MC905-A

MODERATE SEDATION/DEEP SEDATION

PROVIDER COURSE

A SELF-DIRECTED LEARNING MODULE

MODERATE SEDATION/DEEP SEDATION PROVIDER LEARNING MODULE

INTRODUCTION:

This learning material is prepared to assist in educating and evaluating non-anesthesiology physicians, dentists, RNs, and PAs who will be administering and monitoring moderate or deep sedation.

OBJECTIVES:

1. To define minimal, moderate, and deep sedation.
2. To learn the Medical Center policy for moderate/deep sedation.
3. To review the pre-moderate sedation assessment of patients.
4. To learn the pre-, intra-, and post-moderate sedation monitoring of the patient.
5. To reduce the risks to patients receiving medications for moderate sedation.
6. To standardize the monitoring and care of patients receiving moderate sedation.
7. To identify dosages, actions, and complications of medications used during moderate sedation.
8. To discuss the medications used for the reversal of opioids and benzodiazepines.
9. To review airway maneuvers and the use of airway adjuncts used in the management of airway obstruction.
10. To describe common complications associated with of moderate sedation.
11. To learn the patient's physical status classification as defined by the American Society of Anesthesiologists.

DEFINITIONS:

Minimal Sedation: (Not addressed by this policy) A drug induced state during which patient respond normally to verbal commands. Although cognitive function and coordination may be impaired, ventilatory and cardiovascular functions are unaffected.

Moderate Sedation: A drug induced depression of consciousness during which patients respond ***purposefully*** to verbal commands, either alone or accompanied by light tactile stimulation. NO intervention is required to maintain a patent airway, and spontaneous ventilation is adequate. Protective reflexes (coughing, gag and/or corneal reflexes) are maintained. Cardiovascular function is usually maintained.

Deep Sedation: A drug induced depression of consciousness during which patients cannot be easily aroused or 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. (While deep sedation may be an undesired effect when attempting moderate sedation, there are situations whereby deep sedation is a state that may be preferable).

Anesthesia: (Not addressed by this policy) Consists of general anesthesia and spinal or major regional anesthesia and does not include local anesthesia. General anesthesia is a drug-induced loss of consciousness during which patients are not arousable even by painful stimuli. The ability to independently maintain ventilatory function is often impaired.

PURPOSE:

Moderate or deep sedation will be used to minimize patient's discomfort, anxiety and/or pain during diagnostic and therapeutic procedures. Moderate or deep sedation will be used to reduce risks and complications that are associated with the use of general anesthesia.

GOALS OF MODERATE SEDATION:

1. Maintain consciousness and patient cooperation
2. Provide sedation and/or relief of anxiety
3. Provide pain control
4. Achieve control of patient's physiologic parameters

CHARACTERISTICS OF PATIENTS UNDER MODERATE SEDATION:

1. Patient is cooperative.
2. Patient is conscious.
3. Anxiety is controlled.
4. Amnesia may be present.
5. Vital signs are stable.
6. Protective reflexes are active and intact without reasonable expectation of loss of airway reflexes.
7. The risk of complications if reasonably low.
8. None to infrequent post-sedation complications.

GOALS OF DEEP SEDATION:

1. Minimize or prevent patient movement.
2. Provide sedation and/or relief of anxiety.
3. Provide pain control.
4. Achieve control of patient's physiologic parameters.

CHARACTERISTICS OF PATIENTS UNDER DEEP SEDATION:

1. Patient may not be conscious.
2. Patient is responsive to painful stimuli only.
3. Anxiety is controlled.
4. Amnesia may be present.
5. Vital signs are stable.

6. Protective reflexes may be compromised.
7. The risk of complications if reasonably low.
8. None to infrequent post-sedation complications.

PATIENTS AT INCREASED RISK FOR DEVELOPING COMPLICATIONS DURING MODERATE OR DEEP SEDATION:

1. Uncooperative patients
2. Patients at extreme of age (pediatric and geriatric patients)
3. Patients with severe cardiac, pulmonary, hepatic, renal or central nervous system disease
4. Morbidly obese patients
5. Patients with Obstructive Sleep Apnea (OSA)
6. Pregnant patients
7. Patients with a history of drug and/or alcohol abuse

PRE-MODERATE/DEEP SEDATION ASSESSMENT:

The non-anesthesiology physicians or dentists will perform an appropriate patient assessment prior to the administration of moderate sedation and must include the following:

1. An informed consent explaining the risks, benefits, and alternatives to the procedure and/or the administration of moderate/deep sedation.
2. Relevant history and review of system with emphasis to cardiac and pulmonary system. History should include previous adverse reactions to anesthesia and/or sedation.
3. Airway assessment including examination of the patient's airway, recognition of high-risk airways and documentation of the Mallampati classification.
4. Ensuring and assessment of a patient's NPO (fasting) status.
5. Assessment of the patient's eligibility for sedation using the American Society of Anesthesiologist's (ASA) physical status classification.
6. Procedure plan with choice of moderate or deep sedation and medications to be utilized.
7. Pain assessment

A. AIRWAY ASSESSMENT

An important aspect in providing moderate or deep sedation is the ability to rescue a patient and maintain an airway for proper ventilation and oxygenation. Positive pressure ventilation, with or without endotracheal intubation, may be necessary if respiratory compromise develops during moderate or deep sedation. This may be more difficult in patients with atypical airway anatomy. Also, some airway abnormalities may increase the likelihood of airway obstruction during spontaneous ventilation.

Taking an adequate history is necessary to anticipate such possible complications. With regards to airway management, the history should focus on prior intubations, anesthetic

history, drug allergies, and confounding illnesses that may hinder airway access. Factors that may be associated with difficulty in airway management include:

1. History of stridor, snoring or obstructive sleep apnea
2. History of difficult intubation
3. History of cervical spine disorder:
    a. Advanced rheumatoid arthritis
    b. Cervical spine immobility
4. Presence of a chromosome abnormality like Trisomy 21 Downs's Syndrome

Physical examination of the patient may reveal other factors that also may hinder appropriate airway management. They include:

1. Significant obesity (body mass index > 35)
2. Presence of excessive facial hair
3. Presence of a receding chin, small mouth opening, short neck
4. Protuberant incisors
5. Multiple dental caries and lose teeth

Other areas of examination that may indicate a high-risk airway include:

1. **Head and Neck:** Short neck, limited neck extension, decreased thyromental distance (< 3 cm in an adult), neck mass, cervical spine disease or trauma, tracheal deviation, and dysmorphic facial features (e.g., Pierre-Robin syndrome)

2. **Mouth:** Small opening (< 3 cm in an adult); protruding incisors; loose or capped teeth; presence of dental appliances; high arched palate; microglossia; tonsillar hypertrophy; nonvisible uvula

3. **Jaw:** Micrognathia, retrognathia, trismus, or significant malocclusion

The examination of the airway involves inspection and evaluation of:

1. Oral cavity (identification of loose, chipped, or capped teeth, presence of dentures)
2. Temporomandibular joint - with particular attention to mouth opening.
3. Thyromental distance - the distance between the prominence of the thyroid cartilage and the bony point of the lower mandibular border should be more than 6 cm. A distance less than 6 cm may indicate that the patient may be difficult to intubate should the need arise during an airway emergency.
4. Range of motion of neck
5. Mallampati airway classification

MALLAMPATI CLASSIFICATION

The Mallampati airway classification attempts to grade the degree of difficulty of endotracheal intubation from grade I to IV. The examination is conducted with the patient

in a sitting position. The patient's head is maintained in a neutral position and the mouth is opened as wide as possible. The patient is encouraged NOT to phonate during the examination. Classification is based on a description of the anatomic area visualized. (See Figure Below)

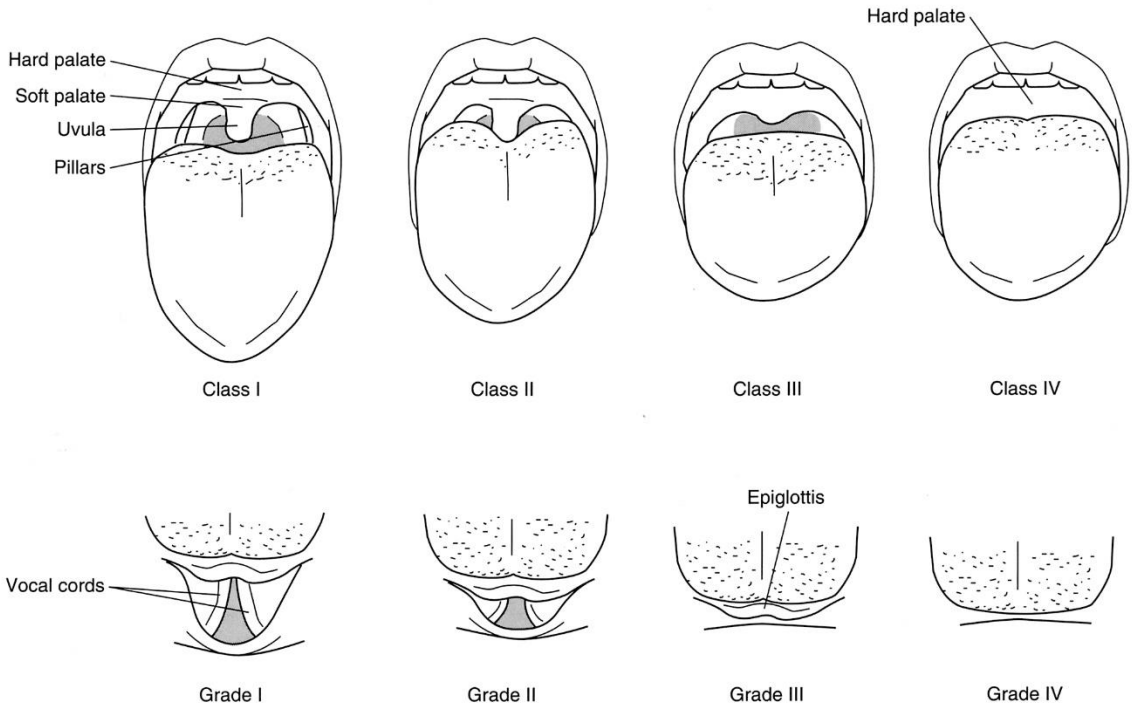
Class I: Tonsillar pillars, soft palate and the entire uvula are easily visualized

Class II: More than the base of the uvula is visualized, along with soft palate but not the tonsillar pillars

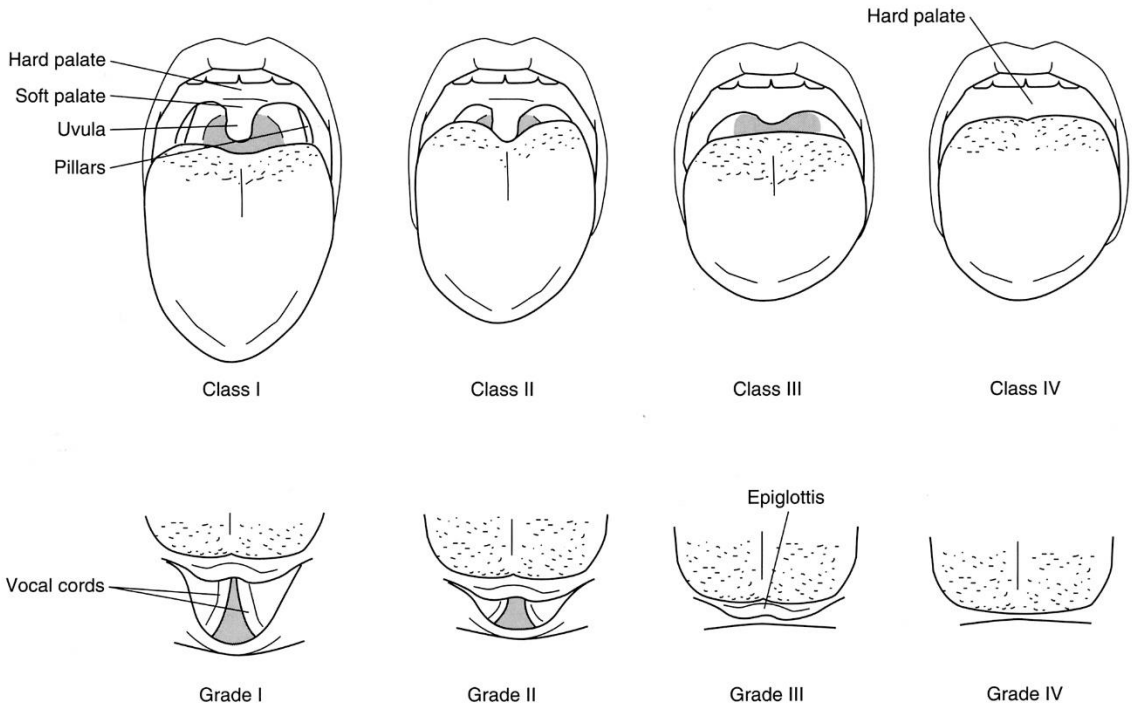
Class III: Only the base of the uvula visualized along with the soft palate

Class IV: No visualization of the uvula or soft palate

In this classification system, Class I and II airways are generally predicted easy to intubate, while Class III and IV are sometimes difficult. The same holds true with being able to bag mask ventilate a patient, Class I and II represent ease in ventilation, while Class III and IV may prove to be difficult to ventilate by this method.



MALLAMPATI CLASSIFICATION



Reference 4

B. PRE-MODERATE/DEEP SEDATION FASTING

Patients undergoing moderate or deep sedation for elective procedures ***should not*** drink fluids or eat solid foods for a sufficient period of time to allow for gastric emptying before their procedure.

Other clinical conditions that may delay gastric emptying and increase their risk of aspiration include:

The following fasting guidelines apply for otherwise healthy patients. **Deviations** from these guidelines may be indicated because of the patient's clinical presentation.

Minimal NPO Guidelines

Clear Liquids see below)	2 hours
Human Breast Milk	4 hours
Cows mil or opaque formulas	6 hours
Light Meal (see below)	6 hours
Full Meal	8 hours

- **Clear liquids** are defined as water, fruit juices without pulp, carbonated beverages, clear tea, and black coffee. The volume of liquid ingested is less important than the type of liquid ingested.
- **Light meal**: While the ASA suggest that patients may have a light meal up to 6 hours before a procedure, a light meal is defined typically as toast and clear liquids. Heavier meals that include fried or fatty foods or meat may prolong gastric emptying and ***require a longer fasting period***.
- In emergency situations where the procedure is necessary and moderate / deep sedation is required, these NPO guidelines do not apply. However, the risk of aspiration must be considered in determining the target of sedation, whether the procedure should be delayed or whether the trachea should be protected by intubation.

Other clinical conditions that may delay gastric emptying and increase their risk of aspiration include:

1. Anxiety
2. Severe pain
3. Autonomic dysfunction (gastroparesis)
4. Hiatal hernia / GERD
5. Morbid obesity
6. Pregnancy
7. Bowel obstruction
8. Multi-trauma
9. Head trauma / increased intracranial pressure
10. Ascites
11. Peritoneal dialysis
12. Oral x-ray contrast

Most importantly, in urgent, emergent, or other situations in which gastric emptying is impaired, the potential for pulmonary aspiration of gastric contents must be considered in determining:

1. The target level of sedation
2. Whether the procedure should be delayed
3. Whether the trachea should be protected by intubation

C. ASA PHYSICAL STATUS CLASSIFICATION

The American Society of Anesthesiologist classification of physical status aids in stratifying risk to the patient from the procedure and moderate or deep sedation. The classification is as follow:

ASA I	Normal, healthy patient with no systemic disease
ASA II	Mild to moderate systemic disease (e.g., controlled HTN, controlled diabetes, obesity, tobacco use).
ASA III	Severe systemic disease with functional limitations that is not incapacitating (e.g., uncontrolled HTN, uncontrolled diabetes, morbid obesity, liver disease).
ASA IV	Severe systemic disease that is incapacitating and a constant threat to life (e.g., ESRD, liver failure, CHF).
ASA V	A moribund patient who is not expected to survive without the operation or procedure (e. g. ruptured aortic aneurysm).

D. MONITORING

Monitoring of the patient during moderate or deep sedation is to be continuous throughout the procedure. Documentation of parameters such as EKG, blood pressure, pulse rate, respiratory rate, oxygen saturation, and level of sedation should be documented at a minimum every 5 minutes or upon any significant change or event. While many of these are standard monitoring, particular emphasis will be discuss regarding some important monitors and assessments.

Faulty equipment requires immediate intervention, and whiles an inconvenience, may require the rescheduling of the procedure.

Alarms should be on at all times. At no time should alarms be silenced. Should an alarm occur assumed the information from the monitor to be true and accurate and assess the patient first before considering an artifact or faulty monitor.

Monitors specifically addressed below are: pulse oximetry, capnography, and the level of sedation.

PULSE OXIMETRY

1. Pulse oximetry measures the amount of oxygen carried on hemoglobin in the arterial blood.
2. There are two forms of oxygen transport in the blood: hemoglobin and plasma: 97% of the oxygen is attached to hemoglobin. 1%-3% of the oxygen is dissolved in the plasma.
3. Pulse oximetry promptly and reliably, excluding artifacts, identifies hypoxemia more quickly than clinical signs such as cyanosis or disorientation which occur much later.

4. The accuracy of pulse oximetry declines below 60% saturation. It ***does not*** measure the patient's ventilation and ***does not*** monitor carbon dioxide accumulation or excretion.
5. It is important to understand that oxygen saturation does NOT equal PaO_2. The oxygen hemoglobin dissociation curve helps determine the correlation between oxygen saturation and PaO2 such that one can equate the following saturation with its corresponding PaO2:

Saturation (%)	PaO2 (mmHg)
95	90
60	50
75	40

Advantages of pulse oximetry:

1. Continuous monitoring
2. Multiple sites
3. Noninvasive: no damage to tissues
4. Calibration not required
5. User friendly
6. Multiple parameters measured: Sp02, Perfusion, Heart rate

Factors that affect the accuracy of pulse oximeter:

1. Slippage of the sensor: always check the position of the sensor first.
2. Movement, shivering, patient positioning.
3. Electrocautery: bipolar may create a false decrease in the Sp02 reading.
4. Low perfusion: bypass, NIBP, tourniquet, severe vasoconstriction, compartment syndrome, hypotension, severe hypovolemia.
5. Contrast/dyes: methylene blue, indigo carmine, indocyamine green, lyzurin dye.
6. MRI.
7. Excessive ambient light: such as infra red lights and surgical lamps.
8. Anemia: Hg < 5 may create a false decrease in Sp02 reading.
9. Hypoxemia: Sa02 < 70% may cause inaccurate readings.
10. Acrylic nails and nail polish, especially blue, green, or red nail polish.
11. Dyshemoglobinemias: methemoglobin, carboxyhemoglobin and sulfahemoglobin.
12. Rapid or erratic heart rates where pulse does not correlate with heart rate.

CAPNOGRAPHY

Capnography (ETCO2) should be used during both moderate or deep sedation. It the event that capnography is not functioning visual respiratory efforts and audible confirmation of breathing must be directly observed.

1. Capnography is the graphic display of the CO2 partial pressure as a waveform.
2. A Capnometer gives a digital display of the CO2 on inspiration and expiration.
3. Capnography usually includes capnometry to provide the digital display of a numeric value along with the waveform.

4. The most common method of measuring end tidal CO2 (ETCO2) is the diverting method: Gas is diverted from the patient's airway, via a small side port, and aspirated through small tubing to the measuring device.
5. A beam of infrared light is passed through the sampled gas. CO2 molecules in the light path absorb some of the infrared light waves. Capnography measures end tidal carbon dioxide (ETCO2) and along with the waveform generated can provide a quick assessment of ventilation and early detector of hypoventilation. A normal range for ETCO2 is 35 to 45 mmHg.

Purpose of Capnography**:**

1. The confirm the placement of an endotracheal tube
   a. Gold Standard
   b. Does not immediately rule out esophageal intubation
2. To determine the adequacy of ventilation
   a. Guides changes needed during mechanical ventilation
   b. Helps assess adequacy of spontaneous ventilation
3. To monitor the metabolic state
   a. Persistent increase in ETCO2 may indicate malignant hyperthermia
4. To monitor circulation for:
   a. Hypovolemic/cardiogenic shock
   b. Pulmonary embolism

LEVEL OF SEDATION / RESPONSIVENESS

It is important to continuously monitor a patient's level of sedation or responsiveness during the administering of medications for moderate sedation/analgesia. Remember that by definition moderate sedation is a drug induced depression of consciousness during which patients respond ***purposefully*** to verbal commands, either alone or accompanied by light tactile stimulation. The response of patients to commands during procedures performed with sedation/ analgesia serves as a guide to their level of consciousness. Spoken responses (when possible) also provide an indication that the patient is breathing.

ADMINISTRATION OF MEDICATIONS FOR MODERATE SEDATION:

Medical Center Policy 905, Moderate Sedation/Deep Sedation, Attachment E, Approved Moderate / Deep Sedation Medications and includes a tabulation of the recommended and suggested drugs and dosages for adult and pediatric patients. It also includes the general precautions and procedures for administrating these drugs.

General Precautions**:**

1. Dosages should be individualized. Certain patients may not tolerate these recommended doses. Some patients may need much less or more than the listed dose.
2. Do not administer intravenous medications rapidly.
3. Individual response may vary with age, physical status, and concomitant medications.

4. Use small increments to achieve appropriate levels of sedation. The drug should be titrated to the desired clinical effect.
5. Wait two or more minutes after each increment to evaluate the sedative effect fully.
6. Combination of sedatives and analgesics may be administered ass appropriate for the procedure being performed and the condition of the patient. Many of these medications have a synergistic respiratory depressant effect when administered in combination. Ideally, each medication should be administered individually to achieve the desired effect.
7. Antagonist for opioids and benzodiazepines should be readily available before the administration of any of these medications for moderate sedation/analgesia.
8. Abbreviations: PR = per rectum; IM = intramuscular; IV = intravenous

DRUGS USED IN MODERATE/DEEP SEDATION

A. OPIOIDS

All opioids produce sedation and analgesia and have the propensity to cause respiratory depression. Commonly used opioids used for moderate sedation/analgesia include morphine, meperidine, and fentanyl. Naloxone is an opioid antagonist that may be used to rescue a patient from respiratory depression.

Morphine

1. Produces sedation, analgesia, and mood alteration.
2. The onset of morphine 5 min for IV doses and 15 minutes for IM doses.
3. The peak effect of morphine is 20 minutes (IV) and 30-60 mins (IM)
4. The duration of action is 3-4 hours
5. Analgesia can occur without loss of consciousness, but large doses can produce obtundation and even coma.
6. Morphine can produce prolonged postoperative somnolence, respiratory depression, nausea, vomiting, and itching.
7. Histamine release and some reduction in sympathetic tone can produce hypotension. In a healthy, normovolemic patient orthostatic hypotension may develop with large doses of morphine.
8. Opioids such as morphine can produce elevation of $PaCO_2$ resulting to increase in cerebral blood flow and elevation of intracranial pressure and arrhythmias.
9. Caution should be taken when administering morphine to patients taking monoamine oxidase inhibitors due to exaggerated hypotension.

Meperidine (Demerol)

1. Meperidine is about one-tenth as potent as morphine IV. It is a synthetic opioid with atropine-like properties.
2. The onset of meperidine is 5 minutes (IV) and 10-15 minutes (IM).
3. The peak effect of meperidine is 5-7 minutes (IV) and 60 minutes (IM).
4. The duration of action is 2-4 hours.

5. Its effects on respiration and ventilation are similar to morphine. They produce moderate effects on tidal volume and slow respiratory rate.
6. In large dosages, it can produce tachycardia and cause negative inotropic effects.
7. Meperidine has an active metabolite (normeperidine) which when accumulated, produces convulsion. In large doses, meperidine can produce tremors, muscle twitching, and seizures. Meperidine should not be used in patients with renal failure since normeperidine excretion is renal dependent.
8. Caution should be taken when administering meperidine to patients taking monoamine oxidase inhibitors due to exaggerated hypertension.

Fentanyl (Sublimaze)

1. Fentanyl has more rapid onset and shorter duration than morphine. It is 100 times more potent than morphine IV.
2. The onset of fentanyl is 30 seconds (IV) and 7-8 minutes (IM).
3. The peak effect of fentanyl is 10 minutes (IV) and 30-45 minutes (IM).
4. The duration of action is 30-60 minutes.
5. Fentanyl in moderate doses of 2-10 microgram/kg or higher doses when given rapidly intravenous can produce skeletal muscle rigidity called "stiff chest syndrome". Sometimes this syndrome is so severe that it is impossible to adequately ventilate the patient.
6. Fentanyl does not have amnesic effect. High dose fentanyl can produce respiratory depression but no direct myocardial depression effect. Fentanyl lacks histamine release and suppresses the stress response associated with surgery or invasive procedures. Fentanyl also depresses the respiratory center in the brainstem so that normal response to hypoxia and hypercarbia is reduced.

Characteristics of Opioid Overdose

1. Altered level of consciousness
2. Respiratory depression
3. Muscle placidity, especially the airway
4. Mitotic pupils, unless pupils are more dilated secondary to hypoxia

B. BENZODIAZEPINES

Benzodiazepines are a group of medications most commonly used for moderate sedation. In addition to their sedative properties, most benzodiazepines have amnesic, anxiolytic, anticonvulsive and hypnotic effects. These drugs have NO ANALGESIC properties. Commonly used benzodiazepines used for moderate sedation include diazepam, lorazepam, and midazolam. Flumazenil is an antagonist that rapidly reverses the effect of benzodiazepines.

Diazepam (Valium) / Lorazepam (Ativan)

1. Diazepam and lorazepam have similar profiles. Lorazepam IV has a shorter duration of action but is approximately 5 times as potent as diazepam IV. Lipid solubility accounts for this prolonged duration of action.
2. Diazepam and lorazepam can produce depression of ventilatory response to carbon dioxide. Sometimes, even in small doses, diazepam may result in apnea particularly in elderly and sick patients.
3. Diazepam and lorazepam can cause mild reductions in blood pressure, cardiac output, and peripheral vascular resistance.
4. Benzodiazepines and opioids can have a synergistic effect and therefore, when used together may result in respiratory depression and apnea.
5. Due to prolonged duration of action of diazepam and lorazepam, they may not be suitable for outpatient procedures.
6. Cimetidine increases the elimination half-life of diazepam.

Midazolam (Versed)

1. Midazolam IV is twice as potent and is shorter acting than diazepam IV.
2. It has sedative, amnesic, anxiolytic, and anti-convulsant properties.
3. Midazolam produces dose related depression of the central respiratory system.
4. Elimination half life could be longer in elderly and obese patients.
5. Benzodiazepines and opioids can have a synergistic effect and therefore, when used together may result in respiratory depression and apnea.
6. Respiratory depression is more pronounced in the elderly and patients with respiratory comorbidity.
7. Midazolam should be titrated slowly to the desired effect. As little as 1 mg may be sufficient for some patients. No more than 2.5 mg (1.5 mg in elderly and debilitated patients) should be given over a period of 2 or more minutes and additional time should be allowed to evaluate the effect of the dose just given.
8. A total dose greater than 5 mg is usually not necessary to reach the desired effect and doses less than 5 mg will be required when given along with opioids.
9. Cimetidine increases the elimination half life of midazolam.

B. OTHER SEDATIVE AGENTS

The following medication must be used with caution since each is capable of inducing deep sedation or even anesthesia requiring airway management or rescue. There are NO reversals or antagonists for the following medications.

Ketamine

1. Ketamine is a phencyclidine derivative that produces a dissociative anesthesia.
2. This dissociative anesthesia resembles a catatonic state where the patient's eyes remain open. Laryngeal and pharyngeal reflexes remain intact and there is no respiratory depression.
3. Ketamine is also a potent analgesia and may be administered IV, IM and orally.
4. Ketamine interacts with *N*-methyl-D-aspartate (NMDA) receptors, opioid receptors, monoaminergic receptors, muscarinic receptors, and voltage-sensitive calcium channels.

5. Sympathetic stimulation by ketamine causes produces increases in heart rate, cardiac output, and blood pressure. Increases in intracranial pressure can also occur.
6. Ketamine is therefore, contradicted in persons with cardiovascular disease and elevated intracranial pressures.
7. Emergence delirium is another unique side effect of ketamine and is associated with high dose use (> 2mg/kg) in young children and females. It is characterized by hallucinations and morbid dreams occurring up to 24 hours after used. Benzodiazepines used prior to ketamine appear to be most effective in preventing emergence delirium.

Methohexital (Brevital)

1. Methohexital is an ultra-short acting barbiturate that exerts its sedative / hypnotic effect via gamma-aminobutyric acid (GABA) receptors in the central nervous system (CNS).
2. Methohexital has a rapid onset reflecting its rapid entrance into the CNS but has a short duration of action contributed by its redistribution to inactive tissues. Its duration of action is between 5 to 10 minutes when given intravenously.
3. Methohexital can also be administered rectally resulting in a longer duration of action (30 to 90 minutes).
4. Respiratory depression and cardiovascular depression resulting in hypotension can occur with the use of methohexital.
5. Pain at the site of injection can occur with the use of methohexital.
6. Slow incremental use of methohexital can be used to provide sedative conditions. Extreme care must be taken since its use can induce deep sedation and even anesthesia requiring airway management.
7. Methohexital is contraindicated in patients with a history of hypersensitivity or porphyria.

Propofol (Diprivan)

1. Propofol is an intravenous sedative-hypnotic agent. It belongs to a class of intravenous anesthetic agents called the alkylphenols.
2. Propofol contains soybean oil and egg lecithin.
3. Propofol has a rapid onset reflecting its rapid entrance into the CNS but has a short duration of action contributed by its redistribution to inactive tissues. Its duration of action is between 3 to 10 minutes.
4. Hypotension, oxyhemoglobin desaturation, apnea, airway obstruction, and/or oxygen desaturation can occur, especially following a rapid bolus of propofol. During initiation of deep sedation, slow infusion or slow injection techniques are preferable over rapid bolus administration, and during maintenance of deep sedation, a variable rate infusion is preferable over intermittent bolus administration in order to minimize undesirable cardiorespiratory effects.

5. Pain at the site of injection can occur with the use of propofol.
6. Slow incremental use of propofol can be used to provide sedative conditions. Extreme care must be taken since its use can induce deep sedation and even anesthesia requiring airway management. Elderly, debilitated, or ASA III/IV patients, rapid (single or repeated) bolus dose administration should not be used for deep sedation.
7. Propofol is not contraindicated in patients with an allergy to egg or soy. Patients with soy allergy or egg allergy can receive propofol without any special precautions. Any patient, whether soy or egg-allergic or not, who has an apparent allergic reaction to propofol should be evaluated by an allergist.
8. Propofol should not be Coad ministered through the same IV catheter with blood or plasma because compatibility has not been established. In vitro tests have shown that aggregates of the globular component of the emulsion vehicle have occurred with blood/plasma/serum from humans and animals. The clinical significance of these findings is not known.

C. ANTAGONISTS

Naloxone (Narcan)

1. Naloxone is a pure antagonist without agonist activity.
2. It's duration of action of about 30 to 45 minutes is very short. Sedation and respiratory compromise may reoccur if the duration of action of the opioid exceeds that of naloxone. Repeated doses of naloxone may therefore be required.
3. Primarily used to reverse respiratory depression. It can also reverse analgesia.
4. Large boluses of naloxone can cause hypertension, ruptured cerebral aneurysm, pulmonary edema, cardiac arrest, and death.
5. Naloxone also may unmask physical dependence, precipitate acute withdrawal syndrome, and elevate catecholamines.
6. Naloxone can also cross the placenta and precipitate fetal withdrawal.

Flumazenil (Romazicon)

1. Flumazenil is a benzodiazepine receptor antagonist. Used to reverse the sedative effects of benzodiazepines that may occur with overdose.
2. Flumazenil has a shorter duration of action (19-50 minutes) than the benzodiazepines being reversed. The duration of action depends on the dose and duration of the benzodiazepine administered and on the dose of flumazenil.
3. Sedation may therefore reoccur requiring a repeated dose of flumazenil.
4. In general, flumazenil has few side effects.
5. Adverse effects on patients dependent on benzodiazepines are headache, dizziness, sweating, nausea/vomiting, and flushing.
6. The patient must be monitored for two hours following administration of flumazenil for signs of re-sedation.

COMPLICATIONS ASSOCIATED WITH MODERATE OR DEEP SEDATION

1. Ineffective ventilation resulting from airway obstruction, respiratory depression causing hypoxia and hypercarbia.
2. Problems with the cardiovascular system including hypotension
3. Drug overdose or reaction (anaphylaxis or anaphylactoid reactions).
4. Aspiration associated with loss of protective airway reflexes.
5. Nausea and vomiting.
6. Problems with equipment compromising patient safety.

Airway and ventilatory compromise represent the most common complications occurring when administering moderate sedation/analgesia. Every practitioner administering moderate sedation/analgesia should be able to recognize a patient in respiratory distress and be able to rescue that patient. Rescuing a patient requires an understanding of the causes of airway and ventilatory compromise and the proper airway management skills to employ. Not all cases of respiratory compromise require the utilization of reversal agents.

What follows is an overview of the most common complications associated with the use of moderate sedation and suggested maneuvers or treatment for each.

AIRWAY OBSTRUCTION

Airway obstruction is most common complication associated with moderate sedation. Airway obstruction is the result from loss of tonicity of submandibular muscles, direct support to the tongue and loss of indirect support to the epiglottis.

Factors which may be associated with difficult airway management include:

1. Previous problems with anesthesia or sedation
2. Stridor, snoring, or sleep apnea
3. Anatomical variance (e.g., Pierre-Robin Syndrome, Trisomy 21)
4. Advanced rheumatoid arthritis
5. Obesity
6. Physical exam showing small mouth, large tongue, short neck, protruding incisors, facial hair, edentulous, short chin

Signs of airway obstruction include:

1. Inspiratory stridor or snoring
2. Sternal retraction
3. Rocking chest movements
4. Absence of breath sounds
5. Hypoxemia (e.g., drop in oxygen saturation)
6. Hypercarbia

A. HYPERCARBIA

In patients receiving moderate sedation, the usual source of hypercarbia is respiratory center depression from medications. All narcotics produce respiratory depression. Benzodiazepines and opioids may act synergistically to also suppress ventilation. Hypercarbia is defined as a $PaCO_2$ greater than 44 mmHg and is the result of hypoventilation.

Monitoring of ventilatory function by observation or auscultation during the administration of moderate sedation is ***imperative*** since hypoventilation may be difficult to detect especially when supplemental oxygen is being administered. In situations where visualization is not possible the use of capnography to monitor ventilation is appropriate. It is important to remember that ventilation and oxygenation are separate processes and the monitoring of oxygenation by pulse oximetry is **NOT** a substitute for monitoring ventilatory function.

In summary:

1. Maintenance of normal $PaCO_2$ is determined by adequate ventilation.
2. Hypercarbia is caused by respiratory center depression and hypoventilation.
3. All opioids cause respiratory depression.
4. Benzodiazepines and opioids may act synergistically to cause hypoventilation.
5. Monitoring of oxygenation by pulse oximetry is not a substitute for monitoring ventilatory function by observation, auscultation, or capnography.

B. HYPOXEMIA

Hypoxemia is present when PaO_2 is less than 60 mmHg or SpO_2 by pulse oximeter is less than 90%. Clinically, patients may become agitated before cyanosis of mucous membranes occurs.

Causes of Hypoxemia:

1. Hypoventilation
2. Low inspired oxygen
3. Increased oxygen consumption (e.g., shivering, sepsis, pain)
4. Low cardiac output
5. Anatomic shunt: refractory to oxygen therapy

Treatment of ventilatory or airway compromise:

1. Provide supplemental oxygen if not already being administered.
2. If airway obstruction is suspected, consider:
    a. Repositioning the patient's head
    b. Providing a head tilt
    c. Applying a chin lift or jaw thrust
    d. Persistent airway obstruction may require the use of airway adjuncts - oropharyngeal and nasopharyngeal airways.

3. Consider oversedation from medication therefore suspend further drug administration and support and maintain the patient's airway by the maneuvers above and consider the use of reversal agents like naloxone or flumazenil.
4. Should the above not correct the situation consider bag-mask positive ventilation and even intubation.

ANAPHYLAXIS AND ANAPHYLACTOID REACTIONS

Anaphylaxis and anaphylactoid reactions are acute and are characterized by wheezing, dyspnea, syncope, hypotension, and upper airway obstruction. Histamine release can be produced by administration of morphine and other agents. Latex allergy should also be considered when suspecting an allergic or anaphylaxis reaction.

Treatment of anaphylactic or anaphylactoid reactions:

1. Prompt recognition of the clinical situation and stopping the administration of the suspected offending drug.
2. Ventilation with 100% oxygen. Securing the airway with endotracheal intubation may be necessary.
3. Prompt use of fluids and epinephrine (IV or SQ) and antihistamines.
4. Supportive care

ASPIRATION

During deep sedation where airway protective reflexes are lost, aspiration is a risk.

Risk factors for aspiration:

1. Inadequate fasting or recent oral intake
2. Diabetes (presence of autonomic dysfunction)
3. Pregnancy
4. Obesity
5. Hiatal hernia or gastric reflux
6. Altered consciousness

Diagnosis of aspiration:

1. Suspect aspiration in patient with the above risk factors having respiratory difficulty, tachypnea, tachycardia, cyanosis, and oxygen desaturation.
2. Blood gases may reveal hypoxemia with mixed metabolic and variable respiratory acidosis.

3. In severe cases of aspiration, systemic hypotension, pulmonary hypertension, and pulmonary edema may occur.
4. Radiographic findings are variable

MODERATE SEDATION/DEEP SEDATION PROVIDER LEARNING MODULE

NAUSEA AND VOMITING

Nausea and vomiting can cause hypertension or hypotension, tachycardia, bradycardia, and aspiration. Nausea and vomiting are the leading cause of unexpected hospital admission.

Predisposing factors of nausea and vomiting are:

1. Age (younger patient more susceptible)
2. Female gender
3. History of postoperative emesis
4. Presence of hypoglycemia, pain, hypotension, or hypoxia.

Treatment of nausea and vomiting:

1. Evaluate and treat causes of hypoglycemia, pain, hypoxia, or hypotension

2. Metoclopramide (Reglan) - Adult: 10-20 mg. IV; Pediatric 0.15 mg/kg IV
3. Ondansetron (Zofran) - Adult: 4-12 mg. IV; Pediatric

4. Droperidol* - Adult: 0.625-1.25 mg IV; Pediatric: 0.01-0.02 mg/kg IV

* *Droperidol while a very effective anti-emetic, has a black box warning by the FDA requiring that a 12 lead EKG be obtained prior to its use to rule out prolonged QT interval, and that the patient have EKG monitoring for 2-3 hours after administration of droperidol.*

References:

1. "Practice Guidelines for Sedation and Analgesia by Non-anesthesiologists", ASA Task Force, Anesthesiology 2002; 96: 1004-17
2.