ventilatory assistance
- Morbid obesity (BMI > 35)

D. Endocrine

- Non-diet controlled diabetes (insulin or oral hypoglycemic agents)
- Adrenal disorders
- Active thyroid disease

E. Hepatic

- Any active hepatobiliary disease or compromise

F. Oncologic

- Currently undergoing course of chemotherapy
- Oncologic process with significant physiologic compromise

G. Neuromuscular

- History of seizure disorder or other significant central nervous system disease (multiple sclerosis, myasthenia gravis)
- History of myopathy or other muscle disorders
H. Musculoskeletal
- Kyphosis or scoliosis causing functional compromise
- Temporomandibular joint disorder
- Cervical or thoracic spine injury

I. Gastrointestinal
- Hiatal hernia
- Symptomatic gastroesophageal reflux

Anesthesia Testing Guidelines:
The studies below should be ordered when the patient presents with the following diseases prior to dental surgery performed under sedation. A minimum directed physical examination should include an assessment of the airway, lungs, and heart. Routine preoperative tests do not make an important contribution to the process of perioperative assessment and management of the patient by the sedation dentist. Specific tests and their timing should be individualized and based upon information obtained from sources such as the medical record, history, physical examination and type of planned procedure. The evaluation should be performed at least before the day of surgery. [http://www.surgerymanagement.com/presentations/pre-anesthesia-screening.php - top](http://www.surgerymanagement.com/presentations/pre-anesthesia-screening.php - top)

**Electrocardiogram**
- Age > 50
- Hypertension
- Hx Cardiac disease
- Hx Circulatory disease
- Diabetes mellitus & age > 40
- Renal, thyroid, metabolic disease
- High risk surgical procedure

**Chest x-ray**
- Asthma, COPD
- Severe cardiothoracic disease
- High risk surgical procedure

**Serum Chemistries**
- Renal disease
- Adrenal or thyroid disorders
- Diuretic therapy
- Chemotherapy
- High risk surgery

**Urinalysis**
- Diabetes mellitus
- Renal disease
- Recent GU infection
- Metabolic disorder
- High risk surgical procedure

**Complete Blood Count**
- Hematologic disorder
- Vascular procedure
- Chemotherapy
- High risk procedure

**Coagulation Studies**
- Hematologic disorder
- Anticoagulation therapy
- Vascular procedure
- High risk procedure
Current Health History:

<table>
<thead>
<tr>
<th>Medical History</th>
<th>Yes</th>
<th>Don't Know</th>
<th>Explain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you have heart problems?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have you had a heart attack?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you experience angina-like chest pain?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you have an irregular heartbeat?</td>
<td></td>
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<tr>
<td>Do you get short of breath when walking up 2 flights of stairs?</td>
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<td></td>
</tr>
<tr>
<td>Do you have any lung problems?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you have COPD? (Chronic obstructive lung disease)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you use oxygen?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you have sleep apnea?</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Have you taken Prednisone or other steroids for your breathing in the last 3 months?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you smoke?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have you had pneumonia or bronchitis in the past 6 months?</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Do you have any liver problems, or ever had jaundice?</td>
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<td></td>
</tr>
<tr>
<td>Do you have diabetes?</td>
<td></td>
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<tr>
<td>☐ Diet or ☐ Pill Controlled?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>☐ Insulin Dependent?</td>
<td></td>
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</tr>
<tr>
<td>Do you have seizures or convulsions?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recent hospitalization, surgery, or any serious illness?</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Are you seeing a specialist, or being treated by any other physician besides your primary care doctor?</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Do you have gastric reflux (GERD)?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you have allergies?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>☐ Food?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>☐ Latex? (rubber, sneakers, etc.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>☐ Environment?</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

History of Past Medical Conditions:

<table>
<thead>
<tr>
<th>Conditions: Check conditions you have or have had in the past.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>AIDS</td>
<td>Cancer</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>Chemical Dependency</td>
</tr>
<tr>
<td>Anemia</td>
<td>Diabetes</td>
</tr>
<tr>
<td>Appendicitis</td>
<td>Emphysema</td>
</tr>
<tr>
<td>Asthma</td>
<td>Epilepsy</td>
</tr>
<tr>
<td>Bleeding Disorders</td>
<td>Glaucoma</td>
</tr>
<tr>
<td>Blood Clots</td>
<td>Heart Disease</td>
</tr>
<tr>
<td>Bronchitis</td>
<td>Hepatitis</td>
</tr>
<tr>
<td>Bulimia</td>
<td>High Cholesterol</td>
</tr>
<tr>
<td>Eating Disorder</td>
<td>Urinary Tract Infection</td>
</tr>
<tr>
<td>HIV Positive</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Kidney Disease</td>
<td>Kidney Stones</td>
</tr>
<tr>
<td>Liver Disease</td>
<td>Migraine Headaches</td>
</tr>
<tr>
<td>Mononucleosis (Mono)</td>
<td>Multiple Sclerosis</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Pacemaker</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>Prostate Problem</td>
</tr>
<tr>
<td>Rheumatic Fever</td>
<td>Stroke</td>
</tr>
<tr>
<td>Stroke, Mini (TIA)</td>
<td>Suicide Attempt</td>
</tr>
<tr>
<td>Thyroid Problems</td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>Ulcers</td>
<td>Other</td>
</tr>
</tbody>
</table>
Current Medication History:

<table>
<thead>
<tr>
<th>Current Medications</th>
<th>Medication Allergies</th>
<th>Reaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Include Over-The-Counter and Herbal Supplements</td>
<td>Medication</td>
<td>Dose</td>
</tr>
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</tbody>
</table>

Where do you have your prescriptions filled? _______________________________________________

Surgical History:

Surgical History: Check all that apply

- No prior surgeries
- Angioplasty
- Appendectomy
- Arthroscopy
- Breast Biopsy
- Cardiac Bypass
- Cataract
- D & C
- Gallbladder

- Heart Cath
- Heart Valve Replaced
- Hemorroidectomy
- Hernia
- Hysterectomy
- Kidney Removal
- Mastectomy
- Pacemaker
- Implanted Defibrillator

- Prostate
- Spine (back/neck)
- Splenectomy
- Tonsils/Adenoids
- Total Hip
- Total Knee
- Tubal Ligation
- Other – please list

Sedation & Anesthesia History:

- Have you ever had a problem with Sedation or Anesthesia?
- Have you been told that you have a difficult airway?
- Have you ever had post sedation nausea / vomiting?
- Are you pregnant?
- Do any of your family members have a history of problems with anesthesia?
- Do you or any family members have a history of malignant hyperthermia?
EVALUATION FLOW CHART

☐ TABLE 1
Consider the patients ASA Status

☐ TABLE 2
Are there active cardiac conditions or red flags?

☐ TABLE 3
What is the risk of surgery?

☐ TABLE 4
What is the patient's functional capacity?

☐ TABLE 5
Consider major and minor clinical predictors.

TABLE 1
ASA PHYSICAL STATUS CLASSIFICATION

Physical Status PI *A normal healthy patient
Physical Status P2 *A patient with mild systemic disease
Physical Status P3 *A patient with severe systemic disease
Physical Status P4 *A patient with severe systemic disease that is a constant threat to life
Physical Status P5 *A moribund patient not expected to survive without the operation
Physical Status P6 *A declared brain-dead patient whose organs are being removed for donor purposes

TABLE 2
RED FLAGS = ACTIVE CARDIAC CONDITIONS

1. Unstable angina, recent MI (6 months), decompensated CHF, significant arrhythmias, severe valvular disease
2. Recent coronary artery angioplasty with stent
3. Uncharacterized or undocumented cardiac findings such as chest pain that has not been evaluated, murmur of unknown etiology, new EKG abnormalities, LBBB
TABLE 3
SURGICAL PROCEDURE TIMING
1. ELECTIVE: Cosmetic, chronic conditions such as amputation for degenerative joint disease
2. SEMI-URGENT: Cholecystectomy for chronic cholecystitis, laminectomy for spinal stenosis
3. URGENT: Mastectomy and prostatectomy for cancer, colectomy for recurrent diverticulitis, dialysis access
4. EMERGENT: Appendectomy, craniotomy for hemorrhage, aortic dissection

TABLE 4
What is the patient's functional capacity?
1 MET is defined as 3.5 mL O2 uptake/kg per min, which is the resting oxygen uptake in a sitting position). Various activity scales provide the clinician with a set of questions to determine a patient's functional capacity. Indicators of functional status include the following:

- Can take care of self, such as eat, dress, or use the toilet (1 MET)
- Can walk up a flight of steps or a hill or walk on level ground at 3 to 4 mph (4 METs)
- Can do heavy work around the house such as scrubbing floors or lifting or moving heavy furniture or climb two flights of stairs (between 4 and 10 METs).
- Can participate in strenuous sports such as swimming, singles tennis, football, basketball, and skiing (>10 METs)

TABLE 5
MAJOR AND MINOR CLINICAL PREDICTORS
MAJOR PREDICTORS
1. CAD, MI, EKG evidence of infarct or ischemia
2. Congestive heart failure
3. CVA, TIA
4. Diabetes mellitus
5. Renal insufficiency

MINOR PREDICTORS
1. Age >70
2. EKG abnormalities such as LVH, BBB, arrhythmias
3. Non-sinus rhythm or paced rhythm
4. Poorly controlled hypertension
5. Low functional capacity
6. 3 minors predictors are equal to 1 major predictor
Prescreening Triage Questionnaire:
Use this triage questionnaire to quickly determine if a patient needs pre sedation clearance from their primary care doctor or a pre screening appointment with the dentist/oral surgeon providing sedation.

1. Do you have heart problems (chest pain, heart attack, heart (coronary) stents, heart failure, valve problems, bypass surgery, irregular heartbeat, aneurysm) or a history of stroke? □ Y □ N
2. Do you have sleep apnea or breathing problems that require oxygen or steroid pills? □ Y □ N
3. Do you have a pacemaker or defibrillator? □ Y □ N
4. Do you have kidney failure requiring any type of dialysis? □ Y □ N
5. Do you take blood thinners other than Aspirin (i.e. Coumadin, Pradaxa, Plavix, Effient)? □ Y □ N Patients answering ‘YES’ to any of questions 1–5 require clearance from their primary care physician.
6. Do you have high blood pressure? □ Y □ N
7. Do you use insulin for diabetes? □ Y □ N
8. Do you find it difficult to climb a full flight of stairs without stopping to rest? □ Y □ N Patients answering ‘YES’ to two or more of questions 6–8 require a PAT Clinic appointment

Summary of Fasting (NPO) Guidelines to Minimize the Risk of Pulmonary Aspiration:

Ingested Material Minimum Fasting Period

- Clear Liquid - 2 hours
- Breast Milk - 6 to 8 hours
- Infant Formula - 6 to 8 hours
- Other liquids and solids - 6 to 8 hours

Airway evaluation:
Preoperative Cardiac Evaluation:

The following patients must have a preoperative cardiac evaluation by a cardiologist prior to all procedures, including endoscopy procedures:

- Patients who have undergone placement of a coronary artery stent in the last 12 months.
- Patients who are on Plavix for protection of a coronary stent placed at any time in the past.

If a patient with any of these problems does not have the appropriate preoperative evaluation and documentation, the procedure will be cancelled on the day of their dental surgery. Patients with a history of cardiovascular disease will need preoperative cardiac evaluation prior to most procedures. This includes history of the following:

- Coronary artery disease (including angioplasty, stents, or bypass surgery)
- Congestive Heart Failure
- Heart valve disease
- Arrhythmias (atrial fibrillation, supraventricular tachycardiac (SVT), Wolff-Parkinson-White)
- Stroke
UNDERSTANDING PERISURGICAL CARDIAC RISK

Many patients undergoing dental surgical sedation are at risk for a perioperative cardiovascular event. This includes dental surgical procedures without sedation. The risk is related to patient- and surgery-specific characteristics. Identification of increased risk provides the patient (and dentist / oral surgeon) with information that helps them better understand the benefit-to-risk ratio of a procedure and may lead to interventions that decrease risk. This topic will review the initial preoperative cardiac evaluation, which includes an attempt to quantify risk.

I. INCIDENCE AND MECHANISM

The incidence of an adverse cardiovascular outcome is related to the baseline risk. Patients with underlying cardiovascular disease, including peripheral artery disease or stroke, have an increased risk of perioperative cardiac complications compared to patients without extant atherosclerosis for two reasons:

1. They constitute a selected population with a high incidence of significant coronary artery disease. In addition, left ventricular systolic dysfunction (left ventricular ejection fraction ≤40 percent) is five times more common in patients with cerebrovascular disease or peripheral artery disease compared with matched controls.

2. Physiologic factors associated with surgery predisposed to myocardial ischemia, which is more pronounced in patients with underlying coronary disease. These include volume shifts and blood loss, enhanced myocardial oxygen demand from elevations in heart rate and blood pressure secondary to stress from surgery, and an increase in postoperative platelet reactivity.

II. OUR APPROACH

All patients scheduled to undergo dental surgery with sedation should have an assessment of the risk of a cardiovascular perioperative cardiac event. The purpose of this assessment is to help the patient and healthcare providers weigh the benefits and risks of the surgical sedation and optimize the timing of the surgery. On occasion, risk assessment will uncover undiagnosed problems or suboptimally treated prior conditions that need attention. The clinician uses information obtained from the history, physical examination, and type of surgery in order to develop an initial estimate of perioperative cardiac risk.

Risk models estimate the risk based on information obtained from the history, physical examination, electrocardiogram, and type of surgery. When assessing preoperative cardiac risk, we use either the revised cardiac risk index (RCRI), also referred to as the Lee index or the American College of Surgeons’ National Surgical Quality Improvement Program risk (ACS-NSQIP) model. The RCRI is simpler and has been widely used and validated over the past 15 years. The
ACS-NSQIP calculator is more complex and has yet to be validated in other populations. A simpler tool also derived from the NSQIP database is the Gupta MI or cardiac arrest (MICA) calculator. Practitioners should become familiar with one model and use it regularly.

These models provide the user with the risk of a cardiac complication in percent.

1. For patients at low risk (<1 percent), no further testing is indicated.
2. For patients at higher risk, caregivers need to ask the question whether further cardiovascular testing will change management and hopefully improve the outcome. In most cases, the reason to perform additional testing will be based not on desire to lower risk at the time of surgery, but rather lowering long-term risk. That is, the patient should have additional testing done irrespective of the need for surgery. There are few circumstances in which testing should be performed solely because the patient has upcoming surgery.

**Very high-risk patients** — Patients with recent myocardial infarction (MI) or unstable angina, decompensated heart failure (HF), high-grade arrhythmias, or hemodynamically important valvular heart disease (aortic stenosis in particular) are at very high risk for perioperative MI, HF, ventricular fibrillation or primary cardiac arrest, complete heart block, and cardiac death. All such patients should be optimally treated, with possible referral to a cardiologist for further evaluation and management.

**Emergent or urgent surgery** — Patients who require emergent or urgent surgery are at increased risk of a perioperative cardiovascular event at any level of baseline risk. In these cases, risk indices derived from elective surgery cohorts are not accurate, although they may provide an estimate of the minimal risk. (Fractures / Oral infections - abscess)

In many cases, there is not sufficient time for an extensive evaluation of the severity of a patient’s cardiovascular problem and in most cases the benefit of proceeding with surgery outweighs the risk of waiting to perform additional testing. However, these conditions often exclude sedation due to the patients NPO status. In the absence of pre-operative assessment because of the minimal time available before surgery, clinicians must be available postoperatively to help manage the possible cardiovascular complications in at-risk patients.

**III. INITIAL PREOPERATIVE EVALUATION**

Once a determination is made that dental surgical sedation will be considered, the patient should be evaluated for the risk of a cardiovascular complication. If this evaluation is unclear it can be verified by a primary care physician. The information obtained is used to assess risk. In patients assessed to be at increased (intermediate or high) cardiovascular risk, a referral to a cardiologist for further evaluation may be indicated.
At the time of the initial preoperative evaluation, the dentist should inquire about symptoms such as angina, dyspnea, syncope, and palpitations as well as a history of heart disease including ischemic, valvular, or myopathic disease, and a history of hypertension, diabetes, chronic kidney disease, and cerebrovascular or peripheral artery disease.

In addition, cardiac functional status should be determined. Functional status can be expressed in metabolic equivalents (1 MET is defined as 3.5 mL O2 uptake/kg per min, which is the resting oxygen uptake in a sitting position). Various activity scales provide the clinician with a set of questions to determine a patient's functional capacity. Indicators of functional status include the following:

- Can take care of self, such as eat, dress, or use the toilet (1 MET)
- Can walk up a flight of steps or a hill or walk on level ground at 3 to 4 mph (4 METs)
- Can do heavy work around the house such as scrubbing floors or lifting or moving heavy furniture or climb two flights of stairs (between 4 and 10 METs).
- Can participate in strenuous sports such as swimming, singles tennis, football, basketball, and skiing (>10 METs)

One important indicator of poor functional status and an increased risk of postoperative cardiopulmonary complications after major dental surgery under sedation is the inability to climb two flights of stairs or walk four blocks. However, in patients with limitations to walking such as orthopedic problems, it may be hard to assess cardiac functional status. One may use bicycle or arm ergometry stress testing to evaluate a patient’s functional capacity when the inability to walk limits testing.

The physical examination should focus on the cardiovascular system, and include blood pressure measurements, auscultation of the heart and lungs, abdominal palpation, and examination of the extremities for edema and vascular integrity. Important findings include evidence of heart failure or a murmur suspicious for hemodynamically significant valvular heart disease.

Similar to recommendations made in the 2014 American College of Cardiology/American Heart Association and European Society of Cardiology/European Society of Anesthesiology guidelines on noncardiac surgery, we obtain an electrocardiogram (ECG) in many patients with known cardiovascular disease, significant arrhythmia, or significant structural heart disease unless the patient is undergoing low-risk surgery. A preoperative ECG can be obtained in asymptomatic patients without known cardiovascular disease, but it is rarely helpful. Some ECG abnormalities seem to be associated with a worse prognosis in observational studies, but the association is inconsistent across studies. ECG abnormalities are not part of either the Revised
Cardiac Risk Index or the National Surgical Quality Improvement Plan because of the lack of prognostic specificity associated with these findings.

The rationale for obtaining a preoperative ECG comes from the utility of having a baseline ECG should a postoperative ECG be abnormal.

For those patients who receive a preoperative ECG, it should be evaluated for:

1. Q waves
2. Significant ST-segment elevation or depression, which raises the possibility of myocardial ischemia or infarction.
3. Left ventricular hypertrophy
4. QTc prolongation
5. Bundle-branch block
6. Arrhythmias

IV. RISK FACTORS USED IN RISK PREDICTION MODELS

The following clinical and surgery-specific factors are associated with an increase in perioperative risk of a cardiovascular event and are used in one or both of the models discussed below (Revised Cardiac Risk Index [RCRI] or MICA calculator derived from the National Surgical Quality Improvement Plan [NSQIP]). The newer ACS-NAQIP calculator includes 20 patient risk factors in addition to the surgical procedure.

a. Surgery specific risk (RCRI and NSQIP). The reported rate of cardiac death or nonfatal myocardial infarction is more than 5 percent in high-risk procedures, between 1 and 5 percent in intermediate-risk procedures, and less than 1 percent in low-risk procedures. Institutional and/or individual surgeon experience with the procedure may increase or lower the risk. Emergency surgery is associated with particularly high risk, as cardiac complications are two to five times more likely than with elective procedures.

b. History of ischemic heart disease (RCRI)
c. History of heart failure (RCRI)
d. History of cerebrovascular disease (RCRI)
e. Insulin dependent diabetes mellitus (RCRI)
f. Preoperative serum creatinine ≥2.0 mg/dL (RCRI) or >1.5 mg/dL (NSQIP)
g. Increasing age (NQSIIP)
h. American Society of Anesthesiologist class (NSQIP)
i. Preoperative functional status (NSQIP)
Other clinical predictors — While not included in the risk factors above, the following patient characteristics have been associated with increased risk:

- **Atrial fibrillation** – A retrospective, administrative database study demonstrated an association between a history of prior admission for atrial fibrillation (AF) and postoperative complications. The risk associated with AF was higher than that associated with a diagnosis of coronary artery disease.

- **Obesity** – Obese patients are at increased risk for adverse cardiovascular events at the time of noncardiac surgery. However, obesity has not been shown to be an independent predictor. The issue of whether the preoperative approach to obese patients should differ from that in the general population is uncertain. A 2009 scientific advisory on cardiovascular evaluation and management of severely obese patients undergoing surgery from the American Heart Association states that specific tests should be performed only if the results will change management.

V. ESTIMATING PERIOPERATIVE RISK

The history, physical examination, and 12-lead electrocardiogram identify patient-specific risk factors. This information, combined with the risk associated with the surgery, is used to estimate perioperative risk of adverse cardiac events. The risk will determine whether surgery should proceed without further cardiovascular testing, be postponed pending further testing such as stress testing, echocardiography, or 24-hour ambulatory monitoring, changing the planned surgery to a lesser risk procedure (if possible) or a non-surgical alternative (eg, palliative care), or cancelled so that a procedure such as coronary revascularization or heart valve replacement can take place. Multivariable analyses have identified combinations of factors that can be used to estimate perioperative risk [3]. Perioperative risk is expressed as the likelihood (in percent) that a patient will suffer a cardiac complication at the time of noncardiac surgery.

Models such as the Revised (Lee) cardiac risk index (RCRI) and the American College of Surgeons National Surgical Quality Improvement Program (NSQIP) risk prediction calculators (Gupta myocardial infarction/cardiac arrest [MICA] or Bilimoria ACS surgical risk calculator) have included patients with high-risk characteristics and who have been managed with more current standards of care. For these reasons, we suggest using one of these models. The MICA calculator outperformed the RCRI in some circumstances, and the newer ACS-NSQIP surgical risk calculator is more comprehensive and procedure specific. However, as mentioned previously, neither of these have yet to be validated externally. We do not recommend using older models such as the original
Goldman cardiac risk index, the Detsky modified risk index, or the Eagle criteria. Risk assessment should include information from the chosen scoring system with the inherent risk of the surgery.

**Gupta MICA NSQIP database risk model** — The NSQIP database was used to determine risk factors associated with intraoperative/postoperative myocardial infarction or cardiac arrest (MICA). Among over 200,000 patients who underwent surgery in 2007, 0.65 percent developed perioperative MICA. On multivariate logistic regression analysis, five factors were identified as predictors of MICA:

- Type of surgery
- Dependent functional status
- Abnormal creatinine
- American Society of Anesthesiologists’ class
- Increased age

A risk model was developed using these five factors and subsequently validated on a 2008 data set (n = 257,385). The risk model had a relatively high predictive accuracy (C statistic of 0.874) and outperformed the RCRI (C statistic of 0.747).

Please see the attached excel spreadsheet calculator.

<table>
<thead>
<tr>
<th>Percentile</th>
<th>Percent Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>25th percentile</td>
<td>0.05%</td>
</tr>
<tr>
<td>50th percentile</td>
<td>0.14%</td>
</tr>
<tr>
<td>75th percentile</td>
<td>0.61%</td>
</tr>
<tr>
<td>90th percentile</td>
<td>1.47%</td>
</tr>
<tr>
<td>95th percentile</td>
<td>2.60%</td>
</tr>
<tr>
<td>99th percentile</td>
<td>7.69%</td>
</tr>
</tbody>
</table>

**Revised cardiac risk index** — The RCRI, sometimes referred to as the Lee index, was published in 1999 and has been used worldwide since then. In the derivation of the index, 2893 patients (mean age 66) undergoing elective major noncardiac procedures were monitored for major cardiac complications (cardiac death, acute MI, pulmonary edema, ventricular fibrillation/cardiac arrest, and complete heart block). The index was validated in a cohort of 1422 similar individuals. The predictive value was significant in all types of elective major noncardiac surgery except for abdominal aortic aneurysm surgery.

A 2009 systematic review evaluated the ability of the RCRI to predict cardiac complications and mortality after major noncardiac surgery in various populations and settings. The RCRI performed moderately well in distinguishing patients at low compared to high risk for all types of noncardiac surgery, but was somewhat less accurate in patients undergoing only vascular noncardiac surgery. In addition, RCRI did not predict all-cause mortality well. However, this is expected,
as it does not capture risk factors for noncardiac causes of perioperative mortality and only one-third of perioperative deaths are due to cardiac causes.

The risk of major cardiac complications (cardiac death, nonfatal MI, nonfatal cardiac arrest, postoperative cardiogenic pulmonary edema, complete heart block) varied according to the number of risk factors. The following rates of adverse outcomes were seen in various studies:

- No risk factors – 0.4 percent
- One risk factor – 1.0 percent
- Two risk factors – 2.4 percent
- Three or more risk factors – 5.4 percent

There are several factors that probably contribute to the higher event rate in these two later studies:

- The original RCRI risk prediction model did not take all-cause mortality into account.
- In earlier studies, creatine kinase MB fraction was used to diagnose MI, rather than troponins, which are more sensitive.
- The type of myocardial infarction after surgery is changing. The incidence of a type 1, plaque rupture, MI is decreasing while type 2, hemodynamic MI, is increasing.

VI. MANAGEMENT BASED ON RISK

We use estimated risk to categorize patients into low- or higher-risk groups. **Low-risk patients** — Patients whose estimated risk of death is less than 1 percent are labeled as being low risk and require no additional cardiovascular testing. **Higher-risk patients** — Patients whose risk of death is 1 percent or higher may require additional cardiovascular evaluation. Often, these are patients with known or suspected coronary artery or valvular heart disease. Further evaluation may include stress testing, echocardiography, 24-hour ambulatory monitoring, or cardiologist consultation. We generally perform these tests if they are indicated for the patient even if they were not having surgery. There is little evidence that prophylactic intervention solely to improve outcomes during surgery is of benefit. Many studies of patients not at low risk have shown that performing some form of stress testing can further stratify the risk of an adverse perioperative event. However, no study has shown that interventions performed consequent to the results of the test improves outcomes. When we consider further cardiovascular evaluation for higher-risk patients, we use the approach suggested in the 2014 American College of Cardiology/American Heart Association guideline of perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery. In this approach, the patient’s functional capacity plays an important
role. In patients who can perform ≥4 METs of activity, we do not order additional tests. For those whose functional capacity is lower or unknown, additional testing may be indicated if it will influence perioperative care.

VII. FURTHER CARDIAC TESTING

In patients with known or suspected heart disease (ie cardiovascular disease, significant valvular heart disease, symptomatic arrhythmias), we perform further cardiac evaluation (echocardiography, stress testing, or 24-hour ambulatory monitoring) only if it is indicated in the absence of proposed surgery. There is no evidence that further diagnostic or prognostic evaluation improves surgical outcomes, although some of our experts routinely obtain preoperative stress imaging in patients who are scheduled for major vascular surgery. Preoperative cardiac evaluation and testing may differ for patients being evaluated for liver or kidney transplant.

For patients in whom a decision has been made to perform additional cardiovascular testing, its timing should be determined by the urgency of the clinical situation.

**Stress testing** — Stress testing is not indicated in the perioperative patient solely because of the surgery if there is no other indication.

Stress testing with exercise (with or without imaging) and pharmacologic stress testing with imaging have been well studied in patients scheduled to undergo noncardiac surgery. Although there is a clear relationship between the degree of myocardial ischemia found and prognosis, there is no evidence that prophylactic revascularization only to prevent ischemia at the time of surgery improves outcomes.

**Resting echocardiography** — Resting echocardiography is not indicated in the perioperative patient unless there is another indication, such as to evaluate valve function in patients with a murmur or left ventricular systolic function in patients with HF or dyspnea of unknown cause.

The presence of significant left ventricular systolic dysfunction or severe valvular heart disease is associated with a worse outcome, particularly postoperative heart failure, at the time of noncardiac surgery.

**24-hour ambulatory monitoring** — As with echocardiography and stress testing, we do not recommend 24-hour ambulatory monitoring for perioperative diagnostic or prognostic purposes if it is not otherwise indicated. Its use has not been shown to improve outcomes in this setting. The indications for 24-hour ambulatory monitoring are discussed elsewhere and are primarily for patients with syncope or significant bradycardia or tachycardia if not previously evaluated.

**Troponin and BNP** — The potential role of troponin and brain natriuretic peptide testing in perioperative risk stratification is discussed elsewhere.
VIII. SUMMARY AND RECOMMENDATIONS:

1. All patients scheduled to undergo dental surgical sedation should have an assessment of the risk of a cardiovascular perioperative cardiac event.

2. Identification of risk factors is derived from the history, physical examination, and type of proposed surgery.

3. We use the Gupta MICA NSQIP database risk model to establish the patient’s risk.

4. We obtain an electrocardiogram in patients with cardiac disease in large part to have a baseline available should a postoperative test be abnormal.

5. For patients with known or suspected heart disease (ie, cardiovascular disease, significant valvular heart disease, symptomatic arrhythmias), we only perform further cardiac evaluation (echocardiography, stress testing, or 24-hour ambulatory monitoring) if it is indicated in the absence of proposed surgery.
## DISCHARGE CRITERIA

- A & O x 3
- Respiratory Profile
- Cardiovascular Profile
- Ambulatory Status
- Pain Scale
- Caregiver
- Doctors Exam
- Wheelchair: All patient must be screened for the following criteria prior to going home if they are exposed to **ANY Dental Sedation**.

All patient must be screened for the following criteria prior to going home if they are exposed to **ANY Dental Sedation**.

1. A & O x 3: This means the patient is alert to person place and time.
2. The patient's respiratory rate and oxygen saturation is checked on room air.
3. The blood pressure and HR are documented after sedation.
4. A walk test is preformed. How can they get from their car to the house safely if they can't walk in your office?
5. Their pain scale should be at 5 or lower. (1/10)
6. They leave the office in a wheelchair to get to their car.
7. Their transportation and caregiver is present to take them home.
8. The dentist examines them prior to leaving.

### Discharge Instructions:

1. Have a caregiver to stay with you for the next 24 hours until the effects of the sedation have worn off.
2. Don't perform any hazardous activities. Do not drive or operate heavy machinery for 24 hours.
3. Do not take over responsible tasks or take important decisions for at least 24 hours until you are fully mentally alert.
4. If you have any unusual symptoms or feel discomfort, call your dentist. If unable to contact your sedation dentist, call the closest hospital emergency room.
5. Only take pain medication as prescribed by the dentist's directions.
6. You may begin taking you pain medicine at _________ on _______.
7. Only take your pain medicine if you can preform all of the following of your own accord:
   - Read the instructions on the medicine container
   - Remove the lid from the medicine container
   - Identify the pain pill
   - Take the pain pill with a sip of water
Sedation Consent Form

This form is intended to document the discussion we have had regarding your planned conscious sedation procedure. The medications we use are typically sedatives and analgesics. These medications can greatly minimize anxiety that may be associated with going to the dentist. Their use in the office setting induces a relaxed state that progresses to a sedated state. You will still be able to communicate with the dentist while treatment is being performed. This level of sedation is called “conscious sedation.” Benefits of conscious sedation include reduced awareness of unpleasant sights, sounds and sensations associated with the procedure along with reduced anxiety. Most patients fall asleep, but not always. Complete recall of the procedure is a possibility but not common. Even though conscious sedation is safe, effective and wears off rapidly after the dental visit, you should be aware of some important precautions and considerations. Risks of conscious sedations include:

- Nausea/Vomiting
- Allergy to medication
- Irritation and/or pain/swelling to skin and veins (IV only)
- Prolonged sedation
- Amnesia
- Breathing problems, brain damage, cardiac arrest and death
- Intubation (Securing the airway emergently)
- Transfer to an emergency facility
- Organ system failure

The risks of dental sedation have been explained to me by Dr. NAME. I have read, reviewed, and understand the above general risk profile of dental sedation.

(please initial)____________

MEDICAL HISTORY

I understand that it is critically important that I fully discuss my complete medical history and current medications with the dentist before sedative medications are administered. Tell the doctor if you have the following medical conditions:

A. You should not use sedatives or analgesics if you are PREGNANT or BREAST FEEDING.
   (please initial)____________

B. You should not be exposed to office based sedation if you have significant:
   - Cardiovascular disease
   - Respiratory disease
   - Neurologic disease
   - Gastrointestinal and/or Liver disease
   - Kidney disease

C. I have informed the dentist of all of my medical conditions. (please initial)____________

CURRENT MEDICATION LIST

Tell the doctor if you are taking the following medications as they can adversely interact with the sedation medications:

- nefazodone (Serzone)
- cimetidine (Tagamet, Tagamet HB, Novocimetine or Peptol)
- levadopa (Dopar or Larodopa) for Parkinson’s disease
- antihistamines (such as benadryl and travist)
- verapamil (Calan)
- diltiazem (Cardizem)
- erythromycin and the azole antimycotics (nizoral, biaxin, orporanox)
- HIV drugs indinavir and nelfinovir
- alcohol
- Grapefruit juice should also be avoided
- Taking recreational/illicit drugs can also cause untold reactions.

I have informed the dentist of all of my medications. (please initial)____________
The dentist has reviewed the written instructions with me including expectations regarding:

- Food/drink intake - I have had nothing to eat or drink in the previous 6 hour time period (please initial)____________

- Escort - The following escort will drive me to and from the facility. (please initial)____________

  ESCORT NAME:__________ PHONE NUMBER:_________

- Activity after the sedation - The following caretaker will stay with me at all times for a minimum of 24 hours after I am discharged from the facility.

  CAREGIVER NAME:__________ PHONE NUMBER:_________

Sedation can be administered by multiple routes. Dr. ________ has discussed these options with me. I also understand that the sedation plan may need to be changed on the day of the procedure. I acknowledge that no guarantee has been made as to the results that may be obtained. During the discussion, I have had my questions answered to my satisfaction.

I, _____________________________, request and authorize Dr. NAME to administer oral conscious sedation medications and/or nitrous oxide/oxygen to me in conjunction with the planned dental procedure. The dental procedure is: ___________________

The reason I am asking for these medications is: ____________________________________________________________

Patient/Guardian: _____________________________ Date__________

Witness: _____________________________ Date__________

Doctor: _____________________________ Date__________
## SEDATION FLOW SHEET

<table>
<thead>
<tr>
<th>Time</th>
<th>Medication</th>
<th>DOSE mcg</th>
<th>Indication</th>
<th>Route</th>
<th>Sats</th>
<th>RR</th>
<th>BP SBP/DBP</th>
<th>HR</th>
<th>Sedation Depth</th>
</tr>
</thead>
<tbody>
<tr>
<td>START</td>
<td>Halcion</td>
<td>□ Sed</td>
<td>□ Pain</td>
<td>PO</td>
<td>%</td>
<td>n/a</td>
<td>Min</td>
<td>Mod</td>
<td>Deep</td>
</tr>
<tr>
<td></td>
<td>Hydrocodone/APAP</td>
<td>□ Sed</td>
<td>□ Pain</td>
<td>PO</td>
<td>%</td>
<td>n/a</td>
<td>Min</td>
<td>Mod</td>
<td>Deep</td>
</tr>
<tr>
<td></td>
<td>Halcion</td>
<td>□ Sed</td>
<td>□ Pain</td>
<td>SL</td>
<td>%</td>
<td>n/a</td>
<td>Min</td>
<td>Mod</td>
<td>Deep</td>
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<tr>
<td></td>
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<td>□ Sed</td>
<td>□ Pain</td>
<td>SL</td>
<td>%</td>
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<tr>
<td>END</td>
<td></td>
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END OF CASE MEDICATION TOTALS

<table>
<thead>
<tr>
<th>Medication</th>
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<th>Waste Dose</th>
<th>Dentist Signature</th>
<th>Witness Signature</th>
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<td>Halcion</td>
<td>mg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydrocodone/APAP</td>
<td>mg</td>
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PRE SEDATION NOTE

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<tr>
<th>Note:</th>
<th>NPO &gt; 6 hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>PMH reviewed with patient. MD preop (if required) obtained &amp; placed in chart</td>
<td>Completed, Comments:</td>
</tr>
<tr>
<td>Med List Reviewed with patient Placed in chart</td>
<td>Completed, Comments:</td>
</tr>
<tr>
<td>Physical Exam Preformed</td>
<td>Heart: □ rrr no m/r/g</td>
</tr>
<tr>
<td>Lungs: □ Clear</td>
<td>CNS: □ wnl</td>
</tr>
<tr>
<td>GI: □</td>
<td>MS: □</td>
</tr>
<tr>
<td>Skin: □ wnl</td>
<td></td>
</tr>
<tr>
<td>Airway Exam Preformed</td>
<td>Mal: □ I □ II □ III</td>
</tr>
<tr>
<td>Consistent Reviewed with pt and signed</td>
<td>H&amp;N: □ wnl</td>
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<td>24hr Caregiver Confirmed / contact info</td>
<td>Note:</td>
</tr>
<tr>
<td>Escort Confirmed / contact info</td>
<td>Note:</td>
</tr>
<tr>
<td>Studies Reviewed</td>
<td>Note: reviewed</td>
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DISCHARGE

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<tr>
<th>CNS A&amp;Ox3</th>
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<th>CV Profile BP/HR</th>
<th>Walk Test Direct Observation</th>
<th>Pain Scale (1 / 10)</th>
<th>Caregiver/Escort/Wheelchair/Seatbelt</th>
<th>Dentist d/c Exam Signature</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ A&amp;Ox3 comments:</td>
<td>SATS: □</td>
<td>BP: □</td>
<td>HR: □</td>
<td></td>
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</tr>
</tbody>
</table>

A&Ox3=alert to person, place, time  SATS=oxygen saturation  RR=respiratory rate  BP= blood pressure  
pain scale=1 is least, 10 is severe  NPO=nothing per oral  Mal=class of airway  
rrr no m/r/g=regular rate rhythm no murmurs, rubs, gallops  mg=milligrams  
mcg= micrograms

¯ RMS © 68
**INVENTORY NARCOTIC LOG**

<table>
<thead>
<tr>
<th>CONTROLLED SUBSTANCE NAME</th>
<th>Fentanyl</th>
</tr>
</thead>
<tbody>
<tr>
<td>UNIT DOSAGE FROM VENDOR</td>
<td>50 mcg/cc</td>
</tr>
<tr>
<td>(Form of narcotic that exist in each vial)</td>
<td></td>
</tr>
<tr>
<td>NUMBER OF VIALS IN EACH COMERCIAL CONTAINER</td>
<td>5</td>
</tr>
<tr>
<td>NUMBER OF COMERCIAL CONTAINERS</td>
<td>1</td>
</tr>
<tr>
<td>VENDOR NAME</td>
<td>SAS</td>
</tr>
<tr>
<td>DATE RECEIVED / INVOICE NUMBER</td>
<td>1/1/2015  12345</td>
</tr>
<tr>
<td>DAILY INVENTORY</td>
<td>End of Case + Close of Business</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DATE</th>
<th>PT NAME (Last, First)</th>
<th>DOB</th>
<th>CASE</th>
<th>STARTING AMOUNT (#vials)</th>
<th>AMOUNT USED (#vials /volume)</th>
<th>AMOUNT WASTE (#volume)</th>
<th>AMOUNT REMAINING (#vials)</th>
<th>DENTISIT Printed Name/ Signature</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>

**INSTRUCTIONS**

1. Each Inventory Narcotic Log Sheet is filled out for each Type of controlled substance used.
2. The Inventory Narcotic Log Sheet must match the Sedation Flow Sheet.
3. The Sedation Flow Sheet must have the following identifying information at minimum:
   - Last Name
   - First Name
   - DOB
   - Case
   - Date of Case
   - Start Time
   - End Time
   - Indication for Sedation

4. The Pill/Tablet must have the following documented on the sedation flow sheet:
   a. Name of controlled substance as documented on the controlled substance log
   b. The dose of the controlled substance
   c. The time that the individual drug & dose was given
   d. The route that the individual drug & dose was given
   e. The indication for the use of the controlled substance
   f. Every time a new drug & dose is given, the documentation of the above information (a-e) needs to be repeated
   g. Each type of controlled substance needs to be documented individually with the above process (a-f)
   h. The process of a-g is to be documented on a single sedation flow sheet specific for the patient and for the case performed
   i. Document the total mcg or mg dose given for each controlled substance at the end of the case
   j. Place this information on the Inventory Narcotic Log Sheet at the end of each case
DENTAL OFFICE EMERGENCIES

AIRWAY TREATMENT SUMMARY

**Allergic Reaction**
- Mild (no hypotension or airway compromise)
  
  **Benadryl:**
  - Adult: 25 - 50mg IV or IM
  - Pediatric: 1 - 2mg/kg Max dose 50mg

  **Albuterol:** 2.5mg/3cc NSS via powered nebulizer

- Anaphylactic Reaction
  
  1) High flow oxygen
  2) Large bore IV NSS
  3) Epinephrine 1:1,000 – 0.3mg – 0.5mg IM repeat q 10 minutes
  4) IF IV: Epinephrine 1:10,000 – 0.3 – 0.5mg IV q 5 minutes, Pediatric dose 0.01mg/kg not to exceed 0.5 mg
  5) Benadryl (minimum of 50mg on hand)
     a. Adult dose: 25mg – 50mg slow IV push
     b. Pediatric dose: 1mg – 2mg/kg max 50mg.
  6) Glucagon 1mg – 2mg IV if the patient is taking beta or calcium channel blockers and does not respond to the epinephrine.

**Bronchospasm**
- Albuterol MDI – Bronchodilator B2 agonist
  - 4-8 puffs adult dose

- Albuterol 2.5mg/3cc saline vial O₂ powered nebulizer mask.

- Epinephrine
  - Adult dose: 0.3mg IM-0.5mg IM may repeat x 1 in 5-10 min

- If severe Epinephrine
  - Adult dose: 1cc of 1:10,000 IV may repeat in 5 minutes total dose of 5cc.

**Difficult Airway**
1. Head tilt/chin lift
2. Reposition/pull tongue forward
3. Bag Valve Mask
4. Nasal/Oral airway
5. LMA placement
6. Intubation (only if highly trained and proficient in skill)
7. Tracheostomy (only if highly trained and proficient in skill)
**Emesis and Aspiration**
1. Place the patient head down, feet up, roll onto right side.
2. Clear any vomitus in airway with large bore suction.
3. Oxygenate and ventilate as needed.
4. Treat bronchospasm as needed.

**Laryngospasm**
1. Positive pressure ventilation
2. Lidocaine 1-2 mg/kg IV
3. Propofol 0.5 mg/kg IV increments ...... or / if no response
4. Succinylcholine 10 – 40 mg IV

**Seizure**
1. Support the patients airway
2. Monitor cardiovascular vital signs
3. If the seizure does not self resolve in 3 minutes or the patient becomes hypoxic, administer midazolam 5 mg IV x 1 with airway support

**CARDIOVASCULAR TREATMENT SUMMARY**

**Hypertension:** If severe hypertension – Diastolic pressure >120 or Systolic pressure >200
- **Ntg.** 0.4mg SL q 5 minutes or **Esmolol (Brevibloc)** – antihypertensive, shortest acting beta blocker (approx. 30 minutes), B1 Selective, supplied: 10mg/ml (10ml sdv) Dose: *(two ways to administer)* 80mg (approx. 1mg/kg) over 30 seconds or 0.15mg/kg over 1 minute may repeat 0.15mg/kg in 10 minutes, max .30 mg/kg/min

**Hypotension**
1) The easiest, safest and usually most effective way to treat hypotension is with a fluid bolus.
2) Ephedrine: Vasopressor, Supplied: 50mg/ml minimum on hand (2)
   - Dose: Dilute 50mg in 9cc NSS NSS (5mg/ml), 2.5mg – 5mg slow IV push, Repeat until BP is stabilized or 25 mg IM x 1. Effects of drug within 1 minute, peaks at 15 minutes, lasts approx. 1 hour. Drug of choice when you need to increase blood pressure and heart rate.
3) Phenylephrine: Vasopressor, Supplied 10mg/ml *(minimum to keep on hand (2)*, Dilute in 9ml of NSS solution, Discard 9ml and dilute with additional 9ml of saline. Dose: with the 0.1mg.ml concentration at 0.1mg/ml increments and repeat for effect. Effects seen in 1 minute, lasts for approximately 20 minutes.
**Chest Pain / MI / Heart Attack**

- Supplemental oxygen 4 – 15 liters depending on pulse ox and physical findings
- Aspirin: Administer to all patients with ACS unless hypersensitive to ASA, Blocks formation of Thromboxane A2, Reduces overall ACS mortality, Dose: Oral 81mg ASA
- Nitroglycerin, Used to treat pain in suspected cardiac patients, Dose: 0.4 mg tablet or pre-metered dose spray. May be repeated q 5min. as long as BP >90 systolic, Heart rate should be >50 and <100. Nitroglycerine is contraindicated if patient has taken Levitra, Cialis, Viagra, and Revatio within the past 36 hours.

**Narrow Complex Tachycardia** and heart rates >150 and symptomatic (regular rhythm)

- Adenosine (minimum of 30mg on hand), First drug of choice for stable narrow complex tachycardia, Effective in terminating those rhythms due to re-entry involving the SA or AV node, **Does not convert A-Fib, A-Flutter or, V-TACH**
- Adult dose: 6mg rapid IV push may repeat 12mg, Side effects flushing, chest pain, Asystole.
- Pediatric dose: 0.1mg/kg First dose.
  - Max increment dose 6mg.
  - May repeat 0.2mg/kg x 2
  - Max total dose 12mg.

**Wide Complex Tachycardia** life threatening arrhythmia such pulseless V-TACH, V-FIB

- Call 911 and verify
- High quality CPR and place AED
- Shock -> check pulse, Shock -> check pulse
- Resume CPR and give Epinephrine 1 mg IV push (is still acceptable)

**Narrow Complex Tachycardia** Irregular Rhythm with Symptoms. (Atrial Fibrillation)

- Verapamil (minimum on hand 4), Supplied 5mg/2ml, May control ventricular response in A-Fib, A-Flutter, or multifocal atrial tachycardia, It is an alternative to treating SVT. Precautions: Do not use for Wide Complex Tachycardia
- Dose 2.5mg – 5mg slow IV push over 2 minutes, Repeat dose 5-10mg, Total dose 20mg. Do not give if hypotensive
**Bradycardia**
- Heart rate < than 60 that are symptomatic with hypotension
  - IV 250-300cc fluid bolus
  - Oxygen
  - Position
- If still Symptomatic:
  - Atropine (minimum 3mg on hand), Supplied 1mg prefilled syringes, Not effective for AV blocks, Precautions in presence of MI and Hypoxia due to the increased oxygen demand, Dose: 0.5mg Atropine q 5 minutes, maximum dose 3mg
- **2010 guidelines, recommends chronotropic drug infusion as an alternative to pacing.**
- **1mg epinephrine 1:10,000/250cc D5W or NSS and titrate to effect.**

**Cardiac Arrest:** PEA/Asystole
- Adult: Epinephrine 1mg 1:10,000 repeat every 3-5 minutes
  - **2010 guidelines is de-emphasizing the use of atropine**
## ADULT REVERSAL FOR BENZODIAZEPINES - Halcion, Midazolam

### FLUMAZENE (ROMAZICON)

<table>
<thead>
<tr>
<th>VIAL</th>
<th>CONCENTRATION</th>
<th>DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.1 mg/cc</td>
<td>.3 mg SQ/IM/IV every 5 min</td>
</tr>
</tbody>
</table>

Titrating to respiratory and airway stability

---

**Draw up 3cc and inject it SL**

### ADULT REVERSAL FOR OPIOIDS - Hydrocodone, Oxycodone

### NARCAN (NALOXONE)

<table>
<thead>
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<th>VIAL</th>
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<tbody>
<tr>
<td></td>
<td>.4 mg/cc</td>
<td>.2 mg SQ/IM/IV every 5 min</td>
</tr>
</tbody>
</table>

Dilution:

.4 mg in 10cc dilutes Narcan to .04 mg/cc

Titrating to respiratory and airway stability

---

**Draw up 1/2cc and inject it SL**
## PEDIATRIC REVERSAL FOR BENZODIAZEPINES - Halcion, Midazolam

**FLUMAZENILE (ROMAZICON)**
(Pediatric dosing is .01mg/kg IM not to exceed a total maximum 0.05 mg/kg)

<table>
<thead>
<tr>
<th>VIAL</th>
<th>CONCENTRATION</th>
<th>DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.1 mg/cc</td>
<td>.2 to .4 mg SQ/IM/IV every 5 min</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Titrate to respiratory and airway stability</td>
</tr>
</tbody>
</table>

**Draw up 2 to 4cc and inject it SL**

## PEDIATRIC REVERSAL FOR OPIOIDS - Hydrocodone, Oxycodone

**NARCAN (NALOXONE)**

<table>
<thead>
<tr>
<th>VIAL</th>
<th>CONCENTRATION</th>
<th>DOSE</th>
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<tr>
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<td>.4 mg/cc</td>
<td>.4 mg SQ/IM/IV every 5 min</td>
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<tr>
<td></td>
<td>DILUTION</td>
<td>Titrate to respiratory and airway stability</td>
</tr>
<tr>
<td></td>
<td>.4 mg in 10cc dilutes Narcan to .04 mg/cc</td>
<td></td>
</tr>
</tbody>
</table>

**Draw up 1cc and inject it SL**
LARYNGOSPASM: TREATMENT
1. Positive pressure ventilation
2. Propofol 0.5 mg/kg IV increments
3. Succinylcholine 10 – 40 mg IV
4. Lidocaine 1-2 mg/kg IV

BRONCHOSPASM: TREATMENT
1. Albuterol MDI – 4-8 puffs adult dose
2. Epinephrine - Adult dose 0.3 - 0.5 mg IM
3. Epinephrine - Adult dose 0.1 - 0.25 mg IV

Emesis and Aspiration
1. Place the patient head down, feet up, roll onto right side.
2. Clear any vomitus in airway with large bore suction.
3. Oxygenate and ventilate as needed.
4. Treat bronchospasm as needed.

ANAPHYLAXIS: TREATMENT
1. High flow oxygen + Alb MDI
2. Large bore IV NSS
3. Epinephrine: 0.3mg – 0.5mg IM repeat q 10 minutes
4. Epinephrine: 0.1 – 0.25mg IV q 5 minutes
5. Benadryl: Adult dose 25mg – 50mg slow IV push

Difficult Airway
1. Head tilt / chin lift
2. Reposition / pull tongue forward
3. Bag Valve Mask
4. Oral airway
5. LMA placement
6. Intubation (only if highly trained and proficient in skill)
7. Tracheostomy (only if highly trained and proficient in skill)
EXAMPLE: e - cylinder
EXAMPLE: O2 remaining in e-cylinder

A portable e-cylinder O2 tank has 500 psi left in it. At 6 l/min how long do you have to provide O2 at that flow rate?

At 500 psi with a flow rate of 6 liters per min, there are 28 minutes of oxygen left.

Formula:

<table>
<thead>
<tr>
<th>e-cylinder volume (L)</th>
<th>Remaining Volume (L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Service Pressure (psi)</td>
<td>Gauge Pressure (psi)</td>
</tr>
</tbody>
</table>

1. The service pressure for an e-cylinder carrying oxygen is 1900 psi.
2. The volume of oxygen in an e-cylinder is 660 liters.

Now entering the values listed above:

\[
\frac{660 \text{ L}}{1900 \text{ psi}} = \text{remaining contents (in L) / current gauge pressure (in psi)}
\]

I find it easier to look at this formula this way:

\[
0.35 \times \text{psi on gauge} \quad \frac{\text{L/min to be delivered}}{}
\]

The 0.35 comes from dividing 660 by 1900. In the example above:

\[
\frac{0.35 \times 500 \text{ psi}}{6 \text{ L/min}} = 28 \text{ min}
\]