Community Memorial Health System

PROVIDER ADULT PROCEDURAL SEDATION MODULE

I. Introduction

The purpose of this module is to provide non-anesthesiologist licensed independent practitioners with information necessary to safely and appropriately care for a patient receiving moderate/deep sedation within the healthcare setting.

"Moderate sedation will be administered by a nurse with validated competencies under the direction of a credentialed physician performing the procedure. The physician must have been granted moderate sedation privileges by the Community Memorial Health System Medical Staff."

The Procedural Sedation Form has been created to help ensure documentation meets moderate and deep sedation policy guidelines. This form is an approved part of the medical record and is to be used for every episode of moderate or deep sedation at Community Memorial Hospitals in Ventura and Ojai that occurs outside of the operating room, in which an anesthesiologist is not involved. If anesthesia is supporting sedation in the GI lab or interventional radiology suite, the Anesthesia Record may be used in place of the Moderate and Deep Sedation Form.

II. Objectives

After completion of this module, the learner will be able to:

- Identify the difference between minimal, moderate and deep sedation.
- Learn the Community Memorial Health System policy for moderate/deep sedation.
- Identify the purpose of moderate and deep sedation.
- Identify the elements required for pre-sedation, intra-procedure, and post sedation assessment with a focus on identifying high risk patients.
- Learn the standardized monitoring criteria needed to reduce complications associated with moderate and deep sedation including pulse oximetry.
- Identify common medications used for sedation, their indications, effects and possible complications and adverse reactions.
- Be familiar with the medications used to reverse opiates and benzodiazepines.
- Identify the signs of respiratory depression and airway compromise.
- Articulate the elements of airway management during moderate and deep sedation.
- Identify and treat emergency situations arising from moderate and deep sedation.
- Identify discharge criteria for patients who have received moderate and deep sedation.

III. Definitions

The Continuum of Sedation:
Practitioners intending to produce a level of sedation should be able to rescue patients whose level of sedation becomes deeper than initially intended.

Levels of Sedation

- **No Sedation** may occur with the delivery of a medication with a goal to affect analgesia without any impairment in cognitive function or coordination. Some of the same drugs used to achieve sedation may be used in lower doses to achieve analgesia without sedation.

- **Minimal Sedation** (anxiolysis) is a drug-induced state during which patients respond normally to verbal commands (note: reflex withdrawal from painful stimulus is not considered a purposeful response.) Although cognitive function and coordination may be impaired, ventilatory and cardiovascular functions are unaffected.

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- **Moderate Sedation** (formerly referred to as "Conscious Sedation") is a drug-induced depressed level 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 may be adequate. Cardiovascular function is usually maintained.

- **Deep Sedation** is a drug-induced depressed level 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 maintaining a patent airway and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.

- **Anesthesia** consists of general anesthesia, spinal, or major regional anesthesia. It does not include local anesthesia. General anesthesia is a drug-induced loss of consciousness during which patients are not arousable even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.

Because sedation is a continuum, it is not always possible to predict how a particular patient will respond. Individuals providing moderate or deep sedation must be qualified and have the appropriate credentials to manage patients at whatever level of sedation is achieved either intentionally or unintentionally.

	Minimal Sedation (Anxiolysis)	Moderate Sedation/ Analgesia ("Conscious Sedation")	Deep Sedation/ Analgesia	General Anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful response** after repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	no intervention required	Intervention may be required	Intervention often required
Spontaneous Ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular Function	Unaffected	Usually maintained	Usually maintained	May be impaired

REFERENCE: American Society of Anesthesiologist Task force on Sedation and Analgesia by Non-anesthesiologists. Practice Guidelines for Sedation and analgesia by Non-Anesthesiologists. Anesthesiology, 2002; 96:1004-17.

Common procedures that may require Moderate Sedation

- Fiber optic bronchoscopy
- Gastrointestinal endoscopy
- Bone marrow aspiration
- Liver biopsy
- CT Scans
- MRI
- Invasive radiologic procedures
- Chest tube insertion
- Fracture/Joint reduction

Common procedures that may require Deep Sedation

- Cardioversion
- Liver / lung biopsy
- Foreign body removal
- Fracture/Joint reduction
- Extensive I & D / debridement

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IV. Physician Responsibilities for Procedural Sedation

Physicians must be granted clinical privileges for procedural sedation procedures through the Community Memorial Health System credentialing and privileging process. Elements of physician responsibilities include the following:

- Perform a pre-sedation patient assessment and document properly on the chart
- Develop a sedation plan and obtain informed consent prior to sedation
- Understand dose, side effects and reversal agents for medications being used
- Be present during the administration of the drug, duration of the procedure, as well as during immediate post-operative stage until the patient is medically stable
- Be ready and able to provide immediate response in emergent situations

V. Equipment and Supplies

Each designated area where sedation is administered must have emergency resuscitative equipment immediately available that is equivalent to that used in other areas of the hospital, and which is checked and maintained on a scheduled basis. All emergency equipment must be able to accommodate patients of any size or age undergoing procedures in that area. Appropriate equipment for patient care and resuscitation will include:

- Emergency “crash” cart
- Suction device/suction catheters
- Emergency drugs
- Oxygen source
- Defibrillator
- Nasal O2 cannulas/O2 masks
- EKG monitor
- Blood pressure monitor
- Positive pressure oxygen delivery
- CO2 detector with continuous waveform capnography (optional)
- Pulse oximeter
- IV supplies
- Bag-valve-mask, oral airway, nasal trumpet
- Intubation equipment that includes appropriate size endotracheal tubes and laryngoscopes

VI. Personnel Requirements

Moderate sedations must be administered under the direction of a credentialed physician and monitoring must be performed by a licensed nurse who has been deemed competent through the hospital’s competency assessment and validation process to monitor and manage the care of sedated patients.

The number of clinicians needed to perform procedural sedation anesthesia (PSA) and the procedure safely may vary according to the patient and the procedure. In most cases, one clinician performs the procedure while another (usually a nurse) administers the sedative agents and monitors and records the patient's vital signs and clinical status. Whenever possible, we suggest that this minimum standard be met [25].
It remains controversial whether an additional clinician, separate from the clinician performing the procedure, who is skilled in deep sedation administration and airway management should be present [19,30]. Guidelines from the American Society of Anesthesiologists (ASA) call for someone with "advanced life support skills" to be immediately available (within five minutes) for PSA and present in the procedure room whenever deep sedation is being performed [3].

VII. Pre-sedation Assessment

Pre-procedure patient assessment by the physician should include the following:

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- Obtain informed consent from the patient, his/her surrogate decision maker or two physicians in an emergent situation.
- Assess patient's eligibility for sedation using the American Society of Anesthesiologist's (ASA) physical status classification
- Review present medication regimen (prescribed, over-the-counter, and herbal supplements), medication taken within the last 48 hours including any as needed medications; especially opioids or other narcotics
- Review substance use, including alcohol and tobacco
- Verify allergies and sensitivities to medications, latex, chemical agents, foods, and adhesives
- Determine patient's ability to tolerate and maintain the required position for the duration of the planned procedure
- Review relevant aspects of the patient's medical history and review of organ systems with emphasis on cardiac and pulmonary systems. History should include previous reactions to anesthesia and/or sedation.
- Determine time of last oral intake
- Obtain baseline vital signs (blood pressure, pulse, respiratory rate, and O2 saturation)
- Review height and weight
- Perform focused physical examination, including airway, oropharynx, cardiac and pulmonary systems
- Airway assessment with recognition of high risk airways and documentation of Mallampati classification
- Review any relevant lab results
- Review status of pregnancy test (if possible)

For patients that will be discharged from hospital setting, identification of a responsible adult to accompany them home after the procedure. If no ride/responsible adult can be arranged, the procedure will be cancelled or arrangements will be made for observation or admission to the hospital.

VIII. Pre-sedation Risk Assessment

ASA Classification System

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:

CLASS	DESCRIPTION	SEDATION PLAN	EXAMPLES
ASA I	No known systematic disease. Normal, healthy patient	May have moderate sedation without other consideration.	Healthy patient without evidence of systematic disease.
ASA II	Mild or well controlled systematic disease.	May have moderate sedation without other consideration.	Patient who smokes, with well controlled hypertension.
ASA III	. Severe systemic disease	Consider medical consultation.	Patient with diabetes and stable angina.
ASA IV	Severe systemic disease with constant threat to life.	Consider involvement of anesthesia for MAC (monitored anesthesia care).	Patient with diabetes, angina and CHF with dyspnea and chest pain on exertion or not expected to survive without intervention.

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ASA V	Moribund patient who is not expected to survive without procedure.	Patient with little chance of survival. Consider risks vs Benefits of proceeding.	Massive pulmonary embolus or rupture of vascular structures with profound shock.

ASA VI Declared brain dead patient whose organs are being removed for donor purposes

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:
    i. Advanced rheumatoid arthritis
    ii. 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. These 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, poor dental hygiene and/or loose teeth

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

1. Head and 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); loose or capped teeth; presence of dental appliances; high arched palate; macroglossia or large tongue; tonsillar hypertrophy; nonvisible uvula
3. Jaw: Micrognathia, retrognathia, trismus, or significant malocclusion

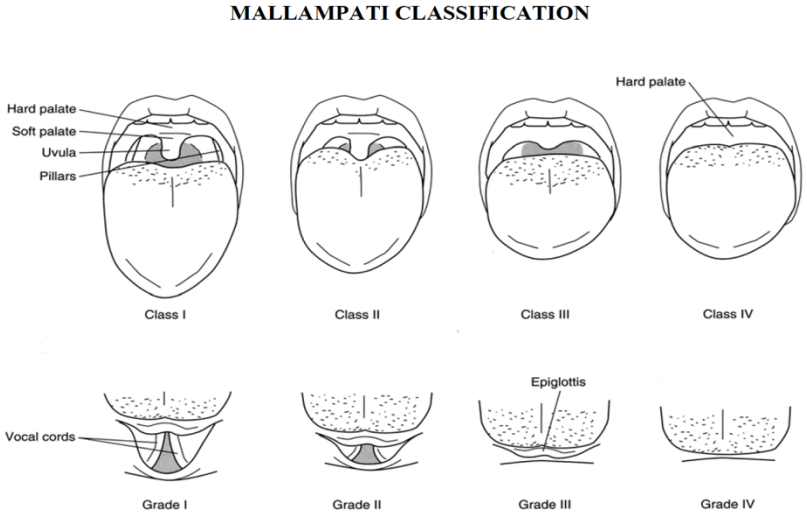
The examination of the airway involves inspection and evaluation of:

1. Oral cavity (identification of loose, chipped or capped teeth, presence of dentures or dental bridges)
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 -The Mallampati airway classification attempts to grade the degree of difficulty of endotracheal intubation from grade I to IV. It is a less than ideal predictor of the Cormack Lehane view obtained during direct laryngoscopy. 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

Cormack Lehane

Pre-moderate/deep sedation fasting

Aspiration risk — Aspiration of gastric contents during anesthesia or PSA is a rare, though much feared complication [13,14]. Patients undergoing emergency procedures requiring sedation are thought to be at increased risk of aspiration because their stomachs are often full, and the procedure cannot be delayed. Aspiration frequently does not cause harm. However, aspirated gastric contents above a critical volume and acidity can cause severe respiratory and systemic consequences [20]. The importance of fasting for preventing aspiration during PSA remains unclear.

Guidelines to reduce aspiration risk were put forth in a consensus statement by the American Society of Anesthesiologists (ASA) [3]. These guidelines are based upon expert opinion and to a lesser extent extrapolated from general anesthesia data, in which aspiration with serious consequences is rare [3,21]. The ASA guidelines recommend that patients undergoing PSA for "elective procedures" fast according to the standards used for general anesthesia. This requires that patients not eat or drink for two hours after drinking clear liquids and six hours after ingesting solid foods or cow's milk [3]. Additional fasting time (about 8 hrs) may be needed in cases of

patients consuming fried foods, fatty foods, or meat. If these standards cannot be met, the guidelines recommend that the clinician consider delaying the procedure, reducing the level of sedation, or protecting the airway with endotracheal intubation.

Implementing these guidelines in the emergency department (ED) presents several problems. First, it is rare that patients requiring emergent PSA meet these fasting criteria. Second, emergent procedures cannot be delayed. Finally, although fasting to reduce the risk of aspiration during PSA or elective surgery makes intuitive sense, there is little evidence to support this approach [21-26].

According to one review, patients who fast for two hours have the same gastric volume and pH as those who fast for longer periods [20]. Furthermore, there is no clear evidence that a relationship exists among fasting time, gastric volume, gastric pH, depth of sedation, and the likelihood of aspiration [13,19,23,25,27,28]. Clinically significant aspiration during emergency department PSA appears to be rare [14]. Furthermore, endotracheal intubation may not protect the patient from aspiration [21,25,27,29-31]. Aspiration can occur despite the presence of an endotracheal tube, while the airway manipulation involved in performing intubation appears to increase the risk of aspiration.

In light of the available literature, the American College of Emergency Physicians policy statement on PSA states: "Recent food intake is not a contraindication for administering procedural sedation and analgesia but should be considered in choosing the timing and target of sedation" [32]. We concur and suggest the following approach to reducing aspiration risk:

●Carefully consider the risks and benefits of performing the procedure emergently. Although there is no proof that longer fasting times reduce aspiration risk, it is reasonable to wait if the patient's stomach is full and the procedure is not a true emergency [32]. This is particularly true when a potentially difficult airway or an increased risk for aspiration exists, such as with the following circumstances [25]:

•Conditions predisposing to esophageal reflux (eg, bowel obstruction, hiatal hernia)
•Extremes of age (<6 months or >70 years old)
•Severe systemic disease (ASA class III or greater)
•Other concerning conditions (eg, depressed mental status)

●Avoid deep sedation. No evidence clearly demonstrates that deeper levels of sedation increase the risk of aspiration [25]. Nevertheless, lighter sedation may permit the patient to maintain protective airway reflexes, which reduces risk.

●We do not suggest administration of pre-procedural antacids or motility agents to reduce aspiration risk. These medications have not been shown to reduce such risk [23].

In some cases it may be best to perform the procedure under general anesthesia in the operating room, although this approach has not been proven to reduce the risk of aspiration [28,29].

●Drug overdose or reaction (anaphylaxis or anaphylactoid reactions).

●Aspiration associated with loss of protective airway reflexes.

IX. Complications of Procedural 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 represents 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

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 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
6. Waveform capnography, if available, should be strongly considered during procedural sedation to monitor ventilation

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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 over-sedation 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

Avoidance of aspiration:
Suction should be connected, tested and immediately available prior to any episode of moderate or deep sedation.

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

NAUSEA AND VOMITING

Nausea and vomiting can cause hypertension or hypotension, tachycardia, bradycardia and aspiration. Nausea and vomiting is 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
3. Ondansetron (Zofran) - Adult: 4-12 mg IV

X. Reportable Conditions Associated with Procedural Sedation

1. Unplanned transfer to higher level of care, including inpatient admission
2. Use of reversal agents
3. Adverse medication reaction
4. Suspected aspiration
5. Hemodynamic instability
6. Unexpected intubation or need for mechanical ventilation
7. Cardiac arrest
8. Patient, family or staff complaint regarding quality of sedation

XI. Sedation Drugs

Many agents on the hospital formulary can cause loss of airway reflex. For the purpose of this study module, only the most common medications used for procedural sedation will be discussed.

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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 medication administration to evaluate the sedative effect fully.
6. A combination of sedatives and analgesics may be administered as 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 COMMONLY USED FOR DEEP SEDATION

Historically, procedural sedation outside of the operating room was commonly achieved with a combination of opioids and benzodiazepine. This often leads to complications associated with sedation due to variable patient specific metabolism, prolonged duration of action and unpredictable effects when used in combination with other drugs. More recently, the use of ultrashort acting sedative/hypnotic medications has become more common. These medications have more predictable effects but often result in deep rather than moderate sedation and their use should only be considered by providers with a comprehensive understanding of their pharmacokinetics, effects and potential adverse reactions.

Ketamine

Mechanism of Action: Is a PCP derivative. It produces a cataleptic-like state in which the patient is dissociated from the environment by direct action on the cortex and limbic system. It can cause hyper-sympathomimetic responses. Ketamine is a non-competitive NMDA receptor antagonist that blocks glutamate. Low doses produce analgesia, modulate central sensitization, hyperanalgesia, and opioid tolerance. It reduces polysynaptic spinal reflexes. Low dose ketamine (0.1-0.2 mg/kg IV) may be used for analgesia avoiding moderate or deep sedation without the need for central monitoring.

Side effects: The reported side effects of ketamine include tachycardia, hypertension, laryngospasm, emergence reactions, nausea and vomiting, increased intracranial and intraocular pressure, and hypersalivation. Effects on intracranial and intraocular pressure have been called into question in recent years and most providers no longer consider increased intracranial or intraocular pressure to be a contraindication to Ketamine. Ketamine can exacerbate schizophrenia and should be avoided in patients with this condition. Tachycardia and hypertension are generally mild and transient, and significant cardiorespiratory events are rare, but consideration of other agents should be considered when a patient is at risk for coronary artery disease or ischemic cerebrovascular disease.

The risk of laryngospasm may be greater in patients with anatomic abnormalities of the upper airway (e.g. tracheal stenosis, tracheomalacia) or those undergoing procedures involving significant or prolonged stimulation of the oropharynx. Guidelines published by the American College of Emergency Physicians recommend preventing secretions or blood from accumulating in the posterior oropharynx and avoiding excessive stimulation of this region with suction devices or other instruments in patients receiving ketamine for procedural sedation. Anti-sialogogues are not recommended unless oral secretions are expected to complicate the procedure or sedation. Consider pretreatment with antiemetic as nausea and vomiting are common with ketamine administration.

Emergence reactions are the most commonly reported side effects. These reactions vary in their intensity and have been described as disorientation, dream-like experiences, or hallucinations that may be frightening. They occur in up to 20 percent of adults but can be prevented or treated by giving a small dose of midazolam.

Etomidate (Amidate)

Mechanism of Action: Ultrashort-acting non-barbiturate hypnotic (benzylimidazole) used for the induction of anesthesia; chemically, it is a carboxylated imidazole, which produces a rapid induction of anesthesia with minimal cardiovascular effects. It produces EEG burst suppression at high doses.

Side effects: Potential side effects of etomidate include myoclonus, respiratory depression, adrenal suppression, and nausea and vomiting. Myoclonus is the most frequently reported side effect. It is thought to be related to subcortical disinhibition and has been reported in up to 80 percent of patients who receive etomidate for procedural sedation. The degree of myoclonus may be dose dependent and ranges from mild and transient to severe enough to prevent completion of the procedure.

According to a systematic review of etomidate for procedural sedation, respiratory depression occurs in approximately 10 percent of cases. In this review, respiratory depression was defined as a fall in oxygen saturation below 90 percent or apnea. No serious complications occurred as a result and respiratory depression resolved quickly without major interventions in the great majority of cases. Nevertheless, clinicians must be prepared to support the patient's airway and breathing in the event of respiratory compromise.

Etomidate can have more profound and prolonged effects in the elderly and patients with renal or hepatic dysfunction. In such patients, doses in the lower dosing range should be used. An important benefit of etomidate is that it maintains cardiovascular stability.

Etomidate has no analgesic properties and often requires the co-administration of a short-acting opioid, such as fentanyl, which increases the risk of respiratory depression. To reduce this risk, smaller doses of fentanyl should be used.

Propofol

Mechanism of Action: Propofol, 2-6 Diisopropophenol, is a short-acting, lipophilic intravenous general anesthetic. Propofol causes global CNS depression, presumably through agonism of $GABA_A$ receptors and perhaps reduced glutamatergic activity through NMDA receptor blockade. It crosses the blood brain barrier rapidly. Propofol acts as a sedative and amnestic but provides no analgesia.

Side effects: Potential side effects include pain on injection (due to the activation of TRPA1 pain receptor found on sensory nerves which can be alleviated by use of a larger IV), hypotension (due to myocardial depression) and respiratory depression. These side effects generally resolve quickly and uneventfully because of the brief duration of action. Because hypotension can produce complications in patients with severe medical problems (sepsis, cardiac dysfunction) or hypovolemia, volume repletion prior to sedation should be performed when possible. Transient hypotension is common in elderly and hypovolemic patients.

“Ketofol”

Ketofol refers to the use of ketamine and propofol together for procedural sedation. The mechanism of action and side effects of these two medications are discussed above. The use of “Ketofol” has been proposed as a mechanism to reduce side effects of ketamine and propofol by using both medications in smaller doses than those typically used when either agent is used alone. The doses that has been studied and shown to be safe in clinical trials are 0.5 mg/kg IV as a starting dose for each medication with titration of either medication up to a dose of 0.7 mg/kg IV. The medications may be combined in a single syringe and be administered together or sequentially in separate syringes with propofol given first. By using the medications together in smaller doses, clinical studies

have shown less respiratory depression and hypotension than when propofol is used alone and less vomiting and emergence reactions than when ketamine is used alone.

Dexmedetomidine

Mechanism of Action: Dexmedetomidine is a relatively selective alpha2-adrenergic agonist with sedative properties that is indicated for procedural sedation in non-intubated patients and prior to and/or during surgical procedures. It mainly has sedative effects but has some analgesic properties as patients receiving dexmedetomidine postoperatively have been shown to require less opioid than those receiving equivalent doses of benzodiazepines.

Side Effects: Most common side effects include hypotension and bradycardia and occur in greater than 2% of patients receiving Dexmedetomidine. Transient hypertension can also occur. Other side effects that occur at a frequency greater than 2% include in patients receiving dexmedetomidine for procedural sedation include: nausea, respiratory depression, pyrexia, and dry mouth. Atrial fibrillation occurs in 4% of patients receiving dexmedetomidine for ICU sedation but was not seen when used for procedural sedation. Administration with sedatives, hypnotics and/or opioids leads to an enhancement of effects.

Drug Name	Initial Dose	Dosing Increments	Considerations
ketamine (Ketalar®)	IM 2 to 4 mg/kg IV 0.5 to 1 mg/kg over 1 min	0.25-0.5 mg/kg IV every 5 min prn	Co-administration with barbiturates, narcotics and benzodiazepines prolong ketamine's recovery time Typical duration of 15 to 30 minutes Low dose ketamine (0.1-0.2 mg/kg IV) may be used for analgesia without sedation and do not require advanced monitoring
Etomidate (Amidate®)	IV: 0.1-0.15 mg/kg	0.05-0.1 mg/kg every 3-5 min prn Some sources say not to give repeated doses of this agent	Adrenal suppression may occur with single doses so use with caution in patients with sepsis Typical duration is 4 to 10 minutes
Propofol (Diprivan)	**Bolus dosing (for procedures)**: up to 0.5 mg/kg initial bolus **Typical bolus dosing**: initial dose of 20 to 40 mg followed by additional doses of 10 to 20 mg	10 to 20 mg every 3-5 min prn	Slow infusion or slow injections over 3 to 5 minutes recommended for older, debilitated, or ASA III or IV risk pts Maximum dose of 1-2 mg/kg Typical duration is 3 to 10 minutes
Dexmedetomidine (Precedex)	**Bolus dosing: Not recommended due to risk of hypotension**	0.2-1.4 mcg/kg/hr titrated q15min	Effects enhanced when used with other sedatives or opioids. Typical duration of action is 6-10 minutes

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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 and fentanyl. Naloxone is an opioid antagonist that may be use to rescue a patient from respiratory depression.

Morphine

1. Produces sedation, analgesia, and mood alteration.
2. The onset of morphine is 5 minutes for IV doses and 15 minutes for IM doses.
3. The peak effect of morphine is 20 minutes (IV) and 1 hour (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, euvolemic patient, hemodynamic instability is uncommon.
8. Opioids such as morphine can produce elevation of $PaCO_2$ resulting in an 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.

Fentanyl (Sublimaze)

1. Fentanyl has more rapid onset and shorter duration than morphine. It is 100 times more potent than morphine.
2. The onset of fentanyl is 30 seconds (IV) and 5-10 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. If stiff chest syndrome does occur, the patient should receive a paralytic agent such as succinylcholine and undergo rapid sequence intubation.
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

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Drug Name **Opioid**	**Initial Dose (average)** **↓ with benzodiazepines**	**Dosing Increments**	**Considerations**
Fentanyl (Sublimaze)	0.5 to 1 mcg/kg IVP over 2 minutes Typical dose: 25 to 50 mcg	0.5 to 1 mcg/kg or 25 to 50 mcg IVP q 5 min prn	Max 250 mcg/hr Typical duration is 30 minutes
Morphine	0.05 to 0.1 mg/kg over 2 min Typical dose: 2 to 5 mg	0.05 mg/kg or 1 to 2.5 mg IVP q 15 min prn	Max 10 mg/hr Typical duration is 3 to 4 hours
Hydromorphone (Dilaudid)	0.0063-0.0125 mg/kg IVP over 5 min Typical dose: 0.2-0.5 IVP over 5 min	0.003 mg/kg or 0.1-0.3 IVP q 15 min prn	Max 2 mg/hr Typical duration is 4 to 5 hours

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. **Benzodiazepines are strongly discouraged for use in procedural sedation in patients over the age of 70.** 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 has a similar duration or action but is approximately 5 times as potent as diazepam. Lipid solubility accounts for this prolong duration of action. The following discussion applies equally to diazepam and lorazepam.
2. Diazepam 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 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 diazepam and lorazepam prolonged duration of action, they may not be suitable for outpatient procedures.
6. Cimetidine increases the elimination half-life of diazepam and lorazepam.

Midazolam (Versed)

1. Midazolam is twice as potent and is shorter acting than diazepam.
2. It has sedative, amnesic, anxiolytic and anticonvulsant 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.

Benzodiazepines	Initial Dose (average) ↓ with narcotics	Dosing Increments	Considerations
Midazolam (Versed)	Typical dose: 0.5 mg to 1 mg IVP	0.5 to 1 mg IVP every 5 min prn	Max 6 mg/hr (in absence of deep sedation agent) Duration of 15 to 60 minutes, depending on patient and dose Greater than 90% of patients will require less than 2mg midazolam to achieve moderate sedation
Diazepam (Valium)	1 to 2 mg IVP over 2 min	1 to 2 mg IVP every 5 min prn	Max 10 mg Typical duration is 15 to 30 minutes
Lorazepam (Ativan)	1 mg IVP over 2 min	0.5 IVP every 15 min prn	Max 2 mg/hr Typical duration is 6 to 8 hours

REVERSAL AGENTS

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.
7. Hypotension can be a side effect.

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 (20 to 90 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

Drug Name Reversal Agent	Initial Dose (average)	Dosing Increments	Considerations
Naloxone (Narcan) For Opioids	Typical dose: 0.1 to 0.4 mg IVP over 30 seconds	Titrate to effect	Maximum of 10 mg/hr Typical duration of 45 minutes to one hour
Flumazenil (Romazicon) For Benzodiazepines	0.2 mg IVP over 15 seconds	Titrate to effect May repeat 0.2 mg every 1 min prn x 4 additional doses	Maximum of 1mg/hr Typical duration of one hour

XII. Monitoring During the Sedation Episode

Monitoring of the patient during moderate or deep sedation is to be continuous throughout the procedure. EKG, blood pressure, pulse rate, respiratory rate, oxygen saturation, continuous waveform capnography and level of sedation should be monitored in all patients undergoing moderate or deep sedation. These parameters should be documented at a minimum every 5 minutes or upon any significant change or event. While many of these are standard monitoring, pulse oximetry and wave form capnography play central roles in sedation monitoring and will be discussed in more detail below.

Faulty equipment requires immediate intervention, and while 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, assume the information from the monitor to be true and accurate and assess the patient first before considering an artifact or faulty monitor.

Monitoring 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 PaO_2 such that one can equate the following saturation with its corresponding PaO_2:

Saturation (%)	PaO_2 (mmHg)
95%	90mmHg
75%	50mmHg
60%	40mmHg

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: $Sp0_2$, 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 $Sp0_2$ 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 $Sp0_2$ reading.
9. Hypoxemia: $Sa0_2$ < 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.

Continuous Wave Form Capnography

Capnography allows continuous measurement of exhaled carbon dioxide and displays the resulting waveform graphically. It provides an advantage over pulse oximetry alone by identifying respiratory depression earlier and more consistently especially when supplemental oxygen is provided.

1. Capnography is the graphic display of the CO_2 partial pressure as a waveform.
2. Capnography allows continuous measurement of exhaled carbon dioxide and displays the resulting waveform graphically.
3. $ETCO_2$ is the highest value of carbon dioxide measured during the end of expiration of each breath. These measurements can be used to assess the adequacy of ventilation during procedural sedation and analgesia.
4. The most common method of measuring $ETCO_2$ 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. CO_2 molecules in the light path absorb some of the infrared light waves.
6. Capnography measures end tidal carbon dioxide ($ETCO_2$) and along with the waveform generated can provide a quick assessment of ventilation and early detector of hypoventilation.
7. A normal range for $ETCO_2$ is 35 to 45 mmHg. When capnography rises above baseline, the patient is hypoventilating. When capnography reaches a level above 55 mmHg, the patient is at immediate risk for respiratory failure and/or sequelae of respiratory acidosis and consideration to be given to assisted ventilation, reversal agents and abortion of the procedure.

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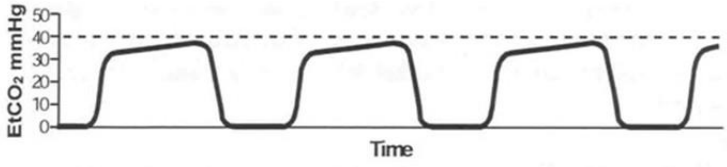
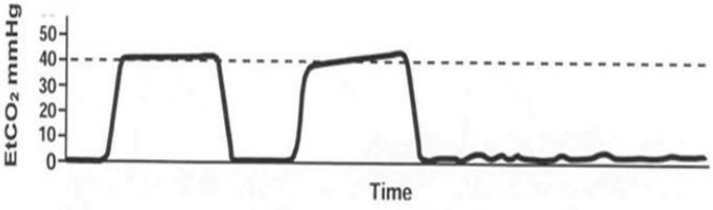
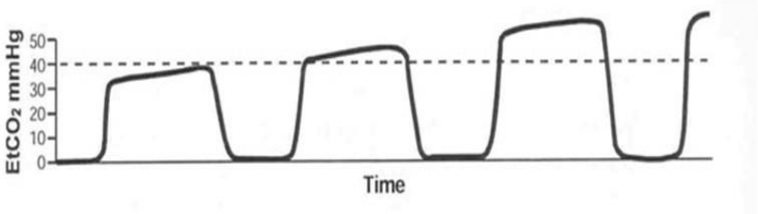
CAPNOGRAPHY WAVEFORMS

Normal





Apnea/Loss of Waveform



Hypoventilation

Level of Sedation

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. The RASS Sedation Scale is a validated tool useful to objectively assess level of sedation.

Richmond Agitation Sedation Scale (RASS)

Score Term Description

+4	COMBATIVE: Overtly combative, violent, immediate danger to staff
+3	VERY AGITATED: Pulls or removes tube(s) or catheter(s); aggressive
+2	AGITATED: Frequent non-purposeful movement, fights ventilator
+1	RESTLESS: Anxious but movements not aggressive or vigorous
0	Alert and calm
-1	DROWSY: Not fully alert, but has sustained awakening (eye-opening/eye contact) to voice (>10 seconds)

-2 LIGHT SEDATION: Briefly awakens with eye contact to voice (<10 seconds)
-3 MODERATE SEDATION: Movement or eye opening to voice (but no eye contact)
-4 DEEP SEDATION: No response to voice, movement or eye opening to physical stimulation
-5 UNAROUSABLE: No response to voice or physical stimulation

Procedure for RASS Assessment

1. Observe patient
   a. Patient is alert, restless, or agitated. (score 0 to +4)
2. If not alert, state patient's name and say to open eyes and look at speaker.
   b. Patient awakens with sustained eye opening and eye contact. (score –1)
   c. Patient awakens with eye opening and eye contact, but not sustained. (score –2)
   d. Patient has any movement in response to voice but no eye contact. (score –3)
3. When no response to verbal stimulation, physically stimulate patient by shaking shoulder and/or rubbing sternum.
   e. Patient has any movement to physical stimulation. (score –4)
   f. Patient has no response to any stimulation. (score –5)

XIII. Post-Sedation Monitoring

Patients undergoing procedural sedation must remain in a monitored setting with one to one nursing ratio until they reach an Aldrete score greater than or equal to 8 or equal to their presedation Aldrete score. On meeting criteria, patients discharged from the hospital setting after procedural sedation shall receive discharge instructions specific to the procedural sedation and be discharged to the care of a responsible adult.

ALDRETE SCORE:

	Respiratory:
2	Adequate respiratory volume and rate
1	Shallow, limited breathing, dyspnea
0	Apnea, no spontaneous exchange
	Circulatory:
2	B/P Stable, Pulse within Normal Limits
1	B/P fluctuating, pulse irregular, weak
0	Unable to Palpate B/P or Pulse
	Consciousness:
2	Fully Awake, Return of Reflexes
1	Arouses to Name, Partial Return of Reflexes
0	No Response to Stimuli
	Activity:
2	Able to Move All Four Extremities
1	Able to Move Two Extremities
0	Unable to Move Extremities
	0_2 Saturation:
2	Able to maintain $Sa0_2$ > 92% on Room Air
1	Needs 0_2 inhalation to maintain $Sa0_2$ > 92% on room air
0	$Sa0_2$ <90% even with 0_2 supplement

Discharge Criteria = a value greater than or equal to 8. Exceptions per physician or pre- procedure deficit.

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Approvals:
CMH MEC: 5/1/18
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