

# Procedural Sedation and Analgesia in the ED

## Focusing on Deep Sedation

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Emergency physicians are well trained in airway management and thus are well qualified to administer procedural sedation and analgesia (PSA) for painful or distressing procedures. Ultra-short-acting agents such as propofol and etomidate, which produce deep sedation, have become a mainstay of the emergency physician's armamentarium, but recent policy guidelines have placed restrictions on nonanesthesiologist-administered deep sedation. However, the most recent revision to CMS guidelines recognizes the emergency physician's unique qualifications to produce deep sedation. This article offers a useful discussion of preparation, monitoring, and medication considerations in deep sedation in the ED. A list of pitfalls to avoid in PSA is also included.

**A**dministration of procedural sedation and analgesia (PSA) is a vital skill for the emergency physician. PSA helps the emergency physician seamlessly perform procedures that would cause significant pain and anxiety and thus would otherwise be nearly impossible to perform. PSA is not without its potential adverse effects; thus, proper selection of patients and medications is essential. The emergency medicine literature demonstrates that emergency physicians can safely and effectively perform PSA, at all levels of sedation—minimal, moderate, and deep.

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### PSA IN THE HANDS OF EPs: A RECENT HISTORY

During the 1990s, the term *procedural sedation and analgesia* replaced the term *conscious sedation* to more accurately characterize what physicians are trying to accomplish. The type of sedation provided by PSA is a continuum with four distinct levels: minimal, moderate, deep, and general anesthesia (Table 1). *Minimal or light sedation* refers to the practice of providing medications for acute pain or anxiolysis (eg, morphine or lorazepam individually). With this level of sedation, patients are able to answer questions appropriately, but their coordination or cognitive function may be mildly impaired. In *moderate sedation*, the patient is able to respond purposefully to verbal commands or to light tactile stimuli. In *deep sedation*, only painful or repetitive painful stimuli will

yield a response. With sedation at this level, independent airway and ventilation function may become impaired. In *general anesthesia* (ie, in the operating room), there is no response to painful stimuli. Airway, ventilatory, and perhaps even cardiovascular function may be impaired and require intervention.

In the 1990s, the most common PSA medication combination used in the ED was fentanyl and midazolam. This combination of two relatively short-acting agents, an analgesic (fentanyl) with a sedative (midazolam), produces sedation in the moderate sedation range. As fentanyl/midazolam was so frequently used, some emergency physicians incorrectly referred to their overall PSA practice as “moderate sedation.” In some institutions, “moderate sedation” became synonymous with PSA use by all nonanesthesiologists, including emergency physicians. Many hospitals developed and required “moderate sedation” courses. Despite the fact that PSA and airway management are core skills in emergency medicine training, certification, and practice, some hospital credentialing committees require emergency physicians to take these “moderate sedation” courses along with other nonanesthesiologists requesting such privileges.

In the late 1990s, emergency physicians began adding ultra-short-acting sedative agents such as propofol, etomidate, and methohexital to their PSA repertoire, thus acquiring the tools to produce “deep sedation.” In the last 10 years, a large body of literature developed supporting the safety of these agents as administered by emergency physicians. These deep sedation agents

were adopted by many emergency physicians over midazolam/fentanyl because they have quicker onset, permit faster titration, provide better sedation, and have shorter recovery times. Since emergency physicians are usually credentialed in hospitals to perform moderate sedation but not deep sedation,<sup>1</sup> some emergency physicians called their use of these newer agents “moderate sedation,” claiming that they were titrating the medications to a moderate and not a deep sedation level.<sup>2</sup> Nevertheless, emergency physicians were producing deep sedation (as defined above) with these newer agents. Table 2 lists possible indications for the use of deep sedation in the ED.

The publicity surrounding the 2009 death of the musician Michael Jackson after propofol was administered as a sedative hypnotic sleep agent in Jackson’s home may have prompted the Centers for Medicare and Medicaid Services (CMS) to issue Revised Hospital Anesthesia Services Interpretive Guidelines (42. CFR.482.52) in February 2010.<sup>3</sup> These revised guidelines created a number of potential barriers to the use of deep sedation agents by emergency physicians. The new guidelines required that all hospital anesthesia services be organized under a single anesthesia service, creating the possibility that anesthesia departments could restrict or forbid emergency physician use of deep sedation agents. Many EDs started to negotiate deep sedation policies with their hospitals’ anesthesia departments. The guidelines also restricted permission to doctors of medicine or osteopathy to administer deep sedation agents (thus excluding registered nurses) and implied

**TABLE 1.** Levels of Procedural Sedation and Anesthesia

Sedation level	Patient responds to	Airway and spontaneous ventilation	Cardiovascular function
Minimal (pain management/ anxiolysis)	Verbal commands	Adequate	Intact
Moderate	Verbal commands or light tactile stimuli	Adequate	Intact
Deep	Painful stimuli; may require repetitive simulation	May be impaired; may require assistance	Usually maintained
General anesthesia	Patient is unarousable to any stimuli	Often impaired; may require assistance	May be impaired

that the professional administering the deep sedation could not also perform the procedure. This made it impossible for single-coverage emergency physicians to perform deep sedation. Additionally, in academic centers, one faculty member could not simultaneously supervise the deep sedation and the procedure; thus, two attendings would be required at the bedside. In January 2011, CMS issued another revision to the interpretive guidelines, allowing specialty-specific guidelines to be used in the clinical areas of the hospital where the specialists (eg, emergency physicians) practice.<sup>4</sup> ACEP's 2005 PSA clinical policy document<sup>5</sup> was used as a specific example of specialty guidelines that could be used to guide PSA use in the ED. Another 2005 ACEP clinical policy<sup>6</sup> quoted in the CMS 2011 revision supports the "delivery of medications used for procedural sedation and analgesia by credentialed emergency nurses working under the direct supervision of an emergency physician. These agents include but are not limited to etomidate, propofol, ketamine, fentanyl, and midazolam." CMS acknowledged that "emergency medicine-trained physicians have very specific skill sets to manage airways and ventilation" and "are uniquely qualified to provide all levels of analgesia/sedation and anesthesia (moderate to deep to general)."<sup>4</sup> Hopefully, the 2011 CMS revision will encourage hospitals to credential emergency physicians to perform deep sedation, although there is still concern among experts in the field of emergency medicine PSA that the deep sedation political battle is not over.<sup>7</sup> For emergency physicians whose PSA practice has

**TABLE 2. Potential Indications for Deep Sedation in the ED**

- Fracture reduction
- Joint reduction
- Incision and drainage
- Chest tube placement
- Electrocardioversion
- Upper endoscopy (with a gastroenterologist)
- Foreign body removal
- Burn or wound debridement

been previously restricted, now is the time to master the art of deep sedation.

### PREPARATION FOR PSA

Preparation is essential for any procedure, but even more so for procedures that may have airway and/or cardiovascular effects. All equipment necessary to perform the PSA and the procedure and to manage any potential complications should be at the bedside. Recommended equipment and documents are listed in Tables 3 and 4. Informed consent should be obtained both for the procedure and the PSA. A presedation assessment should be performed and documented. Immediately before the procedure, a time-out should be carried out. A multidisciplinary time-out (most commonly performed by a doctor of medicine or osteopathy and a registered nurse) helps ensure that the correct procedure is being performed on the correct patient and the correct side of the body (if relevant).

### Fasting

Before undergoing PSA, patients should be asked what they last ate or drank and when. If oral intake was re-

**TABLE 3. Recommended Equipment for PSA**

- Monitoring equipment (continuous telemetry, pulse oximeter, BP; consider continuous end tidal CO<sub>2</sub> monitoring)
- Peripheral IV, normal saline
- Medications for PSA
- Naloxone (if opiates are given)
- Equipment for procedure (eg, scalpel)
- Team (ideally, one practitioner for sedation, one for procedure)
- Airway equipment (oxygen source, nasal cannula/face mask, BVM, suction)
- Rescue airway equipment (ETT<sup>a</sup>, laryngoscope, LMA, nasal trumpet)<sup>b</sup>

<sup>a</sup>Set up with stylet and 10-cc syringe. <sup>b</sup>Suggested.

PSA = procedural sedation and analgesia; BP = blood pressure; CO<sub>2</sub> = carbon dioxide; IV = intravenous line; BVM = bag valve mask; ETT = endotracheal tube; LMA = laryngeal mask airway.

cent, delaying the procedure should be considered. The American Society of Anesthesiologists (ASA) has fasting guidelines that are meant to decrease pulmonary aspiration risk in elective PSA and general anesthesia procedures (Table 5).<sup>8</sup> These are useful general guidelines for the emergency physician; however, these recommendations are based on consensus, not scientific evidence, and they are not directly applicable to emergency PSA. In their recently updated 2011 guidelines, the ASA remarks with reference to each of their fasting recommendations that the “literature is insufficient to evaluate the effect of the timing of ingestion” and the “incidence of emesis/reflux or pulmonary aspiration.”<sup>8</sup> Procedures performed by the emergency physician are often urgent, and delaying them may not be an option. Despite the frequent need to perform PSA in non-fasted patients, pulmonary aspiration is extremely rare. In a review of emergency PSA studies utilizing many different agents, only one case of pulmonary aspiration was described in 4,657 adult and 17,672 pediatric cases; ASA fasting guidelines were not adhered to in many of these patients.<sup>9</sup> The one reported aspiration case was a case report and not from a clinical trial.<sup>9</sup> Although aspiration is a very rare event with moderate and deep sedation, fasting times should not be completely disregarded, and in each case the risks and benefits of nonadherence to fasting guidelines must be weighed.

### Patient Selection

As part of a pre-sedation assessment, it must be determined whether the patient is a proper candidate for deep sedation in the ED. Deep sedation (and PSA) may not be ideal for patients with significant chronic morbidities (eg, sleep apnea, COPD), low baseline oxygen saturations, or anatomic features that would make bag valve mask (BVM) ventilation or maintaining an airway dif-

ficult. The patient should be assessed to determine how difficult BVM ventilation would be if needed (eg, airway obstruction, obesity, large beard, edentulous state). If there is concern that PSA cannot be safely performed in the ED, other options should be considered, including local anesthesia (eg, intra-articular block for a shoulder reduction), light sedation (with just an anxiolytic or analgesic), or sedation in the operating room. At times, the emergency physician may have to perform PSA in less than ideal candidates, either because of lack of specialty backup or because the procedure is emergent (eg, chest tube placement in an elderly, agitated patient). In these cases, it is especially important to have all needed resuscitation equipment at the bedside and to document the necessitating circumstances.

### DURING PSA

#### Patient Monitoring

During deep sedation, the level of consciousness will be severely depressed, and the patient may at times have difficulty keeping an open airway. The patient should be carefully monitored, with particular attention given to the respiratory rate, adequacy of respirations, oxygen saturation, and potentially the end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>; see “End-tidal CO<sub>2</sub> Monitoring” below). If respirations become inadequate, the following should be considered: First, additional doses of the sedative should not be given. Next, the airway should be opened with a head tilt or jaw thrust maneuver; this alone may be enough to encourage more spontaneous respirations. Since the procedure being performed is likely painful, it should be started, as this may stimulate the patient to breathe. Other painful stimuli that do not cause harm to the patient may be attempted (eg, pressing on the fingernails). If all of these measures are ineffective and the patient’s oxygen saturation drops into the low 90s, BVM ventilation should be initiated.

#### End-tidal CO<sub>2</sub> Monitoring

In the ED during PSA, continuous ETCO<sub>2</sub> monitoring (capnography) can be obtained through the use of a nasal cannula detector connected to a monitor. Continuous capnography provides real-time breath-to-breath monitoring of the patient’s ventilatory status during PSA and allows the clinician to trend ventilatory

**TABLE 4.** Documentation in PSA

- Institutional preprocedural sedation form
- Consent form for procedure and PSA
- Time-out form

PSA = procedural sedation and analgesia.

status over the course of the procedure. Continuous capnography provides an early warning that respiratory depression or airway compromise is developing before oxygen desaturation takes place.

Several ETCO<sub>2</sub> patterns should alert the emergency physician to problems during use of capnography for PSA.<sup>10</sup> A rise in ETCO<sub>2</sub> of more than 10 mm Hg from baseline signals hypoventilation with an unobstructed airway. A level greater than 50 mm Hg raises the same concern. A rising ETCO<sub>2</sub> level indicates that the airway is unobstructed (hence the rise in CO<sub>2</sub>), and stimulating the patient to breathe should be considered. On the other hand, if there is hypoventilation in the setting of an airway obstruction (or a developing airway obstruction), CO<sub>2</sub> will not be exhaled and there will be a drop in ETCO<sub>2</sub>. In this setting, loss of an ETCO<sub>2</sub> waveform or a drop of 10 mm Hg is potentially significant. When this occurs, the first step is to open the airway; then, the patient should be stimulated to breathe. With any significant change in the waveform, subsequent sedatives should be withheld. Recent research suggests that using a 10% change in ETCO<sub>2</sub> from baseline rather than a 10-mm Hg change to signal a need for physician intervention in PSA increases the sensitivity of capnography.<sup>10,11</sup>

### Oxygen Supplementation

A common question is whether supplemental oxygen should be used during PSA, and if so, how much. The advantage of not using supplemental oxygen is that it allows the pulse oximeter to be used as an indicator of apnea. Even small amounts of supplemental oxygen (eg, 2 to 4 L via nasal cannula) will delay the fall in oxygen saturation, potentially delaying the detection of apnea. When using ultra-short-acting agents (eg, propofol, etomidate) for quick procedures, some emergency physicians utilize an oxygen face mask and preoxygenate the patient for at least 3 to 5 minutes before the procedure. This provides a large oxygen reservoir. The oxygen face mask combined with preoxygenation allows for a large window before significant oxygen desaturation manifests, which is enough time to perform most ED procedures and for the deep sedative to start wearing off. The face mask technique is best used with capnography, in which ETCO<sub>2</sub> monitoring can provide

the earliest indication of hypoventilation and alert the emergency physician to act well before oxygen desaturation occurs.

## DEEP SEDATION MEDICATIONS

### Propofol

Propofol is highly effective for PSA in the ED, and its safety in the hands of emergency physicians has been extensively studied.<sup>12-16</sup> Propofol exerts its effect through GABA receptors. It is an ideal agent for short ED procedures because it has a very rapid onset (about 45 seconds) and a quick offset (3 to 5 minutes); it also provides powerful sedation and amnesia. Because of this agent's short duration of action, the patient will generally wake up a few minutes after the last dose. Propofol has no analgesic effects. Prior to a painful procedure, some emergency physicians administer an analgesic such as an opiate (eg, morphine or fentanyl) or ketamine.<sup>17</sup> Opiate pretreatment prior to propofol administration should be decided on a case-by-case basis. If this approach is utilized, extra caution should be taken, as the combination of a narcotic and propofol is more likely to result in respiratory depression. Less propofol will be needed if narcotics are preadministered. In

**TABLE 5. ASA Fasting Recommendations<sup>a</sup>**

Type of oral intake	Recommended minimum pre-PSA fasting time
Clear liquids (eg, water, teas, coffee without milk, juices without pulp)	2 hours
Breast milk	4 hours
Light meal, infant formula, nonhuman milk	6 hours
Heavier meal (including fried/fatty foods)	8 hours

ASA = American Society of Anesthesiologists; PSA = procedural sedation and analgesia.

<sup>a</sup>These ASA recommendations are intended to help prevent pulmonary aspiration in healthy patients who are having elective procedures.

Adapted from: American Society of Anesthesiologists.<sup>8</sup>

one study, when fentanyl at a dose of 1.5 µg/kg IV was given prior to propofol, 77.4% of patients experienced oxygen desaturation below 92% and 12.9% had desaturation below 70%.<sup>18</sup> When propofol and fentanyl are used together, smaller doses of fentanyl should be used and there must be very careful titration of the propofol.

**ED Use**—Propofol is an excellent first-line PSA agent for hemodynamically stable patients. It is useful for joint reductions, fracture reductions, abscess incision and drainages, electrocardioversions, and foreign body extractions. In particular, propofol is excellent for prosthetic hip reductions, which require complete muscle relaxation.

**Dosing**—Propofol is generally packaged and distributed in bottles containing a propofol concentration of 10 mg/mL (but it is always prudent to double-check the concentration on the label of the product being used). Propofol is easily titrated. A starting dose for ED PSA is 0.5 to 1 mg/kg IV.<sup>12</sup> Most ED studies on propofol PSA use the 1-mg/kg IV dose,<sup>12,16</sup> which works out to an initial dose of 40 to 80 mg (4 to 8 mL). Consider starting with half of this dose in the geriatric population. Subsequent doses are administered every 2 to 3 minutes in smaller aliquots (0.5 mg/kg IV) and titrated to the desired effect.<sup>12</sup> Dosing in children is the same as in adults, with a starting dose of 1 mg/kg IV followed by 0.5 mg/kg every 3 minutes as required.<sup>14,17</sup>

**Side Effects**—Propofol can quickly take a patient from deep sedation to general anesthesia with a lack of spontaneous respirations if too high a dose is administered. Propofol frequently causes hypotension, but this can usually be treated successfully with a fluid bolus. For patients with normal cardiac function, providing IV hydration before administering propofol may help blunt this hypotensive effect. Propofol's hypotensive effect makes this agent a potentially poor choice for PSA in patients who are already hypotensive, in shock, volume depleted, or bleeding or in those with poor cardiac reserve. Additionally, propofol is contraindicated in patients with an egg or soybean oil allergy. Propofol can cause burning when it is administered.

### Etomidate

Etomidate is well known to the emergency physician, as it is a very commonly used induction agent for rapid

sequence intubation (RSI). Etomidate is a nonbarbiturate, sedative/hypnotic agent that works through GABA receptors. Like propofol, it can produce deep sedation for PSA, with a rapid onset and relatively short duration of effect. Following etomidate sedation, patients are responsive to verbal commands in about 5 minutes, with a recovery time ranging from 6 to 16 minutes.<sup>19</sup> An advantage of etomidate is hemodynamic stability. It is unlikely to cause drops in systolic blood pressure, especially in the smaller doses used in PSA. Like propofol, etomidate has no analgesic properties. Concomitant or preprocedure use of analgesics (eg, fentanyl or morphine) can be considered for painful procedures but will increase the likelihood of respiratory depression or desaturation.

**ED Use**—Etomidate may be a better choice than propofol in the potentially unstable patient. Etomidate may be particularly useful in trauma patients who require a chest tube or a fracture or joint reduction. Etomidate is useful in electrocardioversion, in which hemodynamics are a consideration. Otherwise, etomidate can be used in the same scenarios as propofol.

**Dosing**—In PSA, a starting dose of 0.1 mg/kg IV is most frequently recommended.<sup>12,19</sup> If the sedation achieved is inadequate, subsequent doses of 0.05 mg/kg IV can be used every 3 to 5 minutes.<sup>12,19</sup>

**Side Effects**—Etomidate causes myoclonus in about 20% of cases.<sup>12,20</sup> Myoclonus is not seen during RSI because of the paralytic agent used for this procedure. In the majority of cases, the myoclonus is transient and minor and is not associated with respiratory or physiologic issues for the patient. In very rare cases, the myoclonus can cause full body movements and make the procedure difficult or impossible to perform. In these severe myoclonus episodes, opening the airway or performing BVM ventilation may become difficult. Unlike propofol, which may have antiemetic properties, etomidate may induce vomiting at a rate of about 4%<sup>21</sup>; the rate may be higher (up to 6.7%) in patients older than 65 years.<sup>22</sup> Fasting guidelines might therefore need to be followed more carefully with etomidate. Adrenal suppression is a frequently discussed side effect of etomidate; however, whatever minimal adrenal suppression etomidate may cause will be irrelevant with brief, one-time procedures on generally healthy

ambulatory patients undergoing ED PSA. In two studies, etomidate PSA use has been associated with systolic blood pressures above 200 mm Hg.<sup>21,22</sup> As these events are infrequently reported, it is not clear if they represent a medication side effect that is underreported or instead are a result of pain or agitation during the procedure. As with any agent that produces deep sedation, etomidate will cause respiratory suppression. In one study, 9.5% of patients experienced an oxygen desaturation below 92%, and 3.8% received BVM ventilation.<sup>12</sup>

### Comparing Etomidate With Propofol

In a prospective, randomized (but not blinded) study that compared propofol with etomidate sedation in the ED, the two agents were found to have similar rates of sedation, hypoxia, apnea, and other respiratory depressive events.<sup>12</sup> However, propofol was more effective, with a procedure success rate of 97.2% versus 89.5% for etomidate. Additionally, propofol was associated with a much lower incidence of myoclonus (1.8%) compared to etomidate (20%).

### Barbiturates: Methohexital, Thiopental

The short-acting barbiturates methohexital and thiopental have been used for PSA in the ED setting. Both thiopental and methohexital have relatively quick onset (about 1 minute) and offset (about 10 minutes). Both can cause significant hypotension via direct myocardial depression and venodilation. Additional disadvantages of thiopental, which has not been studied for PSA in the ED, include histamine release (ie, it should be avoided in patients with asthma) and tissue necrosis if extravasation occurs. Methohexital causes myoclonus and increases seizure potential in patients with temporal lobe epilepsy. As with etomidate and propofol, these agents have respiratory-depressive effects. The starting dose for methohexital is 1 mg/kg IV.<sup>23</sup> These agents have no obvious advantages over propofol.

## DISSOCIATIVE ANESTHETIC AGENTS

### Ketamine

Ketamine, a phencyclidine (PCP) derivative, is a dissociative agent. Unlike other agents discussed above, ketamine is not a deep sedation agent, but its effective-

ness and safety make it an important agent to discuss. Ketamine creates a trance-like, cataleptic state that causes “sensory isolation,” in which the cortical centers of the brain do not appreciate painful, visual, or auditory stimulation. This dissociative state provides the patient with sedation, amnesia, and analgesia. Ketamine’s ability to provide both analgesia and sedation is a unique advantage. Ketamine’s effects are different from those of other PSA agents, making classification into a moderate or deep category inappropriate. As part of their trance-like state, patients may keep their eyes open and have random purposeless movements, yet be completely unresponsive to pain.

Of all the PSA agents, ketamine has the best safety profile. Ketamine rarely causes respiratory depression (<1%).<sup>24</sup> With ketamine, the patient retains spontaneous respirations, while airway, gag and cough reflexes are preserved. Ketamine has been used safely around the world; frequently it is used intramuscularly without any significant monitoring. Despite its safety profile, some US hospitals require that IV access be obtained prior to IM administration.

**ED Use**—Ketamine is an ideal agent for procedures in children and young adults; it is useful for fracture reduction, chest tube placement, laceration repair, incision and drainage, and foreign body removal. It is also an ideal agent for emergent trauma procedures, as it will sustain blood pressure and provide analgesia. Ketamine may not be a good choice for difficult joint reductions, as it does not produce complete muscle relaxation.

**Dosing**—For PSA, the initial dose of ketamine is 1 mg/kg IV over 30 to 60 seconds.<sup>24</sup> In pediatric patients, an initial IV dose of 1.5 to 2 mg/kg or an IM dose of 4 to 5 mg/kg can be used.<sup>24</sup> Intravenous administration has the advantages of quicker onset (1 vs 5 minutes) and shorter duration of effect (5 to 10 vs 20 to 30 minutes).<sup>24</sup> The initial loading dose should produce the desired catatonic state, but if it does not, additional 0.5- to 1-mg/kg IV doses may be administered.<sup>24</sup> At lower doses, the effects of ketamine are primarily analgesic with some sedation, but no catatonic state is produced. When enough ketamine has been administered to produce a catatonic state (ie, 1 to 1.5 mg/kg IV), subsequent dosing (0.5 to 1 mg/kg IV) helps maintain the

catatonic state rather than deepen the sedation.

**Side Effects**—In rare cases (0.3%), ketamine can cause laryngospasm, which is typically responsive to BVM ventilation.<sup>25</sup> Laryngospasm is thought to be more common in children who have a concomitant upper respiratory tract infection, in procedures with major stimulation of the oropharynx, and when abnormalities of the larynx and trachea are present. Ketamine causes hypersalivation and increased secretions; anticholinergic coadministration has traditionally been advocated to mitigate these effects. However, in a large meta-analysis of ketamine use, coadministration of an anticholinergic actually increased the rate of airway and respiratory complications.<sup>25</sup> Therefore, routine anticholinergic use (eg, glycopyrrolate, atropine) with

**TABLE 6.** Mistakes to Avoid in PSA

- Performing PSA on a patient that is a poor candidate due to comorbidities
- Not checking the medication concentration
- Not labeling the medication syringes
- Not having all necessary airway equipment at the bedside
- Pushing PSA agents too quickly, leading to hypotension and/or apnea
- Switching sedative or analgesic agents during the procedure, creating an unpredictable response
- Succumbing to pressure from a colleague or consult to create a deeper level of sedation than is actually needed and is safe
- Continuing to give PSA medications despite some evidence of respiratory compromise
- Not knowing when to stop (ie, failing to recognize that the procedure and/or PSA will not be successful)
- Not fully monitoring the patient post procedure, leading to unrecognized apnea
- Not monitoring a patient for sufficient time after use of a reversal agent (eg, naloxone or flumazenil)

PSA = procedural sedation and analgesia.

ketamine is no longer recommended.<sup>24,25</sup> Ketamine is a sympathomimetic, increasing heart rate, blood pressure, and myocardial oxygen consumption. Its use is frequently avoided in older adults out of concern that the sympathomimetic effects will unmask coronary ischemia. It is unclear whether this is a valid issue for older adults who do not have symptomatic coronary artery disease. Vomiting occurs in 5% to 15% of cases, most commonly during recovery.<sup>26</sup> Coadministration of an antiemetic, specifically ondansetron, can be considered in children and young adolescents who are at high risk for vomiting.<sup>24</sup>

Emergence reactions are a feared side effect of ketamine, with symptoms of agitation, confusion, physical aggressiveness, and hallucinations. These symptoms are rare in children, with mild emergence reactions (dreams, hallucinations) occurring in 7.6% of cases and more severe reactions occurring in 1.4%.<sup>27</sup> Typically, only severe reactions need treatment. Emergence reactions occur more frequently in adolescents and adults, but not as often as once thought. To decrease the incidence of recovery agitation/emergence reactions, a benzodiazepine, typically midazolam 0.03 mg/kg IV, can be administered in adults.<sup>28</sup> One randomized controlled study found this dose of midazolam administered with the initial dose of ketamine decreased the incidence of recovery agitation (NNT =6) and did not significantly increase recovery time.<sup>28</sup> In children, emergence reactions are rare enough that routine benzodiazepine use is no longer recommended; instead, benzodiazepines should be administered only if clinically significant symptoms develop.<sup>24</sup> Ketamine use should be avoided in patients with psychosis or schizophrenia.

## MEDICATIONS OF FUTURE INTEREST

### Ketofol

Not FDA approved but increasingly described in the medical literature, “ketofol” is a mixture of propofol and ketamine. This PSA combination works synergistically, with ketamine adding analgesia to propofol without the high rates of respiratory depression found when a narcotic (eg, fentanyl) is combined with propofol.<sup>18</sup> In fact, concomitant ketamine use may decrease the respiratory depression effects of propofol. Ketamine’s sympathomimetic effects may blunt the hypotensive effects of pro-



propofol. On the other hand, concomitant propofol is likely to decrease the incidence of emergence phenomena,<sup>29</sup> decrease the incidence of vomiting, and produce more profound muscle relaxation than ketamine used alone. Although this combination has definite promise, a more extensive evidence base is needed before this combination is ready for widespread ED use.

### Dexmedetomidine

A newer agent, dexmedetomidine is a centrally acting  $\alpha_2$  adrenergic agonist (the same drug class as clonidine). The agent is of potential interest to emergency physicians because it produces both sedation and analgesia with minimal effects on ventilation. With this agent, patients maintain their usual ventilatory response to hypercarbia. Patients are described as sedated but arousable, in an unusual state of “cooperative sedation.” As an  $\alpha_2$  adrenergic agonist, dexmedetomidine decreases sympathetic nervous system activity, reduces norepinephrine levels, and may decrease heart rate and blood pressure. The biggest obstacle to ED use at this time is the way the agent is administered. It is recommended to intravenously load the medication over 10 minutes, followed by a continuous infusion. This duration may be suitable for ongoing sedation in an intubated patient, but it is entirely too long for ED PSA. Currently, there are very limited data on use of dexmedetomidine for ED PSA, and it is quite expensive.

### POSTPROCEDURE CARE

Although there are pitfalls to avoid at various points during the PSA procedure (Table 6), the most dangerous time may be the end of the procedure. The painful stimuli (eg, dislocated hip) that prevented the patient from entering deeper states of sedation are now gone. At this time, the staff may pay less attention to the patient’s respiratory status, and apnea may go unrecognized. At minimum, a nurse should remain at the bedside until the patient has been at his or her baseline level of arousal for a number of minutes. The patient should not be sent out of the ED (eg, to radiology for postreduction radiographs) until he or she has returned to preprocedure baseline and remained at this level for a significant period (at least 5 to 10 minutes). This is another reason why propofol and etomidate may be

better choices than the fentanyl/midazolam combination, as recovery time will be much shorter and less observation time will be needed.

Before hospital discharge, patients should have regained full preprocedure mental and functional capacity, and their vital signs should have normalized. They should be instructed not to drive or participate in any activities that would require a high level of alertness or careful coordination for the next 24 hours. They should also be advised to return to the ED if they have questions or problems. Ideally, a patient should be discharged to the care of a responsible adult.

### CONCLUSION

Deep and dissociative sedation agents allow emergency physicians to perform difficult procedures safely, effectively, and with relatively short recovery times. Emergency physicians are well trained in airway management; they should feel confident that they have the clinical skills and the necessary training to safely provide deep sedation to their ED patients. **EM**

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