Learning Objectives

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

1. Summarize the continuum of sedation and the monitoring needs of each
2. Define the education, training, experience and skills required for physicians administering moderate sedation to the neonate.
3. Describe the current state of neonatal procedural sedation from the pre-sedation risk assessment to the recovery phase.
4. Determine the effects of common sedative drugs on consciousness, anxiety and respiratory drive.
5. Explain the most common adverse events that occur during sedation and how effectively to manage them.
Definitions

**Moderate Sedation**: a medically controlled state that:

1. allows protective reflexes to be maintained
2. retains the patient’s ability to maintain a patent airway
3. permits appropriate response by the patient to stimulation

**Neonate**: any patient cared for in the Neonatal Intensive Care Unit through discharge.

Overview

Infants are not small adults, particularly when it comes to medication administration and moderate sedation. Moderate sedation practices are influenced by the recommendations and guidelines established by the American Academy of Pediatrics (AAP), the American Society of Anesthesiology (ASA) and the Joint Commission.

Practitioners intending to sedate a neonate for purposes of performing a procedure will need to assess the patient to determine the infant’s ability to tolerate sedation. Existing disease states can make it a risky situation for the patient and the decision to sedate will rest with the provider to determine if the risk/benefit ratio justifies the potential complications. The level of sedation ranges from minimally impaired consciousness to complete unconsciousness, which is equivalent to general anesthesia. Those providers delivering sedation need to be able to recognize that different levels of sedation are possible and are not specific to a given drug.

These guidelines do not refer to the delivery of general anesthesia and monitored anesthesia care, nor do they address the sedation of mechanically ventilated infants or pre-intubation sedation in the NICU.

<table>
<thead>
<tr>
<th>Goals of Moderate Sedation</th>
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<tbody>
<tr>
<td>*Alteration of LOC</td>
</tr>
<tr>
<td>*Maintenance of consciousness and cooperation</td>
</tr>
<tr>
<td>*Elevation of the pain threshold</td>
</tr>
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<td>*Minimal variation of vital signs</td>
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<tr>
<td>*Safe and prompt recovery</td>
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</table>
Levels of Sedation

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, ventilator and cardiovascular functions are unaffected.

2. **Moderate sedation** 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 ventilator function is often impaired. Patients may require assistance in maintaining a patent airway and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained in the presence of adequate ventilation.

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 ventilator 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 may also be impaired.

Physician Qualifications

Physicians must be granted clinical privileges for moderate sedation procedures through the hospital’s medical staff credentialing process. The physician must:

- be skilled in the use of such techniques evidenced by completion of a training program in his/her specialty where the use of pharmacological agents for sedation was a routine part of performing those therapeutic or diagnostic procedures;
- be capable of managing complications and providing first line therapy which includes establishing an airway, administering positive pressure ventilation and managing cardiovascular emergencies;
- agree to comply with Baptist Health’s policy on Moderate Sedation.

Elements of the physician’s responsibilities include:

- performing a pre-sedation patient assessment of relevant information from the patient’s medical history;
- a focused physical examination with the determination of the patient’s current physical risk status;
→ developing a sedation plan;
→ obtaining informed consent;
→ evaluating patient’s level of consciousness, ventilation and oxygenation status, and hemodynamic variables.
Anatomical Differences

The most important aspect of safe sedation in the neonatal population is the ability to assess and manage the airway. The upper airway is composed of three segments: the supraglottic, laryngeal and intrathoracic area.

A. The supraglottic larynx is the most poorly supported and the most collapsible segment of the upper airway. It is comprised of the pharyngeal structures and is the most impacted portion of the airway during sedation

B. The larynx or glottis is comprised of the vocal cords, the subglottic area and the cervical trachea.

C. The intrathoracic consists of the thoracic trachea and bronchi.
Infants are more predisposed to airway obstruction during sedation because of their large tongue and "floppy" long epiglottis. Additionally, when they are positioned recumbently, their large occiput places the head in a naturally flexed position which further exacerbates airway obstruction.

Airway obstruction during moderate sedation occurs in the supraglottic area due primarily to the soft palate and epiglottis "falling back" to the posterior pharynx. It was previously thought the base of the tongue was the primary cause of upper airway obstruction during sedation or periods of unconsciousness, but MRI studies have shown the soft palate and epiglottis are the most likely structures causing pharyngeal obstruction.

The other primary cause of upper airway obstruction during sedation is laryngospasm which may be partial or complete. Laryngospasm is defined as a muscular spasm in the glottis area and has multiple risk factors which include airway secretions, airway manipulation, recent upper respiratory infection, gastroesophageal reflux disease, passive tobacco smoke exposure, use of an airway device, young age, and higher ASA classification. Complete laryngospasm can be differentiated from supraglottic obstruction by the lack of response of simple airway maneuvers.

**The Respiratory Drive**
The basic drive to breathe originates from within the central respiratory center located in the brainstem. Output from the respiratory center is modulated by a number of chemical (e.g., CO2, O2) and mechanical (e.g., lung mechanics) controllers. Changes in carbon dioxide concentration are among the most important determinants of respiratory drive from the medullary respiratory center.

**Adverse Events**
1. The vast majority of adverse outcomes during sedation are preceded by a respiratory event.
2. The greater the depth of sedation, the greater the risk of complications.
3. The majority of poor outcomes related to adverse sedation events are due to a rule violation or insufficient education and skills of the practitioner.
4. Adverse sedation events are not associated with either a specific sedative drug class or route of administration.

The keys to appropriately managing the neonatal airway during sedation are proper airway positioning and application of positive pressure ventilation when required. Routine neonatal airway management includes placing the patient’s head/neck in a sniffing position and administering blow-by oxygen. If obstruction persists, the following steps can be implemented:

- **M:** Adjust the mask on the face.
- **R:** Reposition the head to ensure an open airway and then re-attempt ventilation.

*If not effective:*
- **S:** Suction the mouth and nose
- **O:** Ventilate with the baby’s mouth slightly open and lift the jaw forward; re-attempt Ventilation
If not effective:
P: Gradually increase pressure every few breaths (cautiously and to a maximum of 40 cm H2O) until there are bilateral breath sounds and visible chest movement

If still not effective:
A: Consider airway alternative (endotracheal tube or laryngeal mask airway)

Pre-Sedation Phase – Risk Assessment
A pre-sedation assessment is essential to identify high-risk patient populations and anticipate/reduce adverse sedation events. Studies have shown that background knowledge and skills in resuscitation (particularly airway management), education in sedative pharmacology, and pre-sedation risk assessment reduce the frequency and severity of adverse events during sedation. All in all, the majority of preventable adverse sedation events tend to occur as a consequence of inadequate practitioner experience and skills (insufficient education) and violation of hospital policy and procedure (rule violation). In order to optimize performance and minimize risk during procedural sedation, the provider must understand all sedation aspects associated with the patient, the procedure, and the sedation provider.

Patient Factors
General History: Each patient must undergo a general physical exam that includes the airway and the respiratory and cardiovascular systems. This is to be done by a medical staff member who is credentialed to administer Moderate Sedation. The pre-sedation history should include the following:
1. Allergies and previous adverse drug reactions
   • Adverse reactions: it is important to note if a patient has experienced paradoxical reactions to sedative medications, such as chloral hydrate, in the past.
   • Allergies: an allergy history needs to be obtained prior to sedation with the type of reaction identified. Allergies are rarely seen in newborns.
2. Current medications
3. Sedation/anesthesia history with focus on complications and airway problems
4. Major medical illnesses, physical abnormalities and neurologic problems
5. Last oral intake of fluids and solids (see below)
6. Recent acute illnesses (e.g., upper respiratory infection, fever, etc.)
7. Relevant family history (e.g., anesthesia)
8. Review of systems with focus on pulmonary, cardiac, renal and hepatic functions

Focused System Review: Developmental, Cardiac, Pulmonary, Aspiration Risk
• Developmental: Neonates are considered at higher risk than older children but regardless of age, the neurodevelopmental status of the patient should be assessed. Sedation requirements will change for any patient who is severely delayed.
• Cardiac: Most congenital heart disease patients who are thriving will tolerate sedation without problems. However, some sedative drugs can significantly affect vascular resistance and may alter systemic and pulmonary blood flow in patients with large intra-cardiac shunts.
Additionally, the use of some sedation may increase pulmonary hypertension, hypoxemia, or hypercarbia in a cardiac patient.

- **Pulmonary:** Patients with reactive airway disease, upper respiratory tract infections, and Chronic Lung Disease need to be carefully evaluated prior to the use of sedation.

- **Aspiration Risk:** Prior to any sedation procedure, a history of the last oral intake needs to be obtained. Data associated with aspiration injury and neonatal sedation cases is not definitive; most experts advise fasting guidelines similar to those required for anesthesia. Neonatal sedation does not follow a clear cut sedation depth so it should be assumed that with any sedation, the airway reflexes may be lost necessitating steps be taken to minimize the risk. There are no national standard guidelines for fasting prior to sedation. Generally accepted guidelines differentiate between clear liquid intake and heavy meals graded as indicated in the table below.

<table>
<thead>
<tr>
<th>Food</th>
<th>Hrs. of Fasting Required</th>
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<tbody>
<tr>
<td>Clear liquids</td>
<td>2 hrs.</td>
</tr>
<tr>
<td>Breast milk</td>
<td>4 hrs.</td>
</tr>
<tr>
<td>Formula</td>
<td>6 hrs.</td>
</tr>
<tr>
<td>Solids</td>
<td>8 hrs.</td>
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</table>

Each provider needs to weigh the urgency of the procedure against the relative risk of the “full stomach.” In these circumstances, the risk/benefit ratio needs to be weighed prior to the procedure. The recommendation is to strive for a reasonable fasting interval when sedating neonatal patients, especially for elective procedures.

**General Physical Examination**

The general physician examination prior to a procedure requiring moderate sedation needs to include at a minimum—blood pressure, heart rate, respiratory rate, and oxygen saturation by pulse oximetry. The exam should focus on the upper airway, lungs, cardiovascular system, and baseline neurological status.

**American Society of Anesthesiologist (ASA) Classification**

The American Society of Anesthesiology (ASA) has developed a classification system for patients in order to aid in identifying the risks of sedation. This classification categorizes patients on a general health basis prior to receiving anesthesia. It is one of the most important factors used to assess the overall pre-sedation risk (see table below). Rather than focusing on specific disease processes, the ASA status is intended to group patients together based on their health status in order to assess the risk of sedation or anesthesia of a given patient. Multiple studies have documented the fact that sedation risk in children rises with increasing ASA scores. ASA 1 and 2 are considered low-risk and ASA 3 and 4 are high-risk patients.
Factors Relating to the Procedure

Procedure Duration
The sedation provider should consider the time required to accomplish the procedure when choosing a sedation medication or technique. The drug administered should provide sedation for enough time to complete the procedure. Directions for further dosing should be included.

Pain as a Procedural Side Effect
Medication choice with procedures involving pain needs to be considered as part of the sedation plan. Chloral hydrate and the benzodiazepines have no analgesic component; pain will be felt if these medications are used alone and the patient will move and thrash about if pain is experienced. Analgesic medications such as fentanyl will provide pain control, but will not provide sedation. In general, analgesic medications should be included if the procedure is going to be painful while they may be omitted for non-painful procedures.

Procedural Positioning
The average neonate will maintain an open airway in the supine position even when deeply sedated as long as the neck can be slightly extended. It is much more difficult to maintain an airway when the head is flexed for a procedure such as a lumbar puncture or a scan. Care needs to be taken in these situations not to perform deep sedation unless the provider is ready to place an oral airway or endotracheal tube. Generally when neonates are placed on their side or in a prone position, the airway is at least as easy, or possibly even easier, to maintain than when in a supine position. Lastly, the provider must take into account airway management that must be provided remotely (e.g., MRI) as it will not be possible to adjust the airway and assist ventilation until the procedure is completed.

Availability of Rescue Resources
Personnel and equipment must be readily available and equipment should be in good working order for sedation procedures. Sedation critical incidents have shown that the worst outcomes for unexpected apnea events occur when rescue is not readily available. For this reason, defined areas have been identified as acceptable for providing moderate sedation.

Factors Relating to the Provider

Dedicated Person to Monitor Sedation
In addition to the practitioner providing the sedation, the use of moderate sedation needs to include a person whose responsibility is to monitor appropriate physiologic parameters and to assist in any supportive or resuscitative measures as needed. This person needs to be trained in NRP.
**Skills Related to Sedation**

The Joint Commission recommends that the provider must have the skills necessary to “rescue” a patient from sedation consequences one level “deeper” than that intended. The provider must be able to rescue a neonate from “deep” sedation by performing effective bag-mask ventilation with possible intubation. The Medical Staff Office maintains sedation competencies for all sedation providers.

**Back-up Systems and Ability to Rescue**

Studies of sedation related critical events have shown that sedation accidents are clearly most common in instances where a good back-up system is not available. The NICU team needs to be clearly identified and the physician should be available to help in the event of an emergency.

**Intra-Sedation Phase**

**Prior to the Procedure**

A clearly worded informed consent needs to be obtained from the legal guardian any time sedation medications are to be given to a neonate. This is in addition to the informed consent for the procedure itself, when required. The consent needs to include the risks, benefits, and alternatives. The parent or guardian should be educated regarding the risks and potential adverse effects of the sedation, the anticipated sedative effects, the reason for sedation, and potential options.

Universal protocol, which includes a time out, refers to the active process of verifying the correct patient, correct procedure, correct site, correct position, and correct equipment by those in attendance during the procedure and should include the family, if present. The “time out” must be performed by those in attendance prior to the procedure. The family, if present, should be included in the identification process by stating the name and date of birth of the patient.

**Equipment Needs**

Prior to beginning every moderate sedation procedure, key pieces of equipment are crucial to the safe care of a sedated neonatal patient. Confirm that the equipment is size appropriate for the patient and in good working order. A good mnemonic to help remember what equipment is needed is SOAPME.

- **S (suction):** appropriate size suction catheters and a functioning suction apparatus
- **O (oxygen):** adequate oxygen supply and functioning flow meters
- **A (airway):** appropriate size airway equipment, e.g., oropharyngeal airways, laryngoscope blades, endotracheal tubes, stylets, facemask, bag-valve-mask or equivalent. Laryngeal mask Laryngeal mask airways (LMA’s) have become increasingly popular for airway management during management during anesthesia and in emergency situations. The smallest size (1) should be available to aid anyone familiar with their use, particularly with difficult to intubate airways.
- **P (pharmacy):** all the resuscitation drugs needed for an emergency, sedatives, and sedative antagonists
M (monitors): pulse oximeter with size-appropriate probes, EKG, non-invasive blood pressure, end-tidal carbon dioxide (if available). The current American Academy of Pediatrics guidelines does not specifically require a particular set of monitors. They do state, however, the “vital signs, including oxygen saturation and heart rate, must be documented every 5 minutes in a time based record.”

E (extra equipment): special equipment or drugs for a particular case (e.g., stethoscope)

Monitoring

Management of the Infant Receiving Moderate Sedation

In order to promote the highest degree of safety and sedation effectiveness, the effects of drugs administered needs to be monitored. The most important monitoring tools are those to assess breathing as the most significant effect sedatives have are on airway control and the respiratory drive. As the depth of sedation increases, so does the risk, particularly to the respiratory system. Consequently, assessing the level of consciousness during sedation is a key part of the monitoring process.

Respiratory Monitoring

Monitoring Gas Exchange

Monitoring the patient’s respiratory status is imperative to insure the patient is safe during sedation. Pulse oximetry and capnography are the two primary methods used to assess gas exchange during sedation; they are non-invasive and practical alternatives to arterial blood gas analysis, which remains the gold standard to assess gas exchange.

Monitoring Oxygenation

Pulse oximetry needs to be utilized in all infants undergoing moderate sedation. This is a non-invasive method of measuring the saturated hemoglobin in the tissue capillaries and is a useful tool for determining oxygenation. It works by transmitting a beam of light through the tissue to a receiver. As the amount of saturated hemoglobin alters the wavelengths of the transmitted light, analysis of the received light is translated into a percentage of oxygen saturation in the blood. It is important to note these only assess oxygenation and not carbon dioxide elimination. It is important that the probe be positioned on the infant prior to turning the machine on or plugging in the cable, as the monitor attempts to calibrate to the patient/site when powered and plugged. The neonate’s normal baseline saturation should be maintained (with supplemental oxygen). Pulse oximetry requires pulsatile blood flow to determine oxygen saturation. Pulse oximeters vary in accuracy and signal strength in the presence of poorly perfused tissues. Low or absent pulse signals will occur under these circumstances. Similarly, when the patient is actively moving, the pulse oximeter may have difficulty distinguishing pulsatile tissue beds from movement-induced artifact. There is typically a time delay of at least 15-20 seconds between change in oxygen saturation and its detection by a pulse oximeter. Thus, evidence of oxygen desaturation by pulse oximetry gives a comparatively late warning of hypoxia. Placement of the pulse oximeter probe in a more central location may reduce the delay in the determination of oxygen
desaturation. Finally, pulse oximetry does not provide direct information regarding ventilation. Consequently, CO2 and pH are not assessed with pulse oximetry.

Some hints to remember with pulse oximetry:
- Improve circulation by warming cold extremities
- Don’t use on the same extremity as an arterial line, blood pressure cuff, or tourniquet
- Place sensor so the light beams and photo sensor are opposite each other
- Avoid placing sensor on an area that is frequently moved
- Protect sensor from bright external light sources by covering the sensor and the limb
- Place probe on infant prior to plugging it into the monitor to improve accuracy

Monitoring Ventilation
The pulse oximeter gives information about oxygen saturation, but does not give the status of the patient’s ventilation or exchange of CO2. Also, there is a significant delay between apnea and a change in the pulse oximeter reading. A patient may be apneic for 30 seconds (size dependent) before the oxygen saturation changes. The American Academy of Pediatrics guidelines recommend the use of capnography to aid in monitoring ventilation. Capnography is a non-invasive monitoring tool that measures CO2 concentrations in the patient’s exhaled gas and is displayed continuously as a waveform through the respiratory cycle. Capnography can be utilized during sedation to assess real time breath-to-breath analysis of carbon dioxide with delay in CO2 changes of approximately 0.25 seconds, however, it is not required. Capnography is superior to pulse oximetry in detecting acute ventilation changes, diagnosing apnea and reducing hypoxemic events. Hypoventilation is the most common cause of an elevated end tidal carbon dioxide level during sedation. Air exchange can be confirmed with each breath using capnography and apnea can be detected as soon as it occurs. It is important to note that the amount of air moved by an infant may make it difficult to assure accuracy of the actual value displayed by the capnography monitor and that it is more reasonable to assess the trend of the value rather than the actual number displayed by the monitor.

Components of the normal capnogram
- A – (near zero baseline) Exhalation of CO2 free gas contained in dead space
- B – (rapid sharp rise) Exhalation of mixed dead space and alveolar gas
- C – (alveolar plateau) Exhalation of mostly alveolar gas
- D – (rapid sharp down stroke) Inhalation

Monitoring Cardiovascular Status
Standard sedation monitors include EKG, pulse oximeter, and non-invasive blood pressure. The EKG gives heart rate and rhythm information and can be used to confirm pulse oximeter accuracy. Blood pressure is most helpful for deep sedation; the cuff cycling may disturb the patient and may actually inhibit sedation effectiveness during mild or moderate sedation procedures.
Overview of Sedation Medications – General Approach to Sedation

The four primary goals of neonatal procedural sedation include maintaining patient safety, providing effective pain control, reducing anxiety and psychological stress, and promoting conditions conducive to successful performance of the procedure.

Sedative drugs are medications that may result in central nervous system depression. These drugs may lead to loss of protective reflexes with resultant respiratory and/or cardiac dysfunction. The clinical effects of the medications used to achieve sedation are dose-related and need to be individually assessed for each infant. Sedative drugs may be administered orally, intranasally, rectally, parenterally, or by inhalation. Specific types of sedatives can be further defined by their clinical effect. Some of the more common definitions of sedative drugs include:

- **Sedative**: decreases activity, moderates excitement and calms the patient
- **Hypnotic**: produces drowsiness and assists the onset and maintenance of sleep
- **Analgesic**: relieves pain
- **Anxiolytic**: relieves apprehension and fear due to an anticipated act or illness
- **Amnesic**: affects memory so the patient is unable to recall events following the drug delivery

All sedative drugs have a dose dependent effect on central nervous system depression. The most common serious adverse effects associated with sedation are loss of airway control and respiratory depression; the greater the sedation, the greater the degree of respiratory depression. The risk of respiratory depression increases when sedatives are combined or when one drug is given in large doses.

**Sedation Medications**

The following section addresses commonly used neonatal sedation medications. They are identified based on the predominant characteristic feature of the sedative drug:

- **Anxiolytics** – drugs that reduce anxiety
- **Sedative-Hypnotics** – drugs that result in sleep
- **Sedative-Analgesics** – drugs that have analgesic properties

**Primary Sedative – Anxiolytic Drugs**

Anxiolytic drugs make infants comfortable and easier to work with. Benzodiazepines are the most common drugs used in this category. They are particularly useful for non-invasive procedures or distressful procedures that do not require the infant to be immobile. They are also useful as pre-medications and as adjuncts to analgesics. They are not analgesics when used alone and are poor hypnotics.
Benzodiazepines (BNZ): Diazepam, Midazolam, Lorazepam

<table>
<thead>
<tr>
<th>Benzodiazepine</th>
<th>Dose</th>
<th>Repeat Dose</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam</td>
<td>0.1-0.15 mg/kg</td>
<td>0.05-0.1 mg/kg Q 3-5 min.</td>
<td>&lt;60 sec.</td>
<td>15-30 min.</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.05-0.1 mg/kg</td>
<td>0.05 mg/kg Q 3-5 min.</td>
<td>&lt;60 sec.</td>
<td>15-30 min.</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>0.05 mg/kg</td>
<td>0.025-0.05 mg/kg Q 10-15 min.</td>
<td>2-3 min.</td>
<td>1-2 hrs.</td>
</tr>
</tbody>
</table>

Midazolam (Versed) is a water-soluble, short-acting benzodiazepine that has no analgesic properties. It has a predictable onset and short duration making it very popular for moderate sedation. It creates skeletal muscle relaxation, amnesia, and anxiolysis.

Oral Midazolam: produces light sedation, anxiolysis, and amnesia. The drug has a very bitter taste that is difficult to disguise. It should be diluted in as small amount as possible in order not to negate the NPO status of the patient. This medication is the closest of any of the current sedatives available in providing true minimal sedation. It provides a sedated yet arousable and cooperative patient at the indicated doses. One of the most desirable side effects is amnesia produced, although the extent of this effect varies with the patient’s age and the dose administered.

Rectal Midazolam: may be administered at doses of 0.3 to 0.75 mg/kg. Blood levels may be low 30 minutes after administration but sedation and anxiolysis effects remain.

Nasal Midazolam: doses for the intranasal route are 0.2 to 0.4 mg/kg with an intermediate onset time between the oral and intravenous routes of administration (10-15 minutes). This route has shown to be successful as a premedicant for anesthesia, but its use is limited by the burning to the nasal mucose experienced by the patient. Adverse effects, including respiratory depression and synergy with opioids, are similar to those mentioned above.

Summary of Enteral Midazolam Administration

<table>
<thead>
<tr>
<th>Route</th>
<th>Dose</th>
<th>Clinical Onset</th>
<th>+ Attributes</th>
<th>- Attributes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intranasal</td>
<td>0.2-0.4 mg/kg</td>
<td>10-15 min.</td>
<td>fast onset</td>
<td>irritating</td>
</tr>
<tr>
<td>Rectal</td>
<td>0.3-0.75 mg/kg</td>
<td>15-20 min.</td>
<td>age&lt; 3 yo</td>
<td>not older patients</td>
</tr>
<tr>
<td>Oral</td>
<td>0.3-0.75 mg/kg</td>
<td>15-30 min.</td>
<td>easy delivery</td>
<td>variable onset,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>bad taste</td>
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</tbody>
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Intramuscular Midazolam: Midazolam may be given as an intramuscular bolus of 0.08-0.1 mg/kg. Good sedation and cooperation scores were recorded at 15 minutes after this dose in one study. Persistent sedation is minimal 60 minutes after the dose.

Intravenous Midazolam: This is highly lipid soluble and redistributes quickly. It can be titrated to effect with fractionated doses of 0.05 to 0.1 mg/kg and may be repeated at intervals of every 3-4 minutes. It reaches its peak effect in 2-3 minutes. Care needs to be taken to observe for respiratory depression so it needs to be administered slowly. When combined with intravenous opioids for painful procedures,
Midazolam has potent sedative effects making the use of cardiorespiratory monitoring imperative. A maximum intravenous dose of 0.05 mg/kg has been recommended when it is combined with opiates.

Certain underlying conditions or medications may prolong the effects of Midazolam. Heparin decreases protein binding and increases the free fraction. Patients in renal failure may have three times the free fraction of the drug secondary to decreased protein binding.

Intravenous Midazolam is an excellent agent for sedation and anxiolysis in patients in minor procedures when an intravenous line is in place. It provides complementary sedation for patients receiving opioids for very painful procedures due to synergy but extreme caution is warranted when combing the drugs due to respiratory depression.

**Nitrous Oxide (N20)**

Nitrous Oxide is rarely used for moderate sedation. It is an odorless, colorless gas that produces both anxiolytic and analgesic effects. It must be delivered with oxygen in order to avoid a hypoxic gas mixture and is accomplished through the use of flow meters from separate sources or though the delivery of a fixed 50% mixture of N2/Oxygen (Entonox). It may be delivered alone at concentrations of 30-50% for moderately painful procedures or in combination with a mild sedative at lower concentrations for similar effects. Onset of analgesia and sedation occurs in minutes and is rapidly stopped when the gas is discontinued. Nitrous Oxide has minimal cardiovascular and respiratory effects when used alone. Studies in large groups of patients have failed to show any significant risk of cardiopulmonary depression when Nitrous Oxide is utilized at concentrations cited here. Nitrous Oxide concentrations of 30-50% are actually considered minimal sedation by the American Academy of Pediatrics. Cautions when using the medication include the possibility of a hypoxic mixture of gas to the patient if the equipment fails. Deep sedation is possible with high concentrations or when combined with opioids. There is a slight risk of nausea and vomiting associated with its use, but airway reflexes are reliably maintained. Occupational Safety and Health Administration (OSHA) guidelines for room air turnovers and scavenging must be met if any inhalational agent is used. This requirement may make the use of N2O impractical except in dedicated rooms with the appropriate equipment present.

Recommended use: Nitrous Oxide is useful for brief painful procedures like intravenous catheter placement, injections and urologic procedures and may be combined with a mild sedative. Expensive equipment and ventilation apparatus required for delivery may limit its widespread use.

**Primary Sedative-Hypnotic Drugs or “Sleepers”**

Sedative-hypnotic drugs are primarily used to make infants sleep. They are particularly effective for non-invasive procedures requiring a high level of immobility.
Chloral Hydrate is a sedative-hypnotic which is used to reduce mobility for non-painful procedures in Radiology and EEG and is most effective in patients less than 3 years of age. Overall it is less effective than Pentobarbital and fails to provide adequate immobility in about 30-40% of the cases. It can cause airway obstruction more so than respiratory depression. Onset is 30-60 minutes with a duration of 2-8 hours. There is no antidote for this medication.

Certain patient populations are at higher risk of having respiratory depression and oxygen desaturation following its administration. These include patients with chronic lung disease. Isolated mild oxygen desaturations occur in approximately 5% of patients receiving standard doses of chloral hydrate. Other issues are agitation and vomiting. Chloral Hydrate should be used very cautiously in neonates because of its long half-life which is: 40 hours for premature infants and 18 hours for full term infants. Deaths have been reported from infants who have slumped forward in their car seats on the way to or from the hospital and have obstructed their airway as a result.

Barbiturates
Barbiturates are potent sedative, hypnotic, anesthetic agents with strong anti-convulsant properties that do not have analgesic properties. Most sedation experience is with Pentobarbital.

Pentobarbital is one of the barbiturates used for neonatal sedation. It is a very good hypnotic and is very effective for non-painful procedures requiring a high level of immobility such as for CT or MRI scans. Pentobarbital has respiratory depressant effects but usually are generally well tolerated in otherwise healthy infants. It is a negative iontropic drug with vasodilator properties, but healthy individuals have few clinically significant effects when administered for sedation purposes. Hemodynamic effects are most pronounced when the drug is given rapidly and in patients with hemodynamic instability and hypovolemia. During induction with Pentobarbital, it is not uncommon to have excitatory phenomena.

Primary Sedative – Analgesic Drugs
Primary sedative analgesic drugs are drugs that are particularly useful for painful procedures. Analgesic agents may occasionally be combined with anxiolytics or hypnotics to enhance analgesic effects.

Opioid agonists: The effects of opioid agonists are dose dependent and include sedation and analgesia. Other clinical effects are respiratory depression and varying levels of bradycardia which are more common with the synthetic opioids like Fentanyl. Opioids do not provide amnesia.
**Fentanyl**

Intravenous Fentanyl is one of the most common opioid agonists used for neonatal procedural pain. Its peak effect is usually 4-5 minutes with respiratory depression being dose dependent. IV Fentanyl should be given approximately 4-5 minutes prior to the painful procedure. Recommended use and dosing: Intravenous Fentanyl doses of 0.5-2 mcg/kg over 1-2 minutes result in good analgesia. It is critical to administer IV Fentanyl slowly in order to avoid the potentially devastating side effect of *chest wall rigidity*. If this occurs, a reversal agent (Naloxone) can be administered to restore ability to ventilate. Repeat doses of 0.5 mcg/kg may be required during the procedure every 2-3 minutes. Fentanyl's duration of effect is typically 20-30 minutes; however, it may be as prolonged as 60 minutes. Fentanyl combined with other agents like Midazolam and Propofol may have synergistic effects.

**Morphine**

Intravenous Morphine has stood the test of time as a mainstay for controlling neonatal pain. Morphine’s onset of action is slow relative to Fentanyl, making it a less desirable drug for acute procedural pain. Similarly, Morphine’s clinical effect is prolonged, typically 2 to 4 hours. Consequently, Morphine is much better for post-operative pain or chronic pain management. Morphine may have some advantages for prolonged painful procedures. Below are comparisons in dosing, onset of action and duration between intravenous Fentanyl and Morphine.

<table>
<thead>
<tr>
<th>Opioid</th>
<th>Dose</th>
<th>Repeat Dose</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>0.05-0.2 mg/kg</td>
<td>0.05 mg/kg q 10 min.</td>
<td>5-10 min.</td>
<td>3-4 hrs.</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5-2µg/kg</td>
<td>0.5 mg/kg q 2-3 min.</td>
<td>2-3 min.</td>
<td>30-60 min.</td>
</tr>
</tbody>
</table>

**Ketamine**

Ketamine has dissociative sedative, analgesic, and amnestic properties with a long track record of safety as a sedative for painful neonatal procedures. Ketamine is one of the most versatile sedative-analgesic agents and results in a number of desired clinical effects that are dose-dependent. At the lowest of doses, anxiolysis and analgesia occur. Amnesia occurs at slightly higher doses and is often accompanied by perceptual changes. Higher doses result in a sedated state that is described as a “dissociative sedation.” Typically spontaneous respirations and airway reflexes are maintained although may not be totally normal. Ketamine generally causes an increase in heart rate, blood pressure and cardiac output. It is a potent cerebral vasodilator and its use is controversial in head trauma as it may increase intracranial pressure. It is also a potent hallucinogen. Emergence from Ketamine sedation is associated with visual and auditory hallucinations. This occurs more frequently in patients over 16 years of age and those patients with a history of psychosis. Providing pre-sedation education as well as the co-administration of a benzodiazepine will prevent or minimize these reactions. Ketamine does not produce significant respiratory depression when given slowly, however, apnea has been demonstrated following rapid IV administration. Oral secretions are typically only mildly increased, but may require anti-cholinergics to decrease secretions such as Atropine or Robinal. The single most severe adverse
effect with Ketamine sedation is laryngospasm. Ketamine is clinically effective by a number of different routes.

**Oral/Rectal Ketamine:** Oral and rectal doses of Ketamine are 4-10 mg/kg. Onset of sedation occurs in 15-30 minutes and effects may be prolonged by the oral or rectal route lasting 3-4 hours. Following oral administration (10 mg/kg), peak effects occurred in 30-40 minutes in patients undergoing painful cancer procedures. Typically, higher doses of oral Ketamine (8-10 mg/kg) are more effective as a pre-medication than lower doses (3-6 mg/kg).

**Intramuscular (IM) Ketamine:** Intramuscular Ketamine reaches peak blood levels and clinical effects in 5 minutes after 3-10 mg/kg. Recovery from dissociation occurs within 15-30 minutes with coherence and purposeful neuromuscular activity returning in 30-120 minutes. A smaller dose of 3 mg/kg has been utilized to facilitate intravenous catheter placement or acceptance of a mask for anesthesia induction, with no delay in discharge compared to control patients after 60 minutes. The 100 mg/ml formulation of Ketamine is preferred for IM administration in older patients to minimize volume related injection site discomfort. Experience with IM Ketamine is extensive. Sedation is accompanied by excellent analgesia. IM administration of Ketamine is an excellent means of sedating the “out of control” patient for IV placement or mildly painful procedures, however, deep sedation may occur.

**Intravenous (IV) Ketamine:** Ketamine is typically given in doses of 0.5-1 mg/kg although doses of 2 mg/kg can be used. Peak concentrations occur within 1-2 minutes and rapid absorption by the highly perfused cerebral tissues allows almost immediate induction of clinical effects. Ketamine then slowly redistributes into the peripheral tissues, thus decreasing central nervous system levels that correlate with return of coherence which generally takes 10-15 minutes if no additional doses are given. Deep levels of sedation may be achieved. Remarkably painful procedures are tolerated well following administration of Ketamine because of its profound analgesic effects as well as the dissociative sedation it affords.

Although patients will continue to breath and maintain airway tone, silent pulmonary aspiration of oral contents has been reported with deep levels of sedation. Patients may continue to move during sedation and eyes remain open. Emergence delirium is much less common in children than adults and may be prevented or treated by the administration of a small dose of a benzodiazepine. However, a recent study failed to demonstrate a reduction in emergence phenomena when administered with Midazolam. Vomiting is not uncommon, being reported in 12-25% in some series. Co-administration of Ketamine with Midazolam reduced the incidence of vomiting from 12% to 5% in a placebo controlled study of neonatal emergency department patients. Finally, in over 8,000 pediatric Ketamine sedations in patients admitted in the emergency department, risk factors that predicted Ketamine associated airway and adverse respiratory events were high intravenous doses, patients younger than 24 months of age and the co-administration of anti-cholinergics and benzodiazepines. Ketamine dosing by route is listed below.
### Route Dose Repeated Dose Clinical Onset Clinical Peak Duration

<table>
<thead>
<tr>
<th>Route</th>
<th>Dose</th>
<th>Repeated Dose</th>
<th>Clinical Onset</th>
<th>Clinical Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV</td>
<td>0.5-1 mg/kg</td>
<td>0.5 mg/kg q 2-3 min.</td>
<td>&lt;60 sec.</td>
<td>1-2 min.</td>
<td>10-15 min.</td>
</tr>
<tr>
<td>IM</td>
<td>2-4 mg/kg</td>
<td>2-4 mg/kg</td>
<td>1-2 min.</td>
<td>2-4 min.</td>
<td>30-60 min.</td>
</tr>
<tr>
<td>Oral/Rectal</td>
<td>6-8 mg/kg</td>
<td>6-8 mg/kg</td>
<td>~5-10 min.</td>
<td>10-20 min.</td>
<td>2-3 hrs.</td>
</tr>
</tbody>
</table>

### Reversal Agents

**Flumazenil (Romazicon):** A short-acting agent that reverses benzodiazepine-induced sedation. Resedation may occur due to its short duration of action; therefore, additional doses may be necessary. Flumazenil is not useful for barbiturate-or opioid-induced sedation.

*Flumazenil Precautions*
1) Should not be given to patients who are on benzodiazepines as part of therapy for a seizure disorder.
2) Give cautiously to patients who are on medications known to lower the seizure threshold, such as tricyclic antidepressants, theophylline, isoniazid or lithium.

**Flumazenil Dosing**
- Dose: 0.01 mg/kg IV (max dose 0.2 mg). If desired level of consciousness is not obtained after waiting an additional 45 sec., give repeat dose.
- Repeat dose: 0.005-0.01 mg/kg IV
- Induction time: 1-3 min. (peak effect 6-10 min.)
- Duration of effect: usually less than 60 min.
  Duration is related to the dose given and the benzodiazepine plasma concentrations; reversal effects of Flumazenil may be shorter than the effects of the benzodiazepine.
- Resedation may occur because the duration of effect of the benzodiazepine may exceed that of Flumazenil. In the event of re-sedation, repeat doses may be administered at 20-min. intervals as necessary.

**Naloxone (Narcan):** Naloxone is a short-acting agent that reverses opioid-induced sedation. It competes and displaces opioids at opioid-receptor sites. Resedation may occur due to its short duration of action; therefore, repeated doses are usually needed. Naloxone is not useful for barbiturate-, benzodiazepine- or phencyclidine-induced sedation.

*Naloxone Dosing*
- Dose: 0.01 mg/kg (IV) over 30 sec. as undiluted preparation
- Repeat dose: 0.01 mg/kg IV may be repeated every 2-3 min. as needed based on response
- Induction time: within 2 min.
- Duration of effect: 20-60 min.
  Duration is shorter than that of most opiates, therefore repeated doses are usually necessary

**Naloxone Clinical Effects**
- Resedation may occur, because the duration of effect of the opiate may exceed that of Naloxone. In the event of re-sedation, repeat doses may be administered.
- Naloxone may improve alertness, but should not be substituted for an adequate period of post-procedure monitoring. Monitoring (including BP) must continue until the patient returns to and maintains his/her baseline of consciousness.
- Naloxone may precipitate withdrawal symptoms (HTN, sweating, agitation, irritability, shrill cry, failure to feed).
Post-Sedation/Recovery/Discharge

Recovery Area and Equipment
The recovery area should be equipped with suction, oxygen, and equipment for positive pressure ventilation. Monitoring equipment including pulse oximetry, EKG, blood pressure, and ventilation monitoring should be available as well. A record of vital signs should be kept at regular intervals until the patient returns to baseline. Monitoring following moderate or deep sedation must include level of consciousness, oxygen saturation, adequacy of ventilation, continuous heart rate and pain assessment.

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 return to baseline must be achieved to be eligible for discharge.

<table>
<thead>
<tr>
<th>Post-Anesthesia Recovery Score</th>
<th>PAR Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Activity</strong></td>
<td></td>
</tr>
<tr>
<td>0 = unable to lift head or move extremities</td>
<td></td>
</tr>
<tr>
<td>1 = moves 2 extremities voluntarily or on command and can lift head</td>
<td></td>
</tr>
<tr>
<td>2 = able to move 4 extremities voluntarily or on command. Can lift head</td>
<td></td>
</tr>
<tr>
<td><strong>Respiration</strong></td>
<td></td>
</tr>
<tr>
<td>0 = apneic; condition necessitates ventilator assisted respirations</td>
<td></td>
</tr>
<tr>
<td>1 = labored or limited respirations; may have mechanical airway</td>
<td></td>
</tr>
<tr>
<td>2 = can take a deep breath and cough well; has normal respiratory rate and depth</td>
<td></td>
</tr>
<tr>
<td><strong>Circulation</strong></td>
<td></td>
</tr>
<tr>
<td>0 = has abnormally high or low BP ( &gt;50% pre-sedation level)</td>
<td></td>
</tr>
<tr>
<td>1 = BP 20-50% of pre-sedation level</td>
<td></td>
</tr>
<tr>
<td>2 = stable BP and pulse; BP 20% of pre-sedation level</td>
<td></td>
</tr>
<tr>
<td><strong>Neurological</strong></td>
<td></td>
</tr>
<tr>
<td>0 = not responding or responding to painful stimuli</td>
<td></td>
</tr>
<tr>
<td>1 = responds to verbal stimuli but drifts to sleep easily</td>
<td></td>
</tr>
<tr>
<td>2 = awake, alert, oriented to time, place and person</td>
<td></td>
</tr>
<tr>
<td><strong>O2 Sat</strong></td>
<td></td>
</tr>
<tr>
<td>0 = O2 saturation &lt;90% with O2 supplement</td>
<td></td>
</tr>
<tr>
<td>1 = needs O2 inhalation to maintain O2 saturation &gt;90% or &lt;95%</td>
<td></td>
</tr>
<tr>
<td>2 = able to maintain pre-procedure O2 saturation on RA or &gt;95% on O2</td>
<td></td>
</tr>
<tr>
<td><strong>Total Recovery Score</strong></td>
<td></td>
</tr>
</tbody>
</table>
# Modified Post-Anesthesia Discharge Score (MPADS)

<table>
<thead>
<tr>
<th><strong>Vital Signs</strong></th>
<th><strong>MPAD Score</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>0 = within 40% or &gt; of pre-sedation levels</td>
<td></td>
</tr>
<tr>
<td>1 = within 20%-40%</td>
<td></td>
</tr>
<tr>
<td>2 = within 20%</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Pain</strong></th>
<th><strong>MPAD Score</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>0 = severe (8-10)</td>
<td></td>
</tr>
<tr>
<td>1 = moderate (4-7)</td>
<td></td>
</tr>
<tr>
<td>2 = minimal/none (0-3)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Nausea &amp; Vomiting</strong></th>
<th><strong>MPAD Score</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>0 = severe</td>
<td></td>
</tr>
<tr>
<td>1 = moderate</td>
<td></td>
</tr>
<tr>
<td>2 = minimal/none</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Surgical Bleeding</strong></th>
<th><strong>MPAD Score</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>0 = severe</td>
<td></td>
</tr>
<tr>
<td>1 = moderate</td>
<td></td>
</tr>
<tr>
<td>2 = minimal/none</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Ambulation</strong></th>
<th><strong>MPAD Score</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>0 = none/dizziness</td>
<td></td>
</tr>
<tr>
<td>1 = with assistance</td>
<td></td>
</tr>
<tr>
<td>2 = steady gait/no dizziness (age appropriate)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Total Discharge Score</strong></th>
<th><strong>MPAD Score</strong></th>
</tr>
</thead>
</table>

## Monitoring Post-Procedure

Patients should be monitored closely until they have met specific criteria – this should be consistent regardless of the procedure that was performed or the drugs that were used for sedation. The criteria for return to routine care should include: 1) stable vital signs; 2) pain under control; 3) a return to the level of consciousness that is similar to the baseline for that patient; 4) adequate/patent airway; 5) adequate oxygenation/return to baseline requirements; and 6) nausea and/or vomiting should be controlled and the patient should be adequately hydrated. Similarly, infants who have had their sedation reversed with Flumazenil or Naloxone should be observed for an extended period of time (2-4 hours) due to the fact that re-sedation can occur as the reversal agent wears off and the sedative agent still has a therapeutic blood level.

## Documentation

Documentation will include observations of the patient’s condition – HR, RR, BP, O2 saturation, level of consciousness (LOC) – and support required as well as the medications administered. The frequency of these assessments will be every 15 minutes minimally until return to baseline. Inform the family that the patient may remain drowsy for the next 24 hours or so, may not feed well or interact as before.

## Emergency States During Sedation

### Airway Obstruction

Upper airway obstruction is the single most serious adverse event during moderate to deep sedation and is due to pharyngeal hypotonia. Most commonly the soft palate and epiglottis “fall back” to the posterior pharynx. Effective management of pharyngeal obstruction requires proper airway positioning,
removal of secretions when necessary, bag-valve-mask technique, and placement of appropriate alternative airways when appropriate.

**Laryngospasm**
Laryngospasm is an emergency observed during induction of, or emergence from, sedation/anesthesia. Laryngospasm may be partial or complete and is defined as glottis musculature spasm. Timely recognition and appropriate intervention with airway maneuvers and positive pressure ventilation with oxygen delivery to maintain baseline saturations are essential for effective treatment.

**Apnea-Hypoventilation**
The emergent response to apnea in the setting of procedural sedation consists of rapid recognition of the problem, assessment of the etiology, and appropriate treatment. Recognition of apnea is best accomplished by constant visualization of the patient, assessing for a patent airway and adequate chest wall movement. Electronic physiologic monitors serve as a valuable adjunct and in certain procedural situations (MRI bore, draped patient), the sedation practitioner will be completely dependent on monitors to detect apnea.

The etiology of apnea can be divided into two broad categories, central or obstructive, and the recognition and response depends on the cause. Central apnea represents the lack of respiratory effort and onset may be abrupt or preceded by a period of progressive hypopnea. In the setting of sedation, the cause of central apnea is most commonly pharmacologic, but is also a developmental aspect of the preterm infant. Treatment of central apnea consists of supporting oxygenation and ventilation with a bag-mask device, and removing or reversing the cause (halting sedative administration, administering reversal agents when indicated). The patient with obstructive apnea will continue to have respiratory effort with chest wall movement, although upper airway obstruction will preclude effective ventilation, eventually resulting in hypoxia. Treatment of obstructive apnea focuses on relieving the obstruction (see treatment of airway obstruction). Supplemental oxygen alone is not sufficient treatment for either central or obstructive apnea.

The absence of ventilation precedes hypoxia, especially in the patient receiving supplemental oxygen. Thus, monitoring airflow with a continuous side stream ETCO2 monitor enables faster response to apnea than does monitoring SpO2 alone, sometimes by several minutes. Upper airway obstruction and abrupt onset of central apnea can be detected immediately and recognition of and response to gradual hypoventilation with escalating CO2 values may prevent delayed onset of central apnea.

**Aspiration**
Pulmonary aspiration is one of the most common serious adverse events during sedation due to loss of protective airway reflexes. Aspiration is defined as the penetration of the airway, either proximal or distal, by gastric contents or oropharyngeal secretions. In a patient with intact airway clearance mechanisms and normal gastro-esophageal tone and motility, this does not occur. Concomitant use of a sedative or narcotic medication in these patients will increasingly diminish GI motility and sphincter tone. Additionally, therapeutic and diagnostic maneuvers or interventions may induce aspiration.
Aggressive suctioning may stimulate a gag reflex or cause coughing, both of which may promote passage of stomach contents into the oropharynx. Immediate recognition of airway secretions and/or aspiration is essential to adequate treatment. Proper airway maneuvers to facilitate removal of secretions, such as suctioning and bag-mask ventilation, may be required.

**Cardiovascular Instability**

The patient’s underlying cardiovascular physiology can contribute significantly to the side effects of sedative/analgesic medications. Patients in a pre-existing hypovolemic state or with an inflammatory response resulting in vasodilatation may have a profound decrease in organ perfusion as a result of the bradycardia, myocardial depression, or vasodilatation caused by deeper levels of sedation. Those with decreased myocardial function at baseline prior to sedation may not tolerate the effects of sedation and/or analgesic medications and in these situations, judicious use of medications that can be titrated may be the most prudent approach. Prior to procedural sedation in an infant, it is important to consider the patient’s underlying respiratory and cardiovascular physiologic state, the important side effects of the sedation/analgesia, and the procedure being performed. Advance preparation and anticipation of cardiovascular effects will contribute to the safe administration of sedation/analgesia outside of the operating room.
NEONATAL MODERATE SEDATION POST-TEST

1. Infants are more prone to airway obstruction during sedation due to:
   a. Large tongue
   b. Floppy epiglottis
   c. Large occiput places head in a naturally flexed position.
   d. All of the above

2. Airway obstruction during moderate sedation occurs in:
   a. Supraglottic area – soft palate and epiglottis
   b. Base of tongue
   c. Larynx
   d. Trachea

3. What are the steps to relieve obstruction? (Acronym)
   M -
   R -
   S -
   O -
   P -
   A -

4. Acronym for equipment needs:
   S -
   O -
   A -
   P -
   M -
   E -

5. Sedative/anxiolytic drugs include all except:
   a. Diazepam
   b. Midazolam
   c. Lorazepam
   d. Phenobarbital
6. The following is true of Midazolam (Versed) except:
   a. short acting benzodiazepine
   b. produces muscle relaxation
   c. dose is 0.05 – 0.1 mg/kg dose
   d. lasts 15-30 minutes
   e. maximum dose when combined with opiates is 0.1 mg/kg secondary to respiration depression

7. The following is true of Chloral Hydrate except:
   a. a sedative-hypnotic most effective in children <3 yo
   b. PO/PR 50 – 75 mg/kg; max 1 g/kg
   c. onset 60-90 min.
   d. no antidote
   e. causes airway obstruction more than respiratory depression

8. The following is true of Pentobarbital except:
   a. initial dose 2-4 mg/kg IV over 30-45 sec
   b. can administer faster than 50 mg/min.
   c. no reversal agent
   d. hemodynamic effects most pronounced when given rapidly
   e. may have excitatory phenomena in induction

9. The following is true of Fentanyl except:
   a. common opioid agonist
   b. peaks in 4-5 min.
   c. IV 0.5 – 2 mcg/kg over 1-2 min.
   d. can cause chest wall rigidity when given quickly
   e. Naloxone is not an effective reversal agent

10. The following is true of Morphine except:
    a. lasts 2-4 hrs.
    b. IV 0.05 – 2 mg/kg dose
    c. onset is faster than Fentanyl
    d. good for post-op/chronic pain secondary to prolonged effect

11. Match the reversal agent with the condition it is used to treat.
    1. _____ Flumazanil            a. _____ Opioid induced sedation
    2. _____ Naloxone             b. _____ Benzodiazepine induced sedation

12. T or F Re-sedation may occur after Flumazanil dosing.
13. The following is true of Naloxone except:
   a. re-sedation may occur
   b. IV 0.01 mg/kg
   c. can repeat every 2-3 min.
   d. lasts 20-60 min.
   e. does not precipitate withdrawal symptoms (HTN, sweating, agitation, irritability)

14. The following is true of Flumazanil except:
   a. IV 0.01 mg/kg
   b. duration < 60 min.
   c. peak effect ≈20-30 min.
   d. re-sedation may occur
   e. give cautiously to patients on meds known to lower the seizure threshold, e.g.,
      antidepressants, theophylline, isoniazid or lithium

15. Criteria for return to routine care include:
   a. stable vital signs
   b. pain free
   c. baseline level of consciousness
   d. adequate airway
   e. adequate hydration and nausea/vomiting under control