Sedation Of The Mechanically Ventilated Patient In The Emergency Department

Abstract

Sedation of critically ill patients is challenging, and current literature and guidelines have contributed to a significant shift in strategies for these patients. Critically ill patients have unique metabolic demands, and patient-specific factors such as organ dysfunction and age must be considered when creating patient-centered sedation plans. Analgesosedation is a prevailing sedation strategy that focuses first on the relief of pain and is associated with improved patient outcomes. Judicious and measured use of sedative medications helps to avoid oversedation, hypotension, increased intensive care unit length of stay, and mortality. This review examines the current literature and describes when supplemental medications may be required and how to titrate these drugs to achieve the desired effect. Ongoing monitoring of sedation is also of prime importance, and several validated sedation scales are discussed. The sedatives, analgesics, and anesthetics currently utilized in the care of critically ill patients are also reviewed.
Case Presentations

It's change of shift in your busy ED, which has been operating beyond capacity for several days. You have just received sign-out on a 35-year-old woman with asthma who is in severe respiratory distress. Noninvasive ventilation has failed, and you performed rapid sequence intubation. Postintubation, she remains diaphoretic and dysynchronous with the ventilator. Despite increased titration of a lorazepam infusion, the airway pressures continue to climb. She is tachycardic and visibly agitated. The nurse advises you that there is absolutely no bed availability in the ICU. You strategize about a long-term plan for postintubation sedation, as her care will be provided in the ED for at least the next 24 hours. The nurse is asking what you would like to do next. What are your options?

Later in the same shift, you are caring for an elderly man from a nursing home who presents with septic shock. The postintubation film shows the endotracheal tube is in its proper position. Infiltrates are present throughout all lung fields, and he remains hypotensive despite aggressive therapy with antibiotics and IV fluids. While you prepare him for central venous access, you notice his heart rate remains elevated. He periodically opens his eyes and struggles against the restraints despite the initial bolus of preintubation sedatives. There is no available ICU bed, and you consider your sedation plan for this patient who is hypotensive, tachycardic, and not responding to fluid boluses. The bedside nurse asks if you’d like to administer another 2 mg of midazolam. What is your next step?

Introduction

Providing optimal sedation for an intubated patient presents challenges to the practicing emergency physician. Overcrowded emergency departments (EDs) are often charged with caring for admitted patients when appropriate intensive care unit (ICU) beds are not available, so the emergency physician should be prepared to continue appropriate sedation of the critically ill and mechanically ventilated patient for the first 24 hours of a patient’s hospitalization.

Although some form of sedation is typically employed for mechanically ventilated patients, there is considerable practice variation with respect to initial drug selection and administration. Moreover, data suggest that the overall quality of analgesia and sedation is low, and that the choice and intensity of early sedation may affect short-term and long-term outcomes (such as time on a ventilator). The Society of Critical Care Medicine recently released new recommendations for the management of pain, agitation, and delirium in adult ICU patients. The methodology used was rigorous and transparent, establishing the foundations for evidence-based sedation practice.

Sedation of critically ill patients begins with aggressive identification and treatment of pain. (See Figure 1.) Well-designed sedation strategies focus first on the alleviation of pain and the avoidance of undesired effects (such as withdrawal, delirium, and hypotension). Pain is commonly experienced by the critically ill as a consequence of mechanical ventilation, routine procedures, care during hospitalization, and/or underlying disease states. Pain contributes to significant patient discomfort and triggers the activation of a stress response, factors that often result in the need for higher doses of sedative medications. Inadequate sedation may result in increased agitation and ventilator asynchrony, and can contribute to myocardial ischemia.

This review provides the practicing emergency physician with the necessary tools to craft an individualized and evidence-based treatment plan, focusing exclusively on continued sedation treatment plan, focusing exclusively on continued sedation treatment plan, focusing exclusively on continued sedation treatment plan, focusing exclusively on continued sedation treatment plan, focusing exclusively on continued sedation treatment plan. Following intubation of critically ill adult patients who are managed in the ED.

Figure 1. Analgesia And Sedation Algorithm

- Numeric Rating Scale
- Behavioral Pain Scale
- Nonverbal Pain Scale
- Morphine (caution with renal failure)
- Hydromorphone (caution with renal failure)
- Fentanyl
- Remifentanil (rapid off)
- Provide light sedation, when possible
- Use objective scale (eg, RASS)
- Consider patient-specific factors
- Benzodiazepine - lorazepam (propylene glycol toxicity possible), midazolam (caution in renal failure)
- Propofol (rapid off, monitor for hypotension), fospropofol (limited data)
- Dexmedetomidine (sedative and analgesic, rapid off)

Abbreviation: RASS, Richmond Agitation-Sedation Scale.
A literature search was performed using Ovid MEDLINE® and PubMed from 2000 to the present, focusing on evidence-based literature regarding critically ill adult patients who are mechanically ventilated and require ongoing sedation for the first 24 to 48 hours of their acute illness. Terms used in the search included: analgesia, sedation, analgesosedation, sedation of the critically ill, and analgesia of the critically ill. Search results were reviewed and relevant articles and citations were searched. Emphasis was placed on relevant randomized controlled trials, systematic reviews, and/or meta-analyses and practice guidelines released by emergency medicine and critical care societies who used robust and transparent methods in their literature review and recommendations.

The literature reveals a significant amount of controversy with respect to ideal dosing regimens and strategies for sedation in mechanically ventilated patients. Recent studies have focused on the need to improve quality and consistency of sedation, and that imperative has contributed to the focus on analgesosedation. The current literature and guidelines have contributed to a significant shift in sedation strategies, as outlined in this issue. Table 1 highlights the 3 most recent and pertinent practice guidelines and clinical policies related to sedation of the critically ill patient.\(^3,10,11\)

### Table 1. Published Guidelines Related To Sedation Of The Critically Ill Patient

<table>
<thead>
<tr>
<th>Organization</th>
<th>Topic</th>
<th>Type of Guideline (Release Date)</th>
<th>Relevant Recommendations</th>
</tr>
</thead>
</table>
| Society of Critical Care Medicine | Clinical practice guidelines for management of pain, agitation, and delirium in adult patients in the intensive care unit.\(^3\) | Evidence-based (2013) | • Pain assessment and analgesia-first sedation in mechanically vented critically ill patients is most important (+2B)
• IV opioids should be first-line drug class for pain (+1C)
• Preemptive analgesia and nonpharmacologic interventions should be performed before invasive and potentially painful procedures (+2C)
• Initiate light (rather than deep) levels of sedation (+1B) with close monitoring and assessment
• Use RASS and SAS as assessment tools to measure quality and depth of sedation (B)
• Give nonbenzodiazepine sedatives (propofol or dexmedetomidine) rather than benzodiazepines to improve clinical outcomes (+2B) |
| American College of Emergency Physicians | Clinical policy: procedural sedation and analgesia in the emergency department.\(^10\) | Evidence-based (2005) | • Obtain thorough history and assessment prior to sedation
• Initiate frequent monitoring in well-equipped setting |
| American Society of Anesthesiologists | Practice guidelines for sedation and analgesia by nonanesthesiologists.\(^11\) | Evidence-based (2002) | • Maintain close monitoring with use of end-tidal CO\(_2\) and cardiac monitors
• Have familiarity with reversal agents |

Abbreviations: CO\(_2\), carbon dioxide; IV, intravenous; RASS, Richmond Agitation-Sedation Scale; SAS, Sedation-Agitation Scale.
**Opioid Analgesics**

The primary mechanism of action for opioids is activation of mu receptors that inhibit central pain responses, causing analgesic and sedative effects. Multiple studies, expert consensus, and best practice recommendations embrace the benefit of pain surveillance and management in critically patients. A paradigm shift towards an “analgesic first” approach to sedation positions opioid medications as the first-line management for agitated adults. The specific opioid to be utilized for sedation is best determined by an assessment of an individual patient condition.

**Fentanyl**

Fentanyl is the prototypical opioid medication since it is readily available and can be administered via bolus dose or infusion. A reasonable initial approach to analgesia involves bolus dosing to achieve relief of pain. Fentanyl can be given at boluses of 0.35 mcg/kg to 0.5 mcg/kg intravenously (IV) and then initiated at an infusion rate of 25 mcg/hour to 100 mcg/hour. Dosing is titrated up every 30 minutes to 1 hour to achieve the desired effect. Benefits of fentanyl include its favorable hemodynamic profile and rapid (within minutes) onset of action. Fentanyl does not cause histamine release, and the discomfort associated with opioid-related pruritus is therefore minimized. Fentanyl infusions have neutral hemodynamic effects and are less likely to cause hypotension. All opioids are implicated in respiratory depression, but this is obviously less of a concern for patients receiving mechanical ventilation. Chest wall rigidity (the “rigid chest syndrome”) has been cited as a negative effect of high-dose fentanyl infusion. Some case studies point to the incidence of chest wall rigidity in patients receiving physiologic doses of fentanyl. Chest wall rigidity will be discussed in more detail in the “Special Circumstances” section. (See page 9.)

<table>
<thead>
<tr>
<th>Table 2. Basic Opioid Analgesic Pharmacology³</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Medication</strong></td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>Fentanyl (Sublimaze®, Actiq®, Durogesic®, et al)</td>
</tr>
<tr>
<td>Remifentanil (Ultiva®)</td>
</tr>
<tr>
<td>Morphine</td>
</tr>
<tr>
<td>Hydromorphone (Dilauid®, Palladone®, Exalgo®)</td>
</tr>
</tbody>
</table>

* These doses represent loading doses for the initiation of sedation. Dosages require modification for procedures, such as rapid sequence intubation. Abbreviations: IV, intravenous; PRN, as needed; q, every.

<table>
<thead>
<tr>
<th>Table 3. Basic Pharmacology Of Selected Benzodiazepines And Anesthetics³</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Medication</strong></td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td><strong>Benzodiazepines</strong></td>
</tr>
<tr>
<td>Midazolam (Versed®, Dormicum®, Hypnovel®)</td>
</tr>
<tr>
<td>Lorazepam (Ativan®)</td>
</tr>
<tr>
<td><strong>Anesthetics</strong></td>
</tr>
<tr>
<td>Dexmedetomidine (Precedex®, Dexdor®, Dexdomitor®)</td>
</tr>
<tr>
<td>Ketamine (Ketalar®)</td>
</tr>
<tr>
<td>Propofol (Diprivan®)</td>
</tr>
</tbody>
</table>

* These doses represent loading doses for the initiation of sedation. Dosages require modification for procedures, such as rapid sequence intubation. Abbreviation: IV, intravenous.
Remifentanil
The shift towards analgesia-based sedation has led to the scrutiny of novel opioid agents such as remifentanil, which is classified separately due to its unique method of metabolism. It is metabolized through a pathway of blood and tissue esterases independent of organ dysfunction. Other unique characteristics include its shorter onset time and very short half-life. Given its broad spectrum of effects, remifentanil has been the subject of several randomized controlled trials. Patients in remifentanil-only trials received fewer medications and experienced an increased duration of “optimal sedation.” The medication is usually well tolerated and its use in the ICU is associated with a sedative-sparing effect. Remifentanil-based analgosedation strategies may result in a decreased ICU length of stay and a shortened duration of mechanical ventilation. Partially due to its brief duration of action, the use of remifentanil may be supplemented with additional parenteral analgesics. In Muellejans et al’s double-blind and randomized controlled study of sedated ICU patients, de-escalation from remifentanil-based sedation was associated with the administration of additional fentanyl and morphine boluses. Therefore, a proactive strategy of analgesia is required, given the medication’s rapid offset.

Remifentanil can be given at boluses of 1 mcg/kg to 1.5 mcg/kg IV, initiated at an infusion of 0.25 mcg/kg/min, and then titrated up to achieve the desired effect. As with every other sedative agent, it is important to consider the potential for adverse effects. Hypotension and bradycardia are listed as side effects of remifentanil infusion, but a meta-analysis did not show an increased incidence of hemodynamic instability. Withdrawal is commonly associated with the de-escalation of long-term opioid infusions, and patients receiving opioid sedation are at risk for delirium and tachycardia during weaning. Patients receiving analgosedation-based drug regimens also experience higher instances of unpleasant recall and hyperalgesia. However, these side effects should be balanced against the more significant risks of respiratory depression, hemodynamic compromise, and drug toxicity associated with traditional multidrug strategies. Analgosedation with remifentanil merits consideration, given the potential for a decreased length of ICU stay and a shortened time on mechanical ventilation. However, controlled trials comparing remifentanil and fentanyl with outcome data across a broad spectrum of different types of critically ill patients still need to be completed before its adoption as the opioid of choice for analgosedation.

Benzodiazepines
Benzodiazepines work through potentiation of neurominhibitory gamma-aminobutyric acid (GABA)-A receptors in the central nervous system. All medications in this class are relatively rapid acting and can produce several desired effects in the management of critically ill patients. At physiologic doses, benzodiazepines act rapidly to cause sedative, hypnotic, anxiolytic, anticonvulsant, muscle relaxant, and amnestic effects. They do not possess any analgesic activity. Benzodiazepines are useful in the management of acute agitation, given their comparatively rapid onset. Benzodiazepines can be administered via intermittent bolus or continuous infusion. The most commonly used drugs in this class for prolonged sedation of critically ill patients are midazolam and lorazepam.

Midazolam is very lipid soluble and it rapidly penetrates the blood-brain barrier, resulting in a rapid onset of action. Lorazepam is less lipophilic, with an intermediate onset of action, but both midazolam and lorazepam will accumulate in adipose tissue and can have prolonged effects when administered by continuous infusion. Benzodiazepines are primarily metabolized by the liver; therefore, prolonged sedation may occur in patients who are elderly, have liver disease, or who are also on medications that inhibit cytochrome enzymes. Midazolam is also broken down into metabolites that can accumulate in patients with renal insufficiency, unlike lorazepam, which is the preferred benzodiazepine in patients with renal failure. Midazolam has been associated with dose-dependent hypotension, and, when compared directly to lorazepam, it has a lower rate of adequate sedation and is not as cost-effective. Adverse effects important for the emergency physician to consider include excessive duration of sedation, risk of withdrawal with rapid discontinuation of infusions, and paradoxical agitation (seen rarely in elderly patients).

Finally, the critical care literature notes propylene glycol toxicity as a consideration in patients undergoing prolonged sedation with benzodiazepines. Propylene glycol is often used as a solvent in parenteral formulations of lorazepam, and toxicity can cause hyperosmolar metabolic acidosis, hypotension, and arrhythmias. Originally thought to occur only at unusually high doses, Yahwak et al’s prospective observational study of 35 patients implicated total daily doses of 1 mg/kg in toxicity. Because critically ill patients may be acidic at baseline, current guidelines recommend using the serum osmolar gap as a screening tool for propylene glycol toxicity. In Yahwak et al’s study, an osmol gap in excess of 12 suggested toxicity.

Benzodiazepines have historically been used as first-line agents, but the current literature is clear that, when compared to newer agents like propofol and dexmedetomidine, benzodiazepines are implicated in and contribute to delayed extubation and worsened outcomes. Although it seems counterintuitive, benzodiazepine use has been linked to the development of delirium in ICU patients.
bензодиазепины’ high affinity for GABA-A receptors within the central nervous system may alter levels of other naturally occurring, more deliriogenic neurotransmitters (such as dopamine and serotonin). The most recent guidelines support sedation strategies using nonbenzodiazepines to improve outcomes in mechanically ventilated patients.3

Anesthetic Agents

Anesthetic agents represent another group of medications useful for continued sedation in the emergency and critical care setting. Most emergency physicians are familiar with propofol, ketamine, and etomidate. However, etomidate is not routinely used for ongoing sedation because of the drug’s supressive effect upon the adrenal cortex, which limits etomidate’s utility as a continuous infusion. Therefore, this review will focus on the maintenance of sedation with propofol and ketamine.

Propofol And Fospropofol

Like several of the benzodiazepines, propofol is a lipophlic agent that produces an extremely rapid onset of anesthesia. Propofol binds to several receptors in the central nervous system and it shares GABA agonist properties with the benzodiazepines. Propofol’s effects are dose-dependent and range from mild sedation to general anesthesia. Propofol readily crosses the blood-brain barrier and induces sedation within minutes.14,15 Continuous infusion is required to maintain sedation because propofol is rapidly cleared following a bolus administration. Propofol has been used in the treatment of status epilepticus refractory to benzodiazepines, and it may have applications in the treatment of delirium tremens.3,14 Because of the drug’s rapid redistribution and high clearance rates, propofol has utility in patients who require frequent neurological reassessment.

Physicians must keep several caveats in mind when using propofol as the primary sedative in a critically ill patient. First, propofol has no analgesic properties.3 Agitation or persistent tachycardia may indicate untreated pain in patients sedated with propofol. Second, patients allergic to soy or egg ingredients should not receive propofol because those ingredients are used in the drug’s lipid formulation. Third, propofol’s lipid composition may result in elevated blood triglycerides. Though derangements in lipid metabolism are not of immediate concern to the emergency physician, propofol can induce hypertriglyceridemia and pancreatitis.3,14 Fourth, respiratory depression and hypotension have been documented following bolus administration of this agent, and it should be used with caution in hemodynamically tenuous patients. Lastly, prolonged infusion of propofol is associated with a unique clinical entity called propofol-related infusion syndrome.16 The pathological mechanism of propofol-related infusion syndrome is not fully elucidated, but its diagnosis is suggested in the presence of bradycardia and cardiac failure, worsening acidosis, renal failure, increasing vasopressor requirements, and rhabdomyolysis.16 Persistent elevations in serum lactate and creatine kinase levels in combination with hypertriglyceridemia should alert physicians to the presence of propofol-related infusion syndrome. Treatment is supportive and includes the immediate discontinuation of propofol.16,31

Fospropofol, a prodrug of propofol, is an emerging water-soluble alternative that has been discussed in recent critical care literature. The onset of action is slightly slower than propofol, but it still acts in a matter of minutes and has a smaller volume of distribution and accumulates less in adipose tissue when given as an infusion.3 Although a pilot study suggests safe and effective short-term use in the ICU setting, further studies must establish its safety and effectiveness for prolonged infusion and use in the ED.32,33

Ketamine

Ketamine has emerged as a useful agent for pain control, procedural sedation, and anesthesia. Ketamine’s versatility stems from its ability to induce a dissociative state. Ketamine administration permits patients to maintain airway protective reflexes while blunting pain input into the central nervous system. Ketamine is structurally related to the hallucinogen phencyclidine (PCP) and works through antagonism at the N-methyl-D-aspartate receptor.34 Effects are achieved within minutes of a bolus dose, and its duration of action is approximately 15 minutes. Positive hemodynamic actions of ketamine include stimulation of the sympathetic nervous system; therefore, ketamine promotes cardiovascular stability and is associated with bronchodilation.14,34 Early studies linked ketamine administration with increased intracranial pressure, but recent reviews argue against this association.24 In fact, ketamine’s ability to maintain (and perhaps increase) mean arterial pressure makes it useful in the induction of sedation in hypotensive patients.14,24 There is good clinical evidence to support the use of subdissociative doses of ketamine in the management of acute pain.35,36 However, outcome studies focused on ketamine infusions for sustained sedation in the critical care setting are lacking. Ketamine’s potential to induce agitation and sympathetic stimulation limits its utility in prolonged sedation. Emergence delirium and hallucinations are known to occur in association with ketamine. Premedication with a benzodiazepine may prevent an emergence reaction, and benzodiazepines are often used to mitigate delirium following the use of ketamine.37 Excessive salivation following ketamine anesthesia complicates ongoing airway management strategies.
Centrally Acting Alpha Agonists
Dexmedetomidine is a centrally acting alpha-2 receptor agonist that inhibits norepinephrine release and possesses qualities of an ideal sedative agent. In addition to its sedative effects, dexmedetomidine has anagelsec and anxiolytic properties. Dexmedetomidine is usually administered as an infusion and titrated up to the desired effect. It has been reliably shown to decrease the need for additional opioids. According to Devlin et al, dexmedetomidine produced fewer cases of delirium, perhaps due to its lack of action at GABA receptors and absent anticholinergic activity.16 Given dexmedetomidine’s alternative mechanism of action, the drug produces sedation patterns that are distinct from other agents. Patients receiving dexmedetomine are more easily aroused and interactive, and noted to not have respiratory depression associated with many of the other sedatives.38 Following intravenous infusion, sedation usually occurs within 15 minutes and reaches peak effect within 60 minutes. The medication is approved by the United States Food and Drug Administration (FDA) for short-term (<24 h) use, but multiple high-quality studies have demonstrated dexmedetomidine’s safety profile at higher doses and at longer durations.14,20,38 Bolus dosing of this drug may reduce the time to onset of sedation, but it is not without risk: dexmedetomidine boluses are linked to cardiovascular instability, namely bradycardia and hypotension.3,16 Bradycardia may be more common with higher doses of dexmedetomidine and in patients receiving calcium-channel blockers or beta blockers.16 Avoiding bolus loading doses and starting slow, continuous infusions mitigate these effects.38,39
Dexmedetomidine is a versatile agent that deserves consideration in an evidence-based sedation plan. Its use results in analgesia and anxiolysis and the avoidance of respiratory depression. Finally, dexmedetomidine administration may reduce the need for additional sedative medications.14

Tools And Techniques: Practical Considerations For Sedation Of The Critically Ill Patient
The provision of adequate pain control requires direct and effective communication in order to objectively assess pain. The Numeric Rating Scale (NRS) (0 = no pain to 10 = worst pain) is a validated and familiar tool that should be used to assess pain in critically ill patients who can speak (or at least point to numbers or physically rate pain). In mechanically ventilated or otherwise noncommunicative patients, it is important to search for physical signs of pain using tools like the Nonverbal Pain Scale (NPS) (see Table 4) or the Behavior Pain Scale (BPS) (see Table 5). Reliance on vital signs alone to predict pain is

Table 5. The Behavioral Pain Scale (BPS)

<table>
<thead>
<tr>
<th>Item</th>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial expression</td>
<td>Relaxed</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Partially tightened (eg, brow lowering)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Fully tightened (eg, eyelid closing)</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Grimacing</td>
<td>4</td>
</tr>
<tr>
<td>Upper limb movements</td>
<td>No movement</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Partially bent</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Fully bent with finger flexion</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Permanently retracted</td>
<td>4</td>
</tr>
<tr>
<td>Compliance with mechanical ventilation</td>
<td>Tolerating movement</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Coughing but tolerating ventilation for the most of time</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Fighting ventilator</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Unable to control ventilation</td>
<td>4</td>
</tr>
</tbody>
</table>


Table 4. The Adult Nonverbal Pain Scale

<table>
<thead>
<tr>
<th>Categories</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Face</td>
<td>No particular expression or smile</td>
<td>Occasional grimace, tearing, frowning, wrinkled forehead</td>
<td>Frequent grimace, tearing, frowning, wrinkled forehead</td>
</tr>
<tr>
<td>Activity (movement)</td>
<td>Lying quietly, normal position</td>
<td>Seeking attention through movement or slow, cautious movement</td>
<td>Restless, excessive activity and/or withdrawal reflexes</td>
</tr>
<tr>
<td>Guarding</td>
<td>Lying quietly, no positioning of hands over areas of body</td>
<td>Splinting areas of the body; tense</td>
<td>Rigid, stiff</td>
</tr>
<tr>
<td>Physiologic I (vital signs)</td>
<td>Stable vital signs (no change in past 4 h)</td>
<td>Change over past 4 h in any of the following: SBP &gt; 20 mm Hg, HR &gt; 20 beats/min, RR &gt; 10 breaths/min</td>
<td>Change over past 4 h in any of the following: SBP &gt; 30 mm Hg, HR &gt; 25 beats/min, RR &gt; 20 breaths/min</td>
</tr>
<tr>
<td>Physiologic II</td>
<td>Warm, dry skin</td>
<td>Dilated pupils, perspiring, flushing</td>
<td>Diaphoretic pallor</td>
</tr>
</tbody>
</table>

Abbreviations: HR, heart rate; RR, respiratory rate; SBP, systolic blood pressure.
discouraged, as it correlates poorly with presence or absence of pain.\textsuperscript{21,40,41} Studies using rigorous psychometric analysis in the ICU setting have proven the BPS to be a valid and reliable method of assessing behaviors as indicators of pain. Implementation of the BPS has been shown to improve pain management and improve clinical outcome, including better use of analgesic medications and shorter durations of mechanical ventilation and ICU stays.\textsuperscript{4,21,42}

Patients are considered to be in significant pain if they self-report their pain intensity $\geq 4$ (0-10 NRS) or have either a BPS score $\geq 6$ (BPS range = 3-12) if they cannot self-report. These scales have their limitations, but patient-centered feedback on the response to administration of analgesics will help guide the successful sedation strategy.

After appropriate analgesia, a patient’s true sedation requirements can be more accurately assessed and sedative medications can be avoided or doses can be significantly decreased.\textsuperscript{1,43} Successful sedation strategies implemented early by the ED stay may improve important short- and long-term patient outcomes (such as earlier ventilator liberation and improved mortality).\textsuperscript{3,44,45} Patient self-reporting is the gold standard criterion to identify pain. Unfortunately, no such standard exists for sedation.\textsuperscript{9} Validated sedation scales and protocols like the Richmond Agitation-Sedation Scale (RASS) (see Table 6) and Sedation-Agitation Scale (SAS) (see Table 7) are important to help assess patient arousal and response to environment and intervention. Implementation of bedside sedation scales can allow physicians to assess and more effectively communicate the patient’s depth of sedation. Multiple subjective sedation scales exist, with varying degrees of quality. They have been studied mostly in the ICU setting and are based on objective assessment of psychometric properties of the scales.\textsuperscript{9} Based on current

Table 6. The Richmond Agitation-Sedation Scale (RASS)

<table>
<thead>
<tr>
<th>Score</th>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>+4</td>
<td>Combative</td>
<td>Overtly combative or violent; immediate danger to staff</td>
</tr>
<tr>
<td>+3</td>
<td>Very agitated</td>
<td>Pulls on or removes tube(s) or catheter(s) or has aggressive behavior toward staff</td>
</tr>
<tr>
<td>+2</td>
<td>Agitated</td>
<td>Frequent nonpurposeful movement or patient-ventilator dysynchrony</td>
</tr>
<tr>
<td>+1</td>
<td>Restless</td>
<td>Anxious or apprehensive but movements not aggressive or vigorous</td>
</tr>
<tr>
<td>0</td>
<td>Alert and calm</td>
<td>Calm, attentive</td>
</tr>
<tr>
<td>-1</td>
<td>Drowsy</td>
<td>Not fully alert, but has sustained awakening ($\geq 10$ sec), with eye contact in response to voice</td>
</tr>
<tr>
<td>-2</td>
<td>Light sedation</td>
<td>Briefly awake ($&lt; 10$ sec) with eye contact in response to voice</td>
</tr>
<tr>
<td>-3</td>
<td>Moderate sedation</td>
<td>Any movement (but no eye contact) in response to voice</td>
</tr>
<tr>
<td>-4</td>
<td>Deep sedation</td>
<td>No response to voice, but any movement in response to physical stimulation</td>
</tr>
<tr>
<td>-5</td>
<td>Unarousable</td>
<td>No response to voice or physical stimulation</td>
</tr>
</tbody>
</table>

Procedure

1. Observe patient. Is patient alert and calm (score 0)? Does patient have behavior that is consistent with restlessness or agitation (score +1 to +4 using the criteria listed under Description)?

2. If patient is not alert, in a loud speaking voice, state the patient’s name and direct the patient to open his eyes and look at you. Repeat once, if necessary. You may prompt the patient to continue looking at you. If the patient has eye opening and eye contact that is sustained for $\geq 10$ sec = score -1. If patient has eye opening and eye contact, but it is not sustained for 10 sec = score -2. If patient has any movement in response to voice (excluding eye contact) = score -3.

3. If patient does not respond to voice, physically stimulate patient by shaking his shoulder; rub sternum if there is no response to shaking the shoulder. If patient has any movement to physical stimulation = score -4. If patient has no response to voice or physical stimulation = score -5.


Table 7. The Sedation-Agitation Scale (SAS)

<table>
<thead>
<tr>
<th>Score</th>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Dangerous agitation</td>
<td>Pulling at endotracheal tube, trying to remove catheters, climbing over bed rail, striking at staff, thrashing side-to-side</td>
</tr>
<tr>
<td>6</td>
<td>Very agitated</td>
<td>Does not calm despite frequent verbal reminding of limits; requires physical restraints, biting endotracheal tube</td>
</tr>
<tr>
<td>5</td>
<td>Agitated</td>
<td>Anxious or mildly agitated; attempting to sit up; calms down to verbal instructions</td>
</tr>
<tr>
<td>4</td>
<td>Calm and cooperative</td>
<td>Calm, awakens easily, follows commands</td>
</tr>
<tr>
<td>3</td>
<td>Sedated</td>
<td>Difficult to arouse; awakens to verbal stimuli or gentle shaking but drifts off again; follows simple commands</td>
</tr>
<tr>
<td>2</td>
<td>Very sedated</td>
<td>Aroused to physical stimuli but does not communicate or follow commands; may move spontaneously</td>
</tr>
<tr>
<td>1</td>
<td>Unarousable</td>
<td>Minimal or no response to noxious stimuli; does not communicate or follow commands</td>
</tr>
</tbody>
</table>

literature, the most valid and reliable sedation scales for adult ICU patients are the RASS and the SAS.

Although not specifically validated in the ED setting, we suggest using the RASS or SAS based on staff familiarity and comfort. RASS feasibility is well documented by ICU nursing surveys that confirmed ease of use and appropriateness in the majority of respondents.\textsuperscript{9,46,47} Frequent pain assessments using these scales (every 2-3 hours or more frequently, as needed) can help guide patient-specific sedation strategies and titration of medications. The literature suggests that patients who have their medications titrated to a light level of sedation (RASS not less than -2) can avoid delayed ventilator liberation, longer hospitalization, and other negative outcomes associated with deeper levels of sedation.\textsuperscript{1,3,48-50} Clear communication of optimal sedation goals, with all members involved, is key to providing care for the critically ill patient. Nursing and physician education and the development of departmental policies and sedation strategies for critically ill patients using objective scales and established institutional protocols will improve the delivery of appropriate care.

**Special Circumstances**

It is impossible for a single ED physician managing multiple patients to possess immediate recall with respect to each agent’s particular adverse effects. Table 8 summarizes common complications linked to specific sedative and analgesic agents. Vigilance for oversedation, respiratory depression, and other complications is important and mandates frequent patient reassessment.

Other patient-specific factors such as genetic variation, body weight, and temperature will individualize drug delivery, metabolism, and response to therapy. Factors such as obesity and hypothermia will further affect the volume of distribution and plasma levels various medications.

### Elderly Patients

As with most diagnostic and treatment regimens, a sedation strategy should be tailored to meet the particular demands of the patient’s pathophysiology. Several factors are important to keep in mind when choosing among agents. As the population continues to age, it is prudent to review how medications affect patients at the extremes of age. The critically ill elderly patient presents several management challenges. Many sedatives are extensively metabolized via hepatic pathways and undergo elimination via the kidneys. Reduced glomerular filtration is exacerbated by low flow states (such as sepsis and shock), so anesthetic and sedative agents may have a prolonged duration of effect. Adverse effects are more commonly encountered, and the sedation plan may require a dosing adjustment prior to administration.

Propofol is associated with a deeper level of sedation in elderly patients and may be linked with increased episodes of apnea during induction of anesthesia.\textsuperscript{16} Respiratory depression and prolonged sedation are likely in elderly patients receiving benzodiazepine-based infusions. Accordingly, respiratory depression can occur following the administration of fentanyl or other opioid and benzodiazepine infusions.\textsuperscript{2,16}

### Multisystem Organ Dysfunction

Multisystem organ dysfunction, preexisting parenchymal disease, and shock states have the potential to decrease the clearance of sedative, anesthetic, and analgesic agents. Drugs administered via intravenous infusion are preferred, given their ability to be titrated to the desired effect. Several medications have specific renal dosing recommendations for patients experiencing acute liver or kidney failure. The use of propofol is discouraged in patients with a vasopressor requirement, given its potential for hypotension.\textsuperscript{2,16} Fentanyl is often preferred in hemodynamically unstable patients given its rapid onset,

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**Table 8. Commonly Used Sedatives And Patient-Specific Factors Linked To Decreased Neurologic Recovery**

<table>
<thead>
<tr>
<th>Patient Factors</th>
<th>Fentanyl</th>
<th>Dexmedetomidine</th>
<th>Lorazepam</th>
<th>Midazolam</th>
<th>Propofol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate renal impairment CrCl 10-30 mL/min</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td>End-stage renal disease CrCl &lt; 10 L/min</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td>End-stage liver disease</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td>Continuous IV infusion</td>
<td>+++</td>
<td>0</td>
<td>+++</td>
<td>+++</td>
<td>0</td>
</tr>
</tbody>
</table>

Abbreviations: 0, no effect; +, minor effect; ++++, major effect; CrCl, creatinine clearance; IV, intravenous.

short half-life, and favorable cardiovascular profile.\(^2\)

Dexmedetomidine undergoes extensive hepatic metabolism and should be used with caution in patients with known or acute liver failure, as patients\(^3,15\) with hepatic dysfunction experience prolonged emergence from dexmedetomidine infusions.\(^3,16\) Remifentanil shows promise as a single-use agent due to its alternative extrhepatic pathway of metabolism. With respect to the benzodiazepine class of sedatives, it is not recommended to use midazolam infusions for patients with end-stage liver or kidney disease. Midazolam is linked to increased ventilator dependence and withdrawal phenomena when compared to other agents.\(^2,3,16,51\)

**Chest Wall Rigidity**

Opioid-induced respiratory depression can be mitigated through careful monitoring and judicious administration. However, the phenomenon of opioid-associated chest wall rigidity persists across the critical care literature. Originally thought to occur only following high doses of fentanyl, muscle rigidity has been documented in both pediatric and adult patients receiving physiologic doses of opioids.\(^5,17-19\) Rigidity that interferes with mechanical ventilation is a rare side effect and more commonly linked to higher dosing regimens.\(^3,16\) The true incidence of chest wall rigidity is difficult to ascertain because isolated case reports comprise the majority of the literature. The syndrome is more commonly encountered in neonatal and infant populations. Chest wall rigidity is clinically diagnosed in the presence of decreasing ventilator compliance following fentanyl administration. Treatment involves abrupt cessation of the fentanyl infusion and antagonization with naloxone. It seems prudent to consider chest wall rigidity in patients who experience worsening ventilator compliance in the absence of mechanical obstruction, endotracheal tube dislodgement, or equipment failure. Paralytic agents may also have a role in the arrest of opioid-induced chest wall rigidity.

**Pregnant Patients**

Care of the critically ill pregnant patient presents unique challenges due to the anatomic and physiological changes that occur in pregnant patients, especially during middle to late pregnancy. Efforts must be taken to aggressively avoid hypoxia and hypotension that will compromise maternal and fetal circulation, and medications must be chosen carefully. All analgesics and sedatives discussed in this issue are class C drugs in pregnancy (except the benzodiazepines [Class D], and propofol [Class B]). Careful sedative and analgesic selection is important when managing the critically ill pregnant patient and potentially harmful benzodiazepines should be avoided. For a detailed review of important steps in the management of critically ill pregnant patients, see the June 2012 issue of *EM Critical Care*, “Supportive Management Of Critical Illness In The Pregnant Patient.”

**Cutting Edge: Neurologic Monitoring**

Commercially available monitors incorporate and display electroencephalographic findings in an attempt to objectively measure brain function and assess the depth of sedation. These devices analyze electroencephalogram (EEG) waveforms and display findings on a visual scale. A variety of monitors and scoring systems exist. Examples include: auditory evoked potentials (AEPs), bispectral index (BIS), Narcornted Index (NI), patient state index (PSI), or state entropy (SE). Recent guidelines do not support the routine use of these devices for noncomatose, nonparalyzed patients. These devices are inadequate substitutes for subjective sedation scoring systems, and experience with these devices is limited in the ED setting (-1B).\(^2\) However, objective measurements and EEG-related indices (BIS, SE, PSI) remain the primary method to monitor depth of sedation in patients experiencing an extended duration of paralysis (+2B). These measurements should, ideally, be made in admitted patients under the direction of appropriately trained clinicians. They are currently of limited utility in the ED.

Clinical scenarios in which ED patients would be subject to prolonged periods of neuromuscular blockade are rare; however, newer technologies may have some utility in screening patients for nonconvulsive status epilepticus. Subjective sedation assessments may be unobtainable in the patient receiving neuromuscular blocking agents. Patients with known or suspected seizure disorders present a significant challenge to the emergency physician. EEG monitoring is recommended to detect nonconvulsive seizure activity in critically ill patients known or suspected to have seizures (+1A).\(^2\) EEG analysis permits accurate administration and titration of electrosuppresserive agents.

Cardiopulmonary monitoring is of utmost importance in the assessment of the stability of the critically ill patient. Bedside assessment and documentation of vital signs, cardiac rhythms, ventilator and ventilator waveforms (when available), continuous waveform capnography, and oxygen saturation can all play a role in detecting deterioration of the critically ill patient and are recommended. For a detailed review of the topic, see the August 2013 issue of *EM Critical Care*, “Ventilator Management In The Intubated Emergency Department Patient.”

Continuous end-tidal carbon dioxide (ETCO\(_2\)) monitoring allows real-time confirmation and monitoring of adequate ventilation through capnographic waveform analysis. Continuous ETCO\(_2\) monitoring is a valuable tool for detecting endotracheal tube
dislodgement, dysfunction, and acute respiratory/cardiac events (ie, apnea from excessive analgosedation, cardiac arrest, etc). For a more detailed review of respiratory monitoring, see the June 2011 issue of *EM Critical Care*, “Respiratory Monitoring In The Emergency Department.”

**Disposition**

The majority of patients receiving sedation will be admitted to an ICU. On occasion, the emergency physician may need to arrange an interfacility transfer in order to route a patient to definitive care. It is important to ensure that patients are transferred with appropriately credentialed personnel and necessary equipment. Furthermore, the mode of transport represents an informed medical decision. Aeromedical transportation is usually reserved for patients requiring a time-sensitive, life-preserving intervention. Ground-based transport is appropriate for critical care transportation over shorter distances. Note that advanced life support ambulances may not provide specialized equipment (such as balloon pumps or chest tube drainage systems). Interfacility transport of critically ill patients may therefore involve specialized providers (such as critical care nurses, respiratory therapists, or perfusionists).

**Summary**

The sedation of critically ill patients is challenging, and emergency physicians should be familiar with recent guidelines. The paradigm of analgosedation has gained traction as the foundation for any solid sedation plan. Pain is aggressively treated, and sedative medicines are administered in conjunction with

---

**Must-Do Markers Of Quality ED Critical Care: Checklist For Sedation**

Critically ill patients routinely experience pain at rest and while receiving critical care, and procedural pain is common. Although the authors are not aware of a specific checklist that has been validated for use in the ED, we have created the following checklist to ensure that pain and sedation needs are appropriately addressed in a practical manner. Recommendations are modified from the recently published pain, agitation/sedation, delirium (PAD) guidelines with attention paid to the limitation of resources available in most EDs.

**Routine Assessment And Treatment Of Pain**

1. Can the patient communicate his pain?
   - If yes, self-reported pain assessment is preferred.
   - Assess and document pain using NRS (0-10).
   - If no, use BPS or equivalent (rather than vital signs) in patients who are motor intact.
   - Assess and document pain using BPS (3-12).

2. Is the patient having significant pain?
   - Pain is significant when NRS ≥ 4, or BPS ≥ 6.
   - When in significant pain:
     - Treat pain as soon as possible (< 30 min), using opioids as first-line therapy for nonneuropathic pain.
     - Ensure patient is in a comfortable position and that tubes, lines, and monitoring equipment are properly positioned.
     - Use bolus-dose IV opioids and infusions as necessary.

**Routine Assessment For Depth And Quality Of Sedation**

- Reassess frequently (every 2-3 hours, or more frequently with change in vital signs or as otherwise needed).
- Provide preemptive analgesia for planned procedures.
- Consider consultation for thoracic epidural analgesia for patients with traumatic rib fractures.

**Routine Assessment For Depth And Quality Of Sedation**

- Use RASS (-5 to +4) or SAS (1 to 7) to assess and document depth of sedation.

1. Is the patient appropriately sedated?
   - Target lightest possible level of sedation (RASS of -2 to 0, or SAS of 3-4). When sedation is suboptimal:
     - Treat pain first.
     - If undersedated, supplement with nonbenzodiazepine sedatives, when possible (unless alcohol or benzodiazepine withdrawal is suspected).
     - If oversedated, hold sedatives until at target, then restart at 50% of previous dose.
   - Use nonbenzodiazepines for sedative infusion when possible (dexametomidine or propofol).
   - Use EEG to monitor suspected nonconvulsive seizure activity.
   - Use appropriately trained and experienced personnel.
   - Maintain continuous cardiopulmonary monitoring (cardiac, SpO₂, ETCO₂).
valuated assessment scores. Newer agents (such as remifentanil and dexmedetomidine) deserve consideration, given their ability to treat both pain and agitation. Communication with ED administration and pharmacy personnel ensures adequate availability of preferred sedative agents. We encourage communication with your department’s administration and pharmacy to inquire about the availability of these newer medications, as availability may be limited. Selection of any single agent is predicated upon an understanding of the particular side effects and mechanisms of action. Generally speaking, opioids are considered first-line agents. Benzodiazepines, propofol, and other sedatives supplement an opioid infusion and treat persistent pain and agitation. Renal and liver dysfunction occur in ICU patients, and sedative agents may require dose adjustment in patients with organ failure. Published guidelines (such as those from the Society of Critical Care Medicine) may assist intensivists with the monitoring and adjustment of sedative infusions. These guidelines can also aid emergency physicians in early implementation of sedation plans that focus on appropriate analgesia, in hopes of improving outcomes for critically ill patients throughout their hospital course.

Case Conclusions

After troubleshooting the ventilator settings and verifying patency of the endotracheal tube, you collaborated with the nursing staff on a plan for patient sedation. Both patients received an initial 100-mcg bolus of fentanyl. The bedside nurses documented the patients’ levels of sedation with the RASS scale.

In the case of the 35-year-old asthma patient with respiratory failure, breakthrough agitation was mitigated through the use of a propofol infusion, and you administered the propofol in accordance with existing ED sedation protocols. The potential for a prolonged ED length of stay prompted you to add lactate level and serum triglycerides to the patient’s order set.

In the case of the elderly nursing home patient with septic shock, a dexmedetomidine infusion was prepared to supplement sedation needs after his pain was controlled. Mean arterial pressures improved with the use of continued fluid resuscitation and norepinephrine infusion. Once proper analgesia was achieved, dexmedetomidine infusion rates remained low and the patient regained hemodynamic stability.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.


33. Candiotti KA, Gan TJ, Young C, et al. A randomized, open-label study of the safety and tolerability of fospropofol for patients requiring intubation and mechanical ventilation in the intensive care unit. Anesth Analg. 2011;113(3):550-556. (Randomized controlled study; 60 patients)


43. Strom T, Martinussen T, Toft P. A protocol of no sedation for critically ill patients receiving mechanical ventilation: a randomised trial. Lancet. 2010;375(9713):475-480. (Randomized controlled trial; 140 patients)


5. Propylene glycol is often used as a solvent in parenteral formulations of lorazepam. Toxicity from prolonged infusions may cause which of the following?
   a. Hyperosmolar metabolic acidosis
   b. Hypotension
   c. Arrhythmias
   d. All of the above

6. Which of the following clinical scenarios best describes the clinical entity known as propofol-related infusion syndrome?
   a. Confusion, acidosis, hypotension
   b. Refractory lactic acidosis
   c. Metabolic acidosis and hypotension
   d. Renal failure, acidosis, hypertriglyceridemia, and rhabdomyolysis

7. How is ketamine’s mechanism of action best described?
   a. Works through the N-methyl-D-aspartate receptor to produce a state of dissociative anesthesia
   b. Potentiation of GABA receptors
   c. Blockade of serotonin reuptake and GABA agonism
   d. Centrally acting alpha-2 receptor agonist

8. Adverse effects associated with the use of ketamine include:
   a. Emergence delirium
   b. Hypotension and bradycardia
   c. Bronchoconstriction
   d. Increased intracranial pressure

9. How is dexmedetomidine’s mechanism of action best described?
   a. N-methyl-D-aspartate receptor antagonism
   b. Potentiation of GABA receptors
   c. Blockade of serotonin reuptake and GABA agonism
   d. Centrally acting alpha-2 receptor agonist

10. Adverse effects associated with the use of dexmedetomidine include:
   a. Emergence delirium
   b. Bradycardia
   c. Hypertension
   d. Increased intracranial pressure
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Walking The Tightrope: Pain Management And Sedation In The Hypotensive Patient

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