Moderate Sedation and Analgesia Self-Study

A Professional Education Training Program

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Moderate Sedation and Analgesia
Self-Study

Learning Objectives

Upon completion of this course, the participant will be able to:

1. Describe the continuum of sedation and differentiate moderate sedation from deep sedation.

2. Discuss the pre-, peri-, and post- monitoring of patients receiving moderate sedation.

3. Review the pharmacological implications for sedation and analgesia in the monitoring of patients receiving moderate sedation.

4. Describe proper airway management of the patient receiving moderate sedation.

5. Identify complications and emergency measures associated with complications of moderate sedation.
MODERATE SEDATION AND ANALGESIA

Definitions

THE CONTINUUM OF SEDATION

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

- Individuals administering Moderate Sedation/Analgesia ("Moderate Sedation") should be able to rescue patients who enter a state of Deep Sedation/Analgesia.
- Those administering Deep Sedation/Analgesia should be able to rescue patients who enter a state of general anesthesia.

DEFINITIONS OF THE FOUR LEVELS OF SEDATION AND ANESTHESIA

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

2. Moderate sedation / analgesia is a drug-induced depression of consciousness during which patients respond purposefully to verbal commands (Note: Reflex withdrawal from painful stimulus is not considered a purposeful response) either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.
3. **Deep sedation** is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients may require assistance in maintaining a patent airway and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.

4. **Anesthesia** consists of general anesthesia and 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.

<table>
<thead>
<tr>
<th>What Moderate Sedation is not:</th>
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</thead>
<tbody>
<tr>
<td><strong>Pre-Medication:</strong></td>
</tr>
<tr>
<td>- Defined as a single dose prior to a medication.</td>
</tr>
<tr>
<td>- Medication is not TITRATED to effect as in moderate sedation</td>
</tr>
<tr>
<td><strong>Post-operative Pain Management:</strong></td>
</tr>
<tr>
<td>- Given for post-op pain, including PCA</td>
</tr>
<tr>
<td>- Medication is not TITRATED to effect as in moderate sedation</td>
</tr>
</tbody>
</table>

**Goals of Moderate Sedation**
- Alteration of LOC and mood.
- Maintenance of consciousness and cooperation.
- Elevation of pain threshold.
- Minimal variation of vital signs.
- Rapid degree of ambulation.
- Safe and prompt recovery.

**Moderate Sedation should result in:**
- A patient that is relaxed, cooperative with:
  - Purposeful responses to verbal communication and instruction.
  - Purposeful response to tactile stimulation
  - Easy and prompt arousal from sleep.
PERSONNEL QUALIFICATIONS

Included in the qualifications of individuals providing moderate sedation is competency-based education, training, and experience in:

1. Evaluating patients prior to performing moderate sedation and analgesia and
2. Performing the moderate sedation to include methods and techniques required to rescue those patients who unavoidably slip into a deeper-than-desired level of sedation or analgesia.

Training and Credentialing

- Airway management training to include:
  - Oropharyngeal airways
  - Positive pressure ventilation devices, such as ambu bag
  - Cardiac Monitoring
  - ACLS
- Pharmacology training to include:
  - Dosages
  - Routes, Side Effects
  - Emergency management, including antagonists

Physicians and nurses who have appropriate qualifications are permitted to administer moderate sedation/analgesia and must be qualified to rescue patients from deep sedation. They must be competent to manage a compromised airway and to provide adequate oxygenation and ventilation.

Staff Qualifications

- Physicians must be granted clinical privileges for moderate sedation/analgesia through the hospital’s medical staff office.
- RN’s must be deemed competent to monitor and manage the care of patients receiving moderate sedation through the hospital’s competency assessment program.

Anesthesia professionals are permitted to administer all types/levels of anesthesia. Anesthesia professionals are qualified to rescue patients from general anesthesia. They are competent to manage an unstable cardiovascular system as well as a compromised airway and inadequate oxygenation and ventilation.
The goals of moderate sedation are to maintain adequate sedation with minimal risk, relieve anxiety and produce amnesia and provide relief from pain and other noxious stimuli. There must be a registered nurse present who has been deemed competent through the hospital’s competency assessment and validation process to monitor and manage the care of sedated patient.

Staff Qualifications

- Physicians must be granted clinical privileges for moderate sedation/analgesia through the hospital’s medical staff office.
- RN’s must be deemed competent to monitor and manage the care of patients receiving moderate sedation through the hospital's competency assessment program.

EQUIPMENT AND SUPPLIES

Each designated area where sedation is administered must have emergency resuscitative equipment immediately available, which 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 cart
- Emergency drugs
- Defibrillator
- EKG monitor
- Blood pressure monitor
- Stethoscope
- Pulse oximeter
- ETCO2 (Capnography) if available
- Appropriate airways
- Suction device/suction catheters
- Oxygen source
- Nasal O2 cannulas / O2 masks
- Intubation equipment
- Positive pressure oxygen delivery device (bag-valve-mask)
- IV supplies
PRE PROCEDURE PATIENT ASSESSMENT

Patient selection for moderate sedation is based on many factors. Patients who would not be ideal candidates include patients who are morbidly obese, have sleep apnea, significant cardiac and or pulmonary disease, significant impairment of kidney or liver function, a physical deformity that could compromise airway management, are mentally challenged, extreme of age or patients in whom moderate sedation alone is not expected to provide adequate conditions. An anesthesiologist should be consulted for the management of these patients.

Pre procedure patient assessment should include evaluation of the following:

- Relevant aspects of the patient’s medical history including abnormalities of the major organ systems, previous adverse experiences with sedation / anesthesia, current medications, allergies, weight, communication level, and pain assessment.
- ASA Physical Risk Classification. This assessment is performed by an anesthesiologist &/or a CRNA.

RN Responsibilities

- Verifying that pre-assessment findings are documented and informed consent is obtained and signed by patient.
- Ensuring that NPO status of patient is confirmed.
- Monitoring patient before, during, and after administration of moderate sedation.
<table>
<thead>
<tr>
<th>ASA Classification</th>
<th>Medical Description</th>
<th>Comment</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA I</td>
<td>No known systemic disease</td>
<td>May have moderate sedation without other consideration.</td>
<td>Healthy patient without evidence of systemic disease</td>
</tr>
<tr>
<td>ASA II</td>
<td>Mild or well controlled systemic disease</td>
<td></td>
<td>Patient who smokes with well controlled hypertension</td>
</tr>
<tr>
<td>ASA III</td>
<td>Multiple or moderate controlled systemic disease</td>
<td>Consider medical consultation.</td>
<td>Patient with diabetes and fairly stable angina</td>
</tr>
<tr>
<td>ASA IV</td>
<td>Poorly controlled systemic disease.</td>
<td>Consider involvement of Anesthesia for MAC</td>
<td>Patient with diabetes, angina and CHF with dyspnea on exertion and chest pain or Unstable patient not expected to survive without intervention</td>
</tr>
<tr>
<td>ASA V</td>
<td>Moribund patient</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASA VI</td>
<td>Declared Brain Dead</td>
<td>Organ removal for donation</td>
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</table>

- Focused physical examination including auscultation of the heart and lungs and evaluation of the airway.
- Pre procedure laboratory testing should be guided by the patient’s underlying medical condition and the likelihood that the results would affect the management of the sedation/analgesia. Adequate Hgb is needed for oxygenation.
- Vital signs to include blood pressure (BP), pulse (P), respirations (RR), and oxygen saturation (O2 sat).
- Level of conscious (LOC).
- NPO status.
NPO GUIDELINES

NPO Guidelines

- Sedation and analgesics tend to impair airway reflexes in proportion to the degree of sedation achieved.
- Aspiration is the most common cause of death associated with IV moderate sedation (1 – 20%).
- Mortality is significant with long hospital stays.

NPO guidelines specify the time-frames for patients to be restricted from consuming fluids or solids by the gastrointestinal route. This includes nasogastric and gastrostomy feedings. These are minimum NPO recommendations that apply to healthy (ASA 1 or 2) patients. These recommendations are not intended for women in labor.

NPO Guidelines

- Gastric emptying can be influenced by many factors, including anxiety, pain, pregnancy or mechanical obstruction.
- The suggested times do not guarantee gastric emptying.
- As a rule for adults, for solids and non-clear liquids, the patient should fast for 6 to 8 hours before the procedure.
## Universal NPO Guidelines

<table>
<thead>
<tr>
<th>Ingested Material</th>
<th>Minimum Fasting Period¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear Liquids²</td>
<td>2 Hours</td>
</tr>
<tr>
<td>Breast Milk</td>
<td>4 Hours</td>
</tr>
<tr>
<td>Infant Formula</td>
<td>6 Hours</td>
</tr>
<tr>
<td>Non-Human Milk³</td>
<td>6 Hours</td>
</tr>
<tr>
<td>Light Meal⁴</td>
<td>6 Hours</td>
</tr>
<tr>
<td>Meal⁵</td>
<td>8 Hours or More</td>
</tr>
</tbody>
</table>

Refer to Procedure No: SMH-IP 2317

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¹ Fasting period applies to all ages

² Examples of clear liquids include water, fruit juices without pulp, carbonated beverages, clear tea and black coffee

³ Since non-human milk is similar to solids in gastric emptying time, the amount ingested must be considered when determining an appropriate fasting period.

⁴ A light meal typically consists of toast and a clear liquid.

⁵ A meal that includes fried or fatty foods or meat might prolong gastric emptying time, both the amount of food and the type of food ingested must be considered when determine an appropriate fasting period.
AIRWAY ASSESSMENT

The administration of sedative and analgesic medications may interfere with the patient's ability to maintain a patent airway; therefore pre procedure evaluation of the lungs and airway is essential. The lungs should be assessed for any abnormal breath sounds such as rales or wheezing. The airway may be assessed by the physician using the Mallampati technique, which is used by anesthesia providers to determine possible intubation difficulty, information of value to the physician.

The Mallampati technique categorized the airway into one of four classes:

Class I: Visualization of the faucial pillars, soft palate and uvula.

Class II: Visualization of the faucial pillars and soft palate. The uvula is marked by the tongue.

Class III: Visualization of only the soft palate.

Class IV: Soft palate not seen.

If possible, the patient should be assessed in a sitting position. The patient is directed to open the mouth as wide as possible and protrude the tongue, exposing the faucial pillars and uvula at the tongue base. If the classification exceeds Class I, the physician should proceed with the appropriate plan of action. This simple precautionary measure alerts the physician to anticipate difficulty in the event of respiratory depression requiring intubation. For patients with a Class II, III and IV airway, a consultation with an anesthesia provider is recommended.
INFORMED CONSENT

The patient must be informed about the risks, benefits and alternatives to sedation as a component of the planned procedure. There must be documentation in the medical record of informed consent (Universal Protocol) and the plan of sedation prior to the procedure.

Here is a general list of risks and complications that could be associated with a sedative procedure:

**Relatively common and short-lived**
- Nausea and vomiting
- Prolonged sleepiness

**Uncommon and short-lived**
- Headache
- Excitability and agitation
- Dizziness
- Low blood pressure
- Nightmares

**Uncommon, but may last a short time**
- Sore lumpy vein (if medication given intravenously)

**Rare**
- Allergic reaction
- Diminished respiratory effort
- Inhalation of stomach contents
- Pneumonia

**Extremely Rare**
- Damage or failure of the heart, liver, stomach, kidneys and or brain
- Cardiopulmonary arrest
- Death

MONITORING

All patients receiving moderate sedation must be continuously observed and physiologically monitored by a designated qualified nurse throughout the sedation period. Assessment data, airway patency, respiratory rate, blood pressure and appropriate LOC and responsiveness (as per

RN Responsibilities

THE RN MONITORING THE PATIENT SHOULD NOT BE ENGAGED IN ANY OTHER ACTIVITY DURING THE PERIOD OF MODERATE SEDATION.
standardized tool) must be documented at least every 15 minutes or more frequently as indicated by the patient’s condition. The sedation period included the period of time during the administration of sedation until the patient has returned to pre-sedation status with regard to airway, breathing, circulation, and level of consciousness. The patients must be reevaluated immediately prior to the initiation of moderate sedation.

**USING CAPNOGRAPHY TO MONITOR VENTILATION**

End-tidal CO₂ is the amount of carbon dioxide in air at the end of exhalation. The measurement of exhaled CO₂ overtime is known as capnography with a waveform called a capnogram. During a procedure where moderate sedation is administered end-tidal CO₂ monitoring can help detect respiratory depression during and after the procedure. End tidal CO₂ monitoring can be done through a special nasal cannula. It is important to monitor the patient’s baseline CO₂ prior to sedation. Monitoring the end tidal CO₂ can help detect respiratory depression during and after the procedure is complete, before the patient returns to baseline level of consciousness.

Carbon dioxide (CO₂) is the most significant factor in monitoring ventilation. Capnography measures the CO₂ in every breath to monitor air exchange in the patient’s alveoli. Measuring her CO₂ levels during procedural sedation can detect problems in her lungs or airway and offers earlier warning of hypoventilation, respiratory depression, hypermetabolism, and hypoperfusion than monitoring SpO₂ alone. Capnography measures CO₂ with infrared technology and gives graphic and digital numeric displays for end-tidal CO₂ (ETCO₂) and SpO₂.

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**What the waveform tells you**

The capnograph waveform plots the patient’s CO₂ level on the vertical axis and time on the horizontal axis. The highest point represents end-tidal CO₂—ETCO₂—the concentration of CO₂ at the end of exhalation, which provides a clinical estimate of alveolar CO₂.

Follow these interventions for any change from baseline:
- Check the patient.
- Stimulate the patient.
- Consider withholding additional sedating medication.
- Inform the practitioner.
- Stop the procedure if necessary.
- Administer a reversal agent if necessary.

ETCO₂ Normal range between 35-40 mmHg
DOCUMENTATION

Careful observation of the patient is important and must include continuous assessment of the patient’s level of consciousness, oxygenation and ability to maintain protective reflexes. Changes in the patient’s condition will determine if the documentation interval frequency could be increased. Documentation in the medical record for sedative procedures should include at least the following:

- Informed consent, patient medical history, assessment data and evidence of appropriate monitoring
- Medications administered, patient’s response, any adverse reactions, and treatment of complications
- Recovery and discharge data
- Discharge instructions for outpatients and release to a responsible adult

Any significant adverse occurrence related to or occurring during a moderate sedation procedure must be reported to Risk Management by completing an incident report indicating the event occurred during a moderate sedation procedure.

Monitoring and Documentation

- Times: time patient was assessed prior to procedure, when initiated, when ended, and when medications given.
- LOC
- Adverse Reactions
- Ramsey scale applies to intra procedure monitoring.
- VS (HR, BP, O2 Sat, EKG, at a minimum every 15 minutes during and at end of procedure.
- Continuous monitoring of HR and O2 Sat, documented every 15 minutes until patient is back to presedation baseline
Ramsay Scale

| Score 1: | Anxious, agitated, and restless |
| Score 2: | Cooperative, oriented, tranquil |
| Score 3: | Responsive to commands only |
| Score 4: | Asleep but brisk response to light glabellar tap |
| Score 5: | Asleep, sluggish response to light glabellar tap |
| Score 6: | No response to light glabellar tap |

RECOVERY AND DISCHARGE

An objective scoring system will be used to assess the patient’s recovery from sedative effects and his or her eligibility for discharge from the procedure area or hospital. The Aldrete Post Anesthesia Scoring System (PARS) or the Modified Post Anesthesia Discharge Scoring System (MPADS) should be used to assess the patient for adequate recovery. A score of eight (8) or greater (PARS) and ten (10) for MPADS must be achieved to be eligible for discharge from unit where procedure and recovery is being performed.

| Activity | 0 = unable to lift head or move extremities |
| Respiration | 0 = apneic. Condition necessitates ventilator assisted respirations. |
| Circulation | 0 = has abnormally high or low BP (> 50% pre sedation level) |
| Neurological | 0 = not responding or responding to painful stimuli |
| O2 Sat | 0 = O2 saturation < 90% with O2 supplement |

<table>
<thead>
<tr>
<th></th>
<th>Post Anesthesia Recovery Score</th>
<th>PAR Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activity</td>
<td>1 = moves two extremities voluntarily or on command and can lift head</td>
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</tr>
<tr>
<td></td>
<td>2 = able t move four extremities voluntarily or on command. Can lift head</td>
<td></td>
</tr>
<tr>
<td>Respiration</td>
<td>1 = labored or limited respirations. May have mechanical airway</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 = can take a deep breath and cough well. Has normal respiratory rate and depth.</td>
<td></td>
</tr>
<tr>
<td>Circulation</td>
<td>1 = BP 20-50% of pre sedation level</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 = stable BP and pulse. BP 20% of pre sedation level</td>
<td></td>
</tr>
<tr>
<td>Neurological</td>
<td>1 = responds to verbal stimuli but drifts to sleep easily</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 = awake, alert, oriented to time, place and person</td>
<td></td>
</tr>
<tr>
<td>O2 Sat</td>
<td>1 = needs O2 inhalation to maintain O2 saturation &gt; 90% or &lt;95%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 = able to maintain pre procedure O2 saturation on room air or &gt; 95% on O2</td>
<td></td>
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</table>

Total Recovery Score
Discharge Criteria

The following criteria should be used to assess the patient’s readiness for discharge from the hospital. A physician is responsible for the discharge of the patient unless the patient meets specific discharge criteria that have been approved by the medical staff. Supportive documentation in the medical record should clearly reflect:

- The patient’s cardiovascular function and airway patency are satisfactory and stable.
- A score of eight (8) or greater (PARS) and ten (10) for MPADS must be achieved to be eligible for discharge.
- The patient is awake and protective reflexes are intact.
- The patient can walk and talk (age or pre-condition appropriate).
- The state of hydration is adequate.
- The patient is accompanied by a responsible adult.
- For a very young child or handicapped person, incapable of the usual responses, the pre-sedation level of responsiveness should be achieved.

Patients who are being discharged from the hospital following the procedure must be given discharge instructions that include at least the following:

- Anticipated or potential behavioral changes
- Limitations of activities or precautions
- Appropriate dietary precautions, if any
- Specific procedure-related instructions when indicated
- A 24-hour telephone contact number
- For outpatients: discharge instructions provided and released to a responsible adult.

### Modified Post Anesthesia Discharge Score (MPADS)

<table>
<thead>
<tr>
<th>Category</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital Signs</td>
<td>0 = within 40% or &gt; of pre-sedation levels&lt;br&gt;1 = within 20% - 40%&lt;br&gt;2 = within 20%</td>
</tr>
<tr>
<td>Pain</td>
<td>0 = severe (8-10)&lt;br&gt;1 = moderate (4-7)&lt;br&gt;2 = minimal / none (0-3)</td>
</tr>
<tr>
<td>Nausea &amp; Vomiting</td>
<td>0 = severe&lt;br&gt;1 = moderate&lt;br&gt;2 = minimal / none</td>
</tr>
<tr>
<td>Surgical Bleeding</td>
<td>0 = severe&lt;br&gt;1 = moderate&lt;br&gt;2 = minimal / none</td>
</tr>
<tr>
<td>Ambulation</td>
<td>0 = none / dizziness&lt;br&gt;1 = with assistance&lt;br&gt;2 = steady gait / no dizziness (age appropriate)</td>
</tr>
<tr>
<td><strong>Total Discharge Score</strong></td>
<td></td>
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</tbody>
</table>
PATIENT EDUCATION

Patients undergoing IV moderate sedation must not operate machinery or a motor vehicle for 24 hours or until the physician deems appropriate. Written discharge instructions are provided to the patient and family member. The patient/family is asked to verbalize their understanding of the instructions and sign for instructions.

It is important the following precautions be taken by the patient (or parent):

- The outpatient should not be left unattended. An adult should be with the patient for the remainder of the day.
- Adults should not drive or operate machinery, drink alcoholic beverages, or make any important decisions that day.
- Children may still be sleepy and nap longer than usual.
- The patient may resume their regular diet but it is recommended that the patient start slowly, progressing from water and bland liquids to jello and broth to prevent nausea and vomiting.
- The patient may resume their medications unless otherwise directed.
Benzodiazepines are a class of drugs very familiar to most nurses. These drugs are utilized on a daily basis for a number of reasons. The actions are predictable and patients' needs are met. The major side effect of these drugs is upon the respiratory system of the patient (Respiratory depression is a side effect; sedation is the expected effect.) The patient can develop a depressed ventilatory response to increasing carbon dioxide levels with subsequent falling levels of arterial oxygenation. Each 0.1 mg per kilogram of midazolam is said to reduce the body’s response to rising carbon dioxide levels by 50%. In addition, there is a rise in pulmonary airway resistance. As the patient’s level of consciousness decreases, the risk of respiratory insufficiency increases greatly. The patient’s compensatory responses are blunted, thus carbon dioxide levels will continue to rise and oxygen levels will continue to fall unless additional therapeutic measures are undertaken. It is imperative that the nurse administering these drugs be aware of this risk and continuously monitor the patient’s respiratory effort and oxygen saturations.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
<th>Onset</th>
<th>Duration</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam (Valium)</td>
<td>IV: 2.5 mg in increments, (not to exceed 5 mg per single dose over 60 seconds). (Individual response is variable.)</td>
<td>Onset: 30 seconds to 2 minutes (may take up to 5 min)</td>
<td>Duration: 60 – 180 mins (may last up to 4 hours); sedative effects usually last for 3 hours</td>
<td>Administer into a large vein; monitor airway, 02 sats and HR. Titrato to shurring of speech. Contraindicated in untreated narrow-angle glaucoma; irritating to veins-may cause phlebitis, thrombosis, and local inflammation. Avoid in pregnant women, esp. during first trimester.</td>
</tr>
<tr>
<td>Midazolam (Versed)</td>
<td>Healthy, Adults &lt; 60</td>
<td>IV: 0.5 to 2.5 mg over at least 2 minutes. Repeat in 2 minutes, if needed, in small increments of initial dose over at least 2 minutes to achieve desired effect. Overall dose 2.5-5 mg. Elderly: Initial dose 0.5 mg slow IV, give no more than 1.5 mg over 1 minute period, waiting another 2 minutes to evaluate sedative effect. Total dose &gt;3.5 mg is rarely necessary.</td>
<td>Onset: 1.5 to 5 minutes</td>
<td>Duration: 2 to 6 hours. Recovery usually occurs within 2 hours, but effects may last as long as 6 hours.</td>
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OPIOIDS

Opioids bind with specific receptors in the central nervous system. The action of each receptor type varies but all provide some level of analgesia and the main use of narcotics during conscious sedation is to provide the patient with some level of pain relief. Additionally, narcotics can produce sedation, and in higher doses, all will produce a profound decrease in the patient’s level of consciousness and a risk of respiratory arrest.

<table>
<thead>
<tr>
<th>Opioid</th>
<th>Description</th>
<th>IV Administration</th>
<th>Onset</th>
<th>Duration</th>
<th>Titration</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meperidine (Demerol)</td>
<td>Pure opioid agonist</td>
<td>Dilute to achieve concentration of 10mg/mL and administer 12.5 – 25 mg over 2 minutes. May repeat incremental dose at 2 minute intervals to achieve desired endpoint for sedation. Do not exceed 200 mg in 1 H. Do not exceed 600 mg. over 24 hour period. (It’s 1/10 as potent as morphine) Reduce dose in elderly or debilitated patients.</td>
<td>5 min</td>
<td>2 – 4 H</td>
<td>Titrate to slurred speech.</td>
<td>Contraindicated in patients with hypersensitivity to the drug and in those who have received an MAO inhibitor within the past 14 days. Normeperidine, a metabolite of meperidine is a CNS endotoxin. Patients with compromised renal function are particularly at risk. Meperidine should not be used for more than 48 hours for acute pain or at a dose greater than 600 mg/ 24 hours.</td>
</tr>
<tr>
<td>Morphine</td>
<td>Pure opioid Agonist</td>
<td>Dilute to achieve concentration of 1mg/mL. Administer 1 or 2 mg over 1 to 2 min. May repeat incremental dose at 5 minute intervals to achieve desired endpoint for sedation. Usual max total dose: 10 mg/24 hours. Reduce dose in elderly or debilitated patients.</td>
<td>5 min</td>
<td>4 – 5 H</td>
<td>Titrate to slurred speech and Ramsay Score of 3. Monitor respiratory rate and depth continuously; respiratory depression may occur. Be prepared to assist ventilation.</td>
<td>Contraindicated if drug allergy; use cautiously in elderly and debilitated patient. Hypotension is possible especially if the patient is hypovolemic. Nausea and vomiting may occur. Less nausea/vomiting versus meperidine.</td>
</tr>
<tr>
<td>Fentanyl (Sublimaze)</td>
<td>Opioid Analgesic</td>
<td>25 mcg to 50 mcg up to 150 mcg. Slow IV administration over 2 minutes.</td>
<td>1 – 2 min</td>
<td>30 – 60 min</td>
<td>Titrate to slurred speech. Monitor respiratory rate and depth continuously; respiratory depression may occur. Crosses the blood brain barrier quickly. With rapid administration, can cause skeletal muscle and chest wall rigidity impairing ventilation. More sedative effects when compared with morphine. Shorter acting when compared to morphine.</td>
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</tbody>
</table>
REVERSAL AGENTS

Although the benzodiazepines and the opioids used in IV moderate sedation and analgesia generally have a proven safety record and are highly effective within a wide therapeutic window, certain instances may require their reversal. Even with close monitoring, the potential exists for overdosing with benzodiazepines and opioids. Deleterious effects such as prolonged sedation, over sedation, airway compromise, and cardio pulmonary depression can result. In addition to treating the complications by the traditional methods, the RN may be required to administer a benzodiazepine or narcotic receptor antagonist in an attempt to reverse the effects of the benzodiazepine and/or opioid. Unfortunately, reversal agents carry with them the potential for adverse effects. To be able to provide total care for the patient, the RN must understand the side effects as well as the expected actions of these agents.

Reversal agents, such as Naloxone and Flumazenil, may be used for complete or partial reversal of opioid / benzodiazepine sedation which has resulted in the deterioration of the patient's clinical condition. A reversal agent should not be used as a routine drug at the conclusion of a sedative procedure. The duration of action of the sedative agent may exceed that of the reversal agent. Therefore, patients who have responded to the reversal agent should be carefully monitored, up to 120 minutes after administration, for resedation.

FLUMAZENIL

Flumazenil (Romazicon), synthesized in 1979 by Roche Laboratories, is a reversal agent that competes with benzodiazepines for the same receptor sites. As Flumazenil binds with benzodiazepine receptors, benzodiazepines are displaced and their effects are blocked. Depressed level of consciousness, psychomotor impairment, and other cognitive deficits are counteracted. Flumazenil does not consistently reverse benzodiazepine-induced amnesia. As Flumazenil removes the sedative effects of benzodiazepines, respiratory depression and hypoventilation improve indirectly. Flumazenil has no effect on respiratory depression if it has been induced by opioids.

Flumazenil should be titrated by the use of a series of small injections instead of a single large bolus. This enables the caregiver to awaken the patient gradually and minimizes the adverse effects. A dose of 0.2 mg IV is administered over 15 seconds. If the desired level of consciousness has not been achieved within 45 seconds, another dose of 0.2mg IV should be administered. This pattern should be continued until a maximum dose of 1 mg is reached.

The duration and degree of reversal depends on the amount of Flumazenil administered and the plasma concentration of the benzodiazepine. In healthy individuals, the half – life of Flumazenil is 0.8 hours. Since Flumazenil is extensively cleared by the liver, its half-life is prolonged to 2.4 hours in patients with severe hepatic disease. The same initial dose of Flumazenil should be
administered to patients with hepatic dysfunction, but subsequent doses should be reduced in size or frequency.

Most benzodiazepines have a half-life that is longer than that of Flumazenil. Therefore the potential for resedation exists as Flumazenil is metabolized. Sufficient amounts of benzodiazepines are left at the receptor site to cause resedation. Resedation occurs in 3% to 9% of all patients. It is most common in patients who have received high doses of benzodiazepine.

After administration of Flumazenil, the patient should be monitored for an adequate period (up to 120 minutes) based on the dose and the duration of the benzodiazepine used. This will allow the caregiver to identify resedation, respiratory depression, or other residual benzodiazepine effects.

The use of Flumazenil has been associated with the occurrence of seizures. Patients at risk for this complication are those who have been administered benzodiazepines for long-term sedation. Patients who are suffering from a serious cyclic antidepressant overdose are also at high risk for seizures. Flumazenil is contraindicated in cases of severe cyclic antidepressant poisoning. The patient should remain sedated and mechanically ventilated until signs of antidepressant toxicity have resolved.

### Flumazenil (Romazicon)

<table>
<thead>
<tr>
<th>Dosage</th>
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</thead>
<tbody>
<tr>
<td>Give IV 0.2mg over 15 seconds; may repeat dose every one minute for a maximum of 1 mg. For resedation, repeat 1 mg every 20 minutes (maximum 3 mg in one hour)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Onset</th>
<th>Duration</th>
<th>Half Life</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV: 1-2 min</td>
<td>IV: 45-90 min</td>
<td>41-79 min</td>
</tr>
</tbody>
</table>

**Flumazenil**

*Half Life*

- **Duration:** IV: 45-90 min
- **Onset:** IV: 1-2 min
- **Benzodiazepine Antagonist.**

- Use has been associated with occurrence of seizures. Monitor for recurrence of sedation. Monitor ventilation, HR, O2 sats.

- **NOTE:** The effects of Flumazenil may wear off before the effects of the benzodiazepine. Repeat doses may be required.

- Flumazenil is not generally recommended for use in children.
NALOXONE

Although the mechanism of action of Naloxone hydrochloride (Narcan) is not fully understood, studies suggest that it competes with narcotics for the mu, kappa, and delta receptors. By binding with these receptors, Naloxone counteracts the sedation, respiratory depression, analgesia, hypotension, and gastrointestinal stasis produced by opioids.

Naloxone is a versatile drug and can be administered via IV, intramuscular, intratracheal, or intralingual routes. In emergent situations the IV route is recommended because it has the most rapid onset of action. When Naloxone is administered intravenously, the onset of action is detected within 2 minutes. The dose of Naloxone should be titrated according to patient need for reversal of opiate-induced sedation.

Abrupt reversal of opiate depression has been known to cause tachycardia, hypertension, diaphoresis, nausea, vomiting, seizures, and cardiac arrest. By administering smaller doses at more frequent intervals, the caregiver can control the rate at which the opiate is reversed. Also, larger than necessary doses of Naloxone has the potential to completely reverse all analgesic effects, leading to the return of pain.

Naloxone is rapidly metabolized in the liver and is excreted in the urine. Duration of action varies (approximately 45 minutes) according to the route administered and depending on the dose of opiate received. Since Naloxone’s duration of action is shorter than that of many opiates, resedation may occur.

### Naloxone (Narcan)

- **Dilute an ampule (0.4 mg in 1 cc) in 9 cc of NS to create concentration of 0.04 mg/cc.**

- **Inject at 1-3 cc increments at 2-3 minute intervals to the desired degree of reversal:**
  - Adequate ventilation and alertness
  - No significant pain or discomfort
Patients should be observed for an adequate period based on the dose and duration of action of the opiate administered. Repeat doses of Naloxone may be required at 1 or 2 hour intervals.

If Naloxone is administered to patients who are physically dependant on opiates, the drug may precipitate acute withdrawal symptoms. Rare occurrences of hypertension, hypotension, pulmonary edema, and ventricular arrhythmias have been reported. These events occurred in postoperative patients who had a history of preexisting cardiovascular disease or who received medications with similar detrimental cardiovascular effects. Although the complications observed have not been directly attributed to Naloxone, caution should still be used with these patient populations.

<table>
<thead>
<tr>
<th>Naloxone (Narcan)</th>
<th>ADULTS</th>
<th>Onset: 2 – 3 min Duration: 45 min to 4 H Half-life Adults - 30-80 min Neonate: 2.5-3.5H</th>
<th>Narcotic antagonist. Contraindicated in patients with hypersensitivity to Naloxone. NOTE: The effects of Naloxone may wear off before the effects of the narcotic. Repeat doses may be required. Naloxone does not reverse and may even exacerbate hyperexcitability response associated with normeperidine toxicity.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Narcotic Antagonist</td>
<td>IV: 0.4 to 2 mg over 2 minutes with repeated doses at 2 to 3 minute intervals. Max total dose = 10 mg</td>
<td></td>
<td></td>
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</tbody>
</table>


PREVENTION OF COMPLICATIONS

The key to prevention of overdose and other potential complications associated with IV moderate sedation and analgesia is vigilant monitoring. In addition to the level of consciousness, principal patient physiological variables such as respiratory rate, oxygen saturation, cardiac rate and rhythm, and blood pressure should be assessed frequently. Early detection of a variation from the norm may avert the occurrence of an adverse effect (Stevens & White, 1995).

HIGH RISK PATIENTS

Through identification of high-risk patients, certain complications can be anticipated. Before the procedure the RN must assess the patient’s medical history to identify co morbidities that could predispose the patient to certain adverse events. The medical history regarding the patient’s response to sedation and analgesia will also assist the nurse in averting preventable complications.

Since older adults have a larger proportion of fat to total body weight, highly lipophilic sedatives such as benzodiazepines have prolonged elimination half-lives in such patients. Therefore reducing the dose of benzodiazepine in patients older than age 60 by 50% should be considered. Because of the potential for prolonged sedation, the duration of post procedure monitoring should be also be lengthened. On the other end of the age spectrum, children with certain cardiopulmonary disorders also require special attention since they may undergo desaturation rapidly after sedation.

Benzodiazepines are metabolized by the liver. Any cause of liver dysfunction, such as structural liver disease, congestive heart failure, or shock, can result in the slower elimination of benzodiazepines. To avoid prolonged sedation, the dose of benzodiazepines administered to patients with hepatic disease should be monitored. Since patients with renal disease exhibit little change in clearance, no dose adjustment is necessary.

Other patient populations at increased risk for complications from IV moderate sedation and analgesia include those with chronic cardiac or pulmonary dysfunction. Because chronic obstructive pulmonary disease (COPD) limits respiratory reserve, the respiratory depressive effect of benzodiazepines is exaggerated. In patients with congestive heart failure normal doses of benzodiazepines may cause cardiac depression from medullary vasomotor depression. Finally, alcoholic patients and other substance abusers are at risk for having the desire for intoxication stimulated by the use of benzodiazepines in IV moderate sedation.
Obese patients who are 30% above their ideal weight are also at increased risk for complications during IV moderate sedation and analgesia. A larger body mass requires a higher cardiac output to perfuse all tissues. An elevated metabolic rate creates more waste production (carbon dioxide) at the cellular level. For excretion of a larger amount of carbon dioxide, a higher rate of ventilation must occur. Therefore these patients may not be able to tolerate a depressed respiratory rate during IV moderate sedation and analgesia. In addition, the risk for aspiration is increased because of abdominal compression of the lungs in the supine position. An anesthesiologist should be consulted for the management of high-risk patients.

NEUROLOGIC COMPLICATIONS

OVERSEDATION

The RN must recognize that sedation is dose dependent and occurs along a continuum beginning with moderate sedation and analgesia. As the dose of sedation increases, the patient progresses toward general anesthesia. As the level of sedation deepens, hypnosis, loss of motor coordination, ataxia and confusion occur. Ultimately the patient becomes obtunded and stuporous and cannot be aroused. As the patient becomes over sedated, he or she becomes flaccid and cannot exhibit any behavioral symptoms of pain. Unfortunately, if the level of analgesia is inadequate, the over sedated patient may still be experiencing pain.

Careful assessment and rapid intervention will prevent overdose. To monitor the patient’s level of comfort and consciousness, verbal interaction with patient should be maintained during the procedure. At no time should the patient lose consciousness. The opioid and the benzodiazepine should both be carefully titrated to the desired end-point of analgesia and sedation. Although it is more time consuming to administer smaller doses more frequently, larger doses given at less frequent intervals tend to overshoot the goals of IV moderate sedation and analgesia. Over sedation occurs with the accumulation of drug from multiple large doses of medication not cleared quickly enough from the system. In the event that a loss of consciousness occurs, the RN should immediately notify the physician, maintain a patent airway, and anticipate administering reversal agents to antagonize the effects of the benzodiazepines and/or opioids.

UNDERSEDATION

Under sedation can be just as devastating a complication as over sedation. During frightening or uncomfortable procedures patients are under stress both mentally and physically. Mental and physical stress causes the activation of the fight-or-flight response from the autonomic nervous system. This is a defensive reaction that assists the body in physiologically coping with noxious stressors. During the fight or flight response, catecholamines are released. Catecholamine
increase heart rate, cardiac output, and blood pressure in an attempt to provide more blood flow to vital organs to allow the body to withstand any deleterious assault. During the fight-or-flight response, the heart works more vigorously, and myocardial oxygen consumption is increased, potentially causing myocardial ischemia. If sedation is ineffective, the patient will exhibit signs of nervousness and anxiety more than 50% of the time. Additional sedation is beneficial if the patient continues to communicate clearly and succinctly. The patient should be sedated until speech is slightly slurred. However, the patient should still be able to follow all verbal commands.

Pain exacerbates anxiety and anxiety exacerbates pain. Therefore, in addition to undersedation, the under treatment of pain during IV moderate sedation and analgesia can initiate the same detrimental stress response. Additional analgesia is required if the patient verbalizes discomfort, resist, grimaces, or pulls away during procedure.

PARADOXICAL REACTIONS

A small number of patients experience paradoxical reactions to moderate sedation and analgesia. Agitation and dysphoria may occur instead of sedation and relaxation. Although such paradoxical reactions are more common in the pediatric population, adults can also respond in this manner. Case studies involving incoherent shouting and agitation have been reported in adults immediately after the administration of Benzodiazepines. Before the patient’s paradoxical reaction is assumed to be a response to a certain medication, other potential causes must be ruled out. Under medication for pain may be a cause for agitation and restlessness. This may not be recognized in patients unable to verbalize their pain.

Over sedation can cause hypoventilation, potentially resulting in tissue hypoxia. Cerebral hypoxia or anoxia will initially lead to agitation followed by a decrease in level of consciousness and coma. While the patient is agitated, the RN should protect him/her from injury, ensure a patent airway, and maintain adequate vital signs. While attempting to identify the cause of a paradoxical response to sedation, the RN should check the patient’s cardiopulmonary and pain status before assuming that additional sedation will correct the problem. Additional sedation in a hypoxic patient will result in general anesthesia and the need for life support.

PULMONARY COMPLICATIONS

AIRWAY OBSTRUCTION

In addition to neurological complications, sedative-analgesic combinations used during IV moderate sedation and analgesia can also cause a variety of pulmonary disorders. In addition to altering protective reflexes that assist in
maintaining a patent airway, benzodiazepines and opioids can produce a loss of submandibular muscle tone. The submandibular muscles provide direct support of the tongue and indirect support of the epiglottis. The tongue may be displaced posterior and occlude the airway at the level of the pharynx; the epiglottis may occlude the airway at the level of the larynx. The tongue, epiglottis or both can occlude the entrance of the trachea. In the unconscious patient the tongue is the most common cause of airway obstruction. If the airway is not cleared, hypoxia and cardiopulmonary collapse will soon follow.

To identify and treat an obstructed airway, the RN carefully observes chest and abdominal movement for coordination with ventilatory efforts. Movement of air at the nose or mouth area should be verified. With partial airway obstruction the patient may exhibit a weak, ineffective cough, a high-pitched noise while inhaling, uncoordinated attempts at ventilation and cyanosis. With complete airway obstruction the movement of air is absent. Cyanosis and hypoxia will rapidly result if not treated.

The airway should immediately be restored via the head tilt – chin lift maneuver. The goal is to anteriorly displace the mandible. One hand is placed on the patient’s forehead, and pressure is applied to tilt the head back. The fingers of the other hand are placed under the bony part of the jaw and lifted to bring the chin forward, assisting the head in tilting back. In many cases the patient will be able to maintain spontaneous respiration with proper positioning.

When airway obstruction persists despite maximal mandibular displacement, insertion of a nasopharyngeal artificial airway may be necessary. However, this device must be used with caution since it might precipitate laryngospasm and vomiting in a semi moderate patient. An oropharyngeal airway should never be used in a semi obtunded patient because of the great risk of laryngospasm. After inserting the airway, the RN should continue to monitor for spontaneous

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**Head Tilt Chin Lift**

- Left hand is used to apply pressure to the forehead to extend the neck.
- Right hand is used to elevate the mandible which will lift the tongue from the posterior pharynx.
respirations. If spontaneous respirations are absent, artificial positive pressure ventilation should be initiated.

**OROPHARYNGEAL AIRWAY**

The oropharyngeal airway is essentially a curved hollow tube that is used to create an open conduit through the mouth and posterior pharynx.

The guide for correct size is as follows: hold the airway beside the patient's mandible, orienting it with the flange at the patient's mouth and the tip at the angle of jaw.

Starting with the curve of the airway inverted, insert the oropharyngeal airway and then rotate the airway as the tip reaches the posterior pharynx. If there are problems ventilating the patient after insertion of the airway then it should be removed and reinserted.

**ASPIRATION**

Aspiration is the most common cause of death associated with IV moderate sedation and accounts for 1% to 20% of all deaths involving sedation and/or anesthesia. Although complications arising from the aspiration of gastric contents may consist of only bronchospasm, most patients suffer from more serious disorders such as pneumonitis or hypoxia-induced multi organ failure. Therefore mortality after aspiration is significant, ranging from 5% to 70%. Patients who survive tend to experience prolonged and costly stays in the hospital. The average hospital stay is 21-28 days, with the majority of time spent in the intensive care unit.

Regurgitation occurs when gastric contents pass from the stomach across the gastro esophageal sphincter into the esophagus and the pharynx. If the larynx is incompetent, gastric contents may be aspirated into the lungs. The likelihood of aspiration is increased with IV moderate sedation and analgesia because protective reflexes, such as the cough and gag reflex may be impaired or absent with excess sedation.

Symptoms of aspiration depend on the extent of pulmonary injury and the material aspirated. Bronchospasm occurs initially because of reflex airway closure. Gastric contents are toxic, causing disruption of the alveolar capillary
membrane. A compromised alveolar capillary membrane may result in atelectasis, noncardiogenic pulmonary edema and even adult respiratory distress syndrome (ARDS). The patient may exhibit all or few of the following common symptoms: wheezing, crackles, coughing, hypoxia, pulmonary edema, cyanosis, fever and hyperventilation.

Prevention is the best way to address gastric aspiration because the majority of associated complications are severe. Prevention of gastric aspiration begins with the identification of high risk individuals. The risk of aspiration during procedure is elevated in any patient undergoing IV moderate sedation and analgesia. This risk escalates for pregnant patients, obese patients, and older adult patients with hiatal hernias or gastro esophageal reflux.

If nausea and vomiting are prevented, the risk for gastric aspiration is reduced; Opioids can induce regurgitation because they stimulate the vomiting center in the medulla. The vomiting center controls the motor impulses required to vomit. Therefore patients receiving benzodiazepines and opioids during IV moderate sedation and analgesia are already at risk for nausea and vomiting. Those at increased risk for nausea and vomiting include children, obese patients, patients with a history of nausea and vomiting associated with sedation, and patients with a history of motion sickness. Reports have also implicated unrelieved pain as a causative factor in post procedure nausea and vomiting. As the pain was treated, the feeling of nausea subsided. In addition, gastrointestinal secretions and decreased gastrointestinal motility can also contribute to nausea and vomiting. To reduce the incidence of nausea and vomiting, the patient should have nothing by mouth (NPO) according to recommended guidelines.

Anti emetics are most effective when administered before the sedative-analgesic combination. The side effects of most antiemetic drugs are drowsiness and sedation. Therefore, unintentional over sedation may result when antiemetics are given with high doses of benzodiazepines and opioids. The RN must be aware of the potential complications that can result from the concurrent use of antiemetics, benzodiazepines, and opioids. To prevent aspiration the patient’s level of sedation should be vigilantly assessed. This will avert over

### Aspiration

- If the patient vomits:
  - Turn to side
  - Suction airway
- Afterwards, may only require observation and supplemental O2.
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sedation and the consequent loss of protective airway reflexes. If the patient begins to vomit during the procedure, immediate placement on his/her side and elevation of the head will decrease the risk of aspiration. The airway should immediately be cleared of vomitus with a rigid pharyngeal catheter (Yankauer) and suction.

Immediately after aspiration, the patient may only require maintenance of a patent airway, oxygen, and observation. Patients with more severe aspiration may require intubation and mechanical ventilation. Therapy may range from the administration of supplemental oxygen to the use of continuous positive airway pressure. Early and aggressive treatment of aspiration may minimize secondary compliance that may arise, such as infections, abscesses, and fistulas.

RESPIRATORY INSUFFICIENCY

Before pulse capnography, oximetry and vigilant respiratory monitoring became routine, mishaps resulting from inadequate ventilation were the most common complications observed during moderate sedation and analgesia and/or anesthesia. Complications caused by respiratory depression were frequent because the respiratory depression was not recognized until it progressed to respiratory arrest, dysrhythmias, or cardiac arrest. When used in combination, benzodiazepines and opioids are synergistic in their respiratory depressant effect. Causes of compromised respiratory function include a direct depression of respiratory drive in response to hypoxia and diminished ventilatory response to hypercarbia. In addition, a reduction of muscle tone leading to a weaker ventilatory effort may result in a ventilation and perfusion mismatch. Respiratory effects caused by benzodiazepines and opioids are dose related. With an overdose the patient can progress from hypoventilation to apnea. The impact of respiratory function is pronounced in patients with a lesser respiratory reserve such as older adults and those with COPD.

Vigilant physical assessment of the patient’s respiratory effort and the use of pulse oximetry and capnography allow for the rapid identification and treatment of respiratory compromise. Observe the patient’s respiratory rate, pattern, and tidal volume. The patient’s breathing should be regular. Thoracic and abdominal ventilatory muscles should be working in coordination to produce deep breaths (10-15ml/kg). Breath sounds should be clear on auscultation. This assessment will identify any muscular difficulties the patient exhibits during ventilation. Hypoventilation may result in hypercapnia since respirations become sluggish and shallow, allowing carbon dioxide to accumulate in the bloodstream. Hypoxia may occur concurrently because the patient is not inspiring enough oxygen to maintain normal tissue oxygenation. Hypoxia and hypercapnia can both cause cell death, depresses mental activity, and a reduced work capacity of the muscles.
Pulse oximetry and capnography are an adjunct in monitoring a sedated patient’s respiratory status. Since arterial oxygen saturation (SaO2) are displayed continuously, hypoxia is identified earlier than by physical assessment alone. Although the following are late signs of inadequate ventilation and oxygenation, they assist the caregiver in identifying severe hypoxia and hypercapnia. Signs and symptoms include a decreased level of consciousness, depressed and shallow respirations, use of accessory muscles, cyanosis (especially in the highly vascular areas such as the lips, nail beds, tip of nose, and underside of the tongue), diaphoresis, nasal flaring, and agitation.

If the patient becomes apneic, a bag-valve device consisting of a self-inflating bag and a non-rebreathing valve may be required until the patient can be emergently intubated and mechanically ventilated. For adequate ventilation and oxygenation, the caregiver must attain a seal around the patient’s face that forces all oxygen into the lungs. The caregiver is positioned near the patient’s head. An open airway must be maintained under the mask through the head tilt-chin-lift maneuver. The mask is applied to the face while the left hand keeps a tight seal around the face. The bag is compressed with the right hand and 15 breaths per minute are administered. To ensure proper technique, the chest is observed for rising and falling during ventilation.

CARDIOVASCULAR COMPLICATIONS

HYPOTENSION
Cautious monitoring of the cardiovascular system provides early warning of deleterious side effects that may be caused by drugs used during IV moderate sedation and analgesia. Benzodiazepines and opioids are synergistic in their impact on the cardiovascular system. Direct actions on peripheral circulation cause vasodilatation with resultant hypotension. With vasodilatation blood pools in the vasculature and is not returned to the myocardium, causing a decrease in cardiac output. The decreased cardiac output may be severe enough to cause cell ischemia and necrosis resulting from an inability to deliver oxygen and nutrients to the tissues. This hypotensive state may be extremely serious in the hypovolemic patient, causing a state of shock. Untreated hypoxia will compound the vasodilatory effects of the drugs and result in a more serious hypotension. Vasodilatation occurs in response to hypoxia to increase the surface areas for gas exchange. Both responses attempt to augment oxygen delivery to the hypoxic tissues.

In response to hypotension due to vasodilatation, the cardiovascular system attempts to compensate with the autonomic nervous system’s fight-or flight response. Endogenous catecholamines such as epinephrine and nor epinephrine are released to increase the heart rate and force of contraction. Those patients with cardiovascular disease must be more vigilantly observed because they may not have enough cardiac reserve to mount a response to
hypotension.

Careful titration of benzodiazepines and opioids can prevent or minimize any hypotension effects. Smaller and more frequent doses administered on an as-needed basis, instead of larger intermittent doses of benzodiazepines and opioids, decrease the occurrence of vasodilatation. To proactively identify cardiovascular complications, the patient’s cardiovascular vital signs should be assessed. The heart rate should be regular. The heart rate and blood pressure should be within normal limits for the patient. Current vital signs should be compared to the patient’s baseline. The patient’s pre procedure blood pressure might not be representative of an average blood pressure because it might be elevated as a result of anxiety. The patient’s peripheral perfusion should be assessed by observing skin color and capillary refill of nail beds. After the procedure the patient should be assisted to first dangle, then sit and then slowly stand to prevent the occurrence of orthostatic hypotension. The patient’s blood pressure should be checked in the various positions to ensure hemodynamic stability. If the systolic blood pressure drops by 15 mm Hg or more, the patient should be maintained in the last position until the blood pressure normalizes.

If the patient experiences severe and sustained hypotension, fluid therapy must be used to restore effective circulating blood volume. Crystalloid solutions such as normal saline or ringers lactate are commonly used to treat a hypotensive episode. Because fluids may be ineffective in extreme circumstances, vasoactive infusions may also be necessary. Supplemental oxygen will increase oxygen delivery to the tissues while the hypotension is being treated. Any hypoxia that is potentially contributing to the hypotension will also be reversed. Endo tracheal intubation and mechanical ventilation may be required if the hypotension is severe enough to depress the level of consciousness through inadequate perfusion of the brain.

MYOCARDIAL ISCHEMIA

Myocardial ischemia and injury are a result of an oxygen supply-demand mismatch either from a reduction in oxygen supply or from an increase in oxygen demand. Hypoxia leads to inadequate myocardial oxygenation because there is less oxygen in the bloodstream. Tachycardia also reduces oxygen supply to the heart because it decreases the time spent in diastole (the time when coronary arteries are perfused). Hypotension caused by vasodilatation also decreases oxygen supply to the heart because it reduces blood flow through the coronary arteries. In older adults with coronary artery disease, myocardial oxygen consumption can exceed oxygen delivery even with an adequate blood pressure. Anything that increases the workload of the myocardium, such as tachycardia and hypertension from pain and anxiety, increases the myocardial oxygen demand. Rapid assessment of myocardial oxygen supply and demand is imperative in the prevention of myocardial ischemia and infarction.
Chest pain is a symptom of myocardial ischemia and/or infarction. The pain may be described as pressure, tightness, heaviness, aching, crushing, squeezing, burning, viselike, constricting or suffocating. The substernal area is the usual location of chest pain, but it may also be retrosternal. The pain may radiate to the neck, jaw, teeth, back, shoulders, arms, elbows, and wrists, usually on the left side. The patient may also complain of palpitations from arrhythmias.

Knowledge regarding a patient’s significant cardiac history will assist in preventing myocardial ischemia and infarction. Oxygen delivery to the heart is always compromised in coronary artery disease because of decreased flow of blood through the constricted coronary arteries.

ARRHYTHMIAS

Ventricular dysrhythmias commonly occur during times of myocardial ischemia and infarction. If not promptly treated, ventricular dysrhythmias will deteriorate into cardiac standstill. The longer that cardiopulmonary compromise persists, the greater the probability of death. The use of electrocardiograph (ECG) monitoring during IV moderate sedation and analgesia allows early identification and treatment of these serious dysrhythmias.

An increased frequency of PVC’s, for treatment of this life-threatening dysrhythmias follow Advanced Cardiac Life Support (ACLS) Guidelines. These are recommendations representing a consensus of experts from a variety of disciplines for emergency cardiac care of patients.
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General Information Resources And Recommended Readings

a. American Society of Anesthesiologists Practice Guidelines for Sedation and Analgesia by Non-Anesthesiologists
b. Association of Operating Room Nurses Recommended Practices for Managing the patient Receiving Conscious Sedation and Analgesia
c. JCAHO: Revisions to Anesthesia Care Standards: Comprehensive Accreditation Manual for Hospitals
d. Insights into Conscious Sedation by Steven J Somerson, CRNA, American Journal of Nursing, June 1995
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