Treating Agitation With Dexmedetomidine in the ICU

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Patients in the intensive care unit frequently experience delirium, anxiety, and agitation, with a variety of treatments used. This article discusses the role of an α-adrenoceptor agonist, dexmedetomidine, and its clinical relevance and advantages for the agitated patient.

Keywords: Agitation, Anxiety, Dexmedetomidine, Sedation

The intensive care unit (ICU) can be an uncomfortable, fear-provoking environment for many patients. Frequently, they experience delirium, anxiety, and agitation. In addition, patients may be subjected to untreated or intractable pain, invasive procedures, prone positioning, sleep deprivation, or adverse drug effects.

Untoward effects of their anxiety and agitation include difficulty breathing, patient-ventilator dysynchrony, hypertension, and tachycardia, as well as combative behavior. Consequently, in addition to receiving analgesics, sedatives are commonly required to reduce agitation and anxiety.

The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) set standards for monitoring pain and anxiety assessment. Because sedation is an essential component of therapy in the ICU, JCAHO has now required scales for such, with the same competence in assessment and monitoring being expected. In 2002, the Society of Critical Care Medicine (SCCM), along with the American Society of Health-System Pharmacists, updated clinical practice guidelines for the continued use of sedatives. These practice guidelines established preferences for sedation in providing the calmest, least agitated, and easily aroused patients. Minimizing agitation and anxiety has been shown to reduce ICU length of stay.

Dexmedetomidine, a selective α2-adrenoceptor agonist, is an effective sedative agent for critically ill patients. The purpose of this article was to discuss the use of dexmedetomidine as an alternative to current commonly used therapies for managing agitation in critically ill patients.

INCREASING AGITATION IN THE ICU

Agitation is defined as an increased irritable tense state that can lead to confusion, excessive psychomotor activity, and hostility. It can have an acute onset or a slow progression lasting as long as it is not treated. In the ICU, agitation represents a sign of an unaddressed health issue. When it lasts for hours, changes in consciousness may produce delirium.

Patients in the ICU experience anxiety and/or agitation for the partial reason that they are placed in a foreign environment. They are then further subjected to confinement to a bed while connected to wires, tubes, and machines. Patients experience stress from a lack of self control, anxiety, and fear. The patient’s perception of his or her general health, type of surgery and anesthesia received, postoperative pain, and disorientation all influence agitation and anxiety. The uncertainty about their future with a loss of independence and a possible fear of death further contribute to anxiety and agitation. Feelings of helplessness, pain, confusion, and memory loss with sleep deprivation aggravate agitation and anxiety.

Already known with pain, anxiety in postsurgical patients has been shown to impair immune responses.
Care providers need to ensure adequate sedation as these surgical patients recover from general anesthesia. Certain medications commonly used in the ICU, for example, zolpidem and furosemide, have been shown to contribute to agitation. Fluid and electrolyte imbalances and temperature fluctuations in the environment are also contributors. Alarms, lights, and environmental noise will also impact the patient’s degree of stress.

**CONSEQUENCES OF AGITATION AND SEDATION ON THE ICU PATIENT**

Agitation and delirium in the ICU patient, while triggering a cascade of increased requirements for relief, are associated with prolonged hospitalizations. Inadequate treatment of agitation initiates the physiological stress response resulting in tachycardia, hypertension, and hyperglycemia, all of which contribute to increased mortality and morbidity in critically ill patients. We know that these responses are primarily mediated through the autonomic nervous system. When the cortex of the brain perceives a threat, the adrenal glands are stimulated to release epinephrine. Respiratory rates increase and become shallow, the heart rate increases, and blood pressure is elevated. Blood is shifted away from the stomach, and glycogenolysis is accelerated with resulting elevated glucose levels. Stress ulcers occur from an increase in gastric acid production.

If not adequately comforted, restless and impaired attention with poor concentration will be seen in the anxious and agitated ICU patient. Inappropriate neurological assessments can occur from a lack of patient cooperation as a result of fatigue and sleep disturbances. Recent studies reveal that patients in the ICU sleep less than 2 hours per day. Using adequate sedation medications must be included in the ICU nurses arsenal of abilities to prevent the somatic effects of tachycardia, hypertension, hyperventilation, tremors, diaphoresis, and palpitations, thus further preventing the secondary behavioral and cognitive responses of agitation and anxiety.

**DIAGNOSIS AND TREATMENT OF ACUTE AGITATION**

Nurses frequently identify agitation when patients exhibit incomprehensible speech, inappropriate screaming, and dysfunctional behavior such as searching their bed for escape routes without acknowledging their safety or are unable to communicate appropriately and become aggressive with hitting or kicking. A pastiche of therapeutic approaches has been shown to alleviate stress and anxiety in critically ill patients. Nonpharmacological techniques, which should be considered first, include repositioning and implementing “sleep hours” and family support at the bedside. When the need for sedation becomes more, pharmacological therapies will include opioid narcotics, benzodiazepines, and barbiturates. Often administered concomitantly, they have a synergistic effect and are beneficial in this patient population.

The central motivation for sedation is to minimize physiological stress rooted by neurosis or psychosis from untreated anxiety and agitation. Patients who have adequate sedation should also be free of respiratory depression, anxiety, and pain; are easily aroused to their surroundings; and hemodynamically stable. The pharmacological agent should have a rapid onset and short half-life, be easily titrated, and lack serious side effects. Manifestations of pain, delirium, anxiety, and agitation are similar; however, they have different etiologies and require different interventions. Therefore, the initial step in the diagnostic evaluation is to draw on reliable assessment tools. Critical care clinicians have used various assessment tools in an effort to comply with JCAHO sedation requirements. The use of any scale enables reaching targeted goals for adequate sedation while maintaining consistency among users. The Ramsay Sedation Scale and the Riker’s Sedation-Agitation Scale are 2 common types, with the former the most widely used in many ICUs. The optimal goal with this scale is for the patient to score a 3 (see Table 1). The goal is a 2 with the Riker’s Sedation-Agitation Scale (see Table 2).

Commonly used pharmacological agents for sedation include propofol, opioids, benzodiazepines, and barbiturates. Each of these agents is associated with adverse side effects, including potential prolongation of

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the ICU stay (see Table 3). Each of these agents also varies in pharmacokinetics, an important consideration when treating acute agitation.

Propofol (Diprivan), mainly used in the operating arena for induction or maintenance of general anesthesia, is a potent sedative and hypnotic anesthetic. It is titrated to desired clinical effects via continuous intravenous (IV) infusion. It is hypothesized that it resembles benzodiazepines with enhancing \(+\)-aminobutyric acid (GABA), a compelling inhibitory neurotransmitter, affinity for its receptors. It can provide conscious as well as unconscious sedation. Its rapid onset of action (30-45 seconds) facilitates titration and minimizes oversedation. One pharmacokinetic mechanism propofol has is a weak analgesic effect at hypnotic concentrations. In approximately 50% of patients, unconsciousness is reached at 3.3 \(\mu\)g/mL.\textsuperscript{11} To suppress all movements, greater than 12 \(\mu\)g/mL is required.\textsuperscript{11} Hypotension and respiratory depression are adverse effects with higher doses.\textsuperscript{12}

Long-term use or ICU sedation makes monitoring triglycerides a necessity. Because the drug is in a lipid emulsion, after 2 days of infusion, triglycerides can be elevated, and its caloric intake should be included in the nutrition prescription.\textsuperscript{11} Lidocaine is often administered before propofol because of pain experienced with initial infusion. Manufacturers recommend changing the tubing and infusion every 12 hours because of the propensity for bacterial growth with the lipid emulsion.\textsuperscript{11}

Additional disadvantages of propofol include untoward problems such as chelation of trace metals, especially zinc, or allergic reactions in asthmatics as a result of preservatives added to propofol.\textsuperscript{3} High-dose propofol can cause “propofol infusion syndrome,” which results in the development of cardiac and renal failure, rhabdomyolysis, and severe metabolic acidosis.\textsuperscript{12} Therefore, propofol for agitation in the ICU is more or less a second resort. Propofol is superior for anesthesia goals.

Like propofol, benzodiazepines enhance the inhibitory tone of GABA receptors, but they are usually given in combination with an opioid, providing a synergistic sedative effect. They also have a rapid central nervous system (CNS) onset of action (2-5 minutes).\textsuperscript{9,13} They provide relief of anxiety at lower doses than if just required for sedation. Lorazepam (Ativan), diazepam (Valium), and midazolam (Versed) are the more common drugs used as sedative agents in the United States.\textsuperscript{9}

According to the SCCM, midazolam is recommended for short-term use only because of its unpredictable awakening time.\textsuperscript{14} The SCCM also recommends midazolam and diazepam for rapid sedation.\textsuperscript{14} Because midazolam has an accumulation effect, lorazepam is preferred for intermittent or a longer continuous infusion.\textsuperscript{14} It also has a

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Guidelines for SAS assessment

1. Agitated patients are scored by their most severe degree of agitation as described.
2. If patient is awake or awakens easily to voice (“awaken” means responds with voice or head shaking to a question or follows commands), that is an SAS 4 (same as calm and appropriate—might even be napping).
3. If more stimuli such as shaking is required but patient eventually does awaken, that is an SAS 3.
4. If patient arouses to stronger physical stimuli (may be noxious) but never awakens to the point of responding yes/no or following commands, that is an SAS 2.
5. Little or no response to noxious physical stimuli represents an SAS 1. This helps separate sedated patients from those you can eventually wake up (SAS 3), those you cannot awaken but can arouse (SAS 2), and those you cannot arouse (SAS 1).

Abbreviation: SAS, Sedation-Agitation Scale.
potent amnesic effect that combines with its satisfactory sedative effect.

The adverse effect of benzodiazepines, with their cumulative ability, will cause respiratory and cardiac depression, hypotension, physical dependence, and sometimes a paradoxical agitation. Prolonged sedation and unpredictable awakening from accumulation of the drug or their active metabolites make them difficult to adjust dosing, thus causing a prolonged recovery. Their amnesic effect may reduce communication, leading to less cooperation during weaning from mechanical ventilation.

In the elderly, especially those with comorbidities, the use of benzodiazepines adds more challenges to healthcare practitioners. Effects such as inadequate clinical assessment, excessive prescribing, and altered pharmacokinetics and pharmacodynamics occur with advancing age. The likelihood to potential side effects increases as a result of changes in distribution and elimination of medications. Benzodiazepines with their long half-lives can accumulate in the body. As we age, our CNS receptors change, becoming more sensitive with increased short-term memory losses and unsteadiness. These are due to normal characteristics of aging such as dementia, hypoalbuminemia, or chronic renal failure. The risk of falls increases with the elderly’s increased psychomotor impairments. Obese patients may require higher doses, but clearance remains relatively the same.

That the primary hesitate issue for treating anxiety and agitation with benzodiazepines is the duration of sedation is not precise enough. There are many clinical caveats in the typical ICU patient. These include inadequate blood flow and metabolic rates, low cardiac outputs or little cardiac reserve, and at times respiratory depression when benzodiazepines are given with pain medications.

We know that agitation and anxiety can be narrowed when pain is treated with opioids. This class of drugs has some advantages. Common ones, such as morphine and fentanyl, are easily titratable to effect with rapid onsets of action. They are dose dependent within their class for sedation and analgesia.

Treating agitation solely with opioids is not recommended. Acute and chronic tolerances can occur. The adverse effect when increasing administration can cause ventilatory depression, hypotension, nausea, and vomiting. Again, when administered with benzodiazepines, it becomes difficult to wean patients from mechanical ventilation.

Some opioids cause vasodilation as a result of histamine release. These opiates cause histamine release from mast cells, resulting in hypotension, urticaria, tachycardia, and pruritus. Morphine, codeine, and meperidine are commonly used opioids that tend to execute this with the effect being dose dependent. Toxicity doses of meperidine can cause dysphoria and agitation. Although life-threatening opioid allergies are rare, caution must be taken with any patient who has shown a past sensitivity to an opioid.

Fentanyl, another common analgesic, has been found to have a low histamine release. Unfortunately, fentanyl can potentially cause bradycardia. Fentanyl inhibits synaptic activity to cardiac vagal neurons. The direct mechanism is not fully understood, but it is believed that one mechanism is the depression of heart rate resulting from the drug inhibiting the frequency and amplitude of GABA neurotransmissions. These transmissions come from the nucleus ambiguous, the site of heart rate and cardiac function origin. Griffioen et al reported that this inhibition was mediated at both presynaptic and postsynaptic sites. Their studies resulted in the demonstration that fentanyl acts on µ-opioid receptors on cardiac vagal neurons and neurons preceding them to reduce GABA neurotransmission and increase parasympathetic activity.

There are many clinical caveats in the typical ICU patient.
Opioids are fine for pain relief, but not solely for anxiety. Euphoria, defined as a feeling of happiness or well-being sometimes exaggerated in pathological states as mania, can be achieved with opioids. Reality is then diminished from the critically ill patient.

Sedating patients with an increase in intracranial pressures or convulsions requires barbiturates. These powerful drugs are similar to benzodiazepines. Barbiturates generate myocardial depression and vasodilation, resulting in tachycardia and hypotension by inhibiting calcium uptake.

Sedation goals for the ICU patient include controlling anxiety for procedures and/or events. An important goal also is blunting the adverse autonomic and hemodynamic responses from the above medications while facilitating mechanical ventilation. This blunting limits self-extubations and reduces oxygen consumption. This results in the enhancement of nursing care of the ICU patient as a result of a more comfortable patient. The ideal sedative allows patients to sustain spontaneous ventilation, protect their airway, and remain cooperative and easily arousable.

Dexmedetomidine (Precedex), an α2-adrenergic receptor agonist inhibits norepinephrine and epinephrine centrally and peripherally. No other agent alone in the current therapeutic arsenal for pain, sedation, and analgesia can provide relief for all. It has a rapid onset (5 minutes) resulting in both sedation and analgesia with no respiratory depression. It is often compared with clonidine, a one-eighth times less sedating α2 agonist with similar properties. Potency for its α2 effect is 1,620 times more than dexmedetomidine’s α1.

Physiological effects of dexmedetomidine include hypotension and bradycardia, reduction of plasma catecholamine levels, and sedation, thereby reducing analgesic and anesthetic requirements. Dexmedetomidine, although used alone, can provide more adequate sedation and pain relief and less anxiety with limited respiratory depression. The drug’s predictable effects of bradycardia and hypotension increase its manageability.

The drug facilitates patient compliance and comprehension with easy arousability. Allowing communication with the nurse and other healthcare providers, while remaining sedated, enables a more comfortable stay for the patient. It is specifically indicated for intubated and mechanically ventilated patients in the ICU and does not have to be discontinued before extubation. In clinical trials, dexmedetomidine has shown significant efficacy for sedation in postsurgical patients as well as those who are agitated and overly anxious uncooperative patients.

### DEXMEDETOMIDINE AND AGITATION

#### A Brief Review of α1 and α2, β1 and β2

Effects of the sympathetic nervous system (SNS), whether from inflammatory responses or drugs, are differentiated into the α and β receptors in the SNS. The activation of their receptors produces either excitatory effects in some tissues or inhibitory effects in others (see Table 4). When the SNS is stimulated, we experience an increase in heart rate and vasconstriction with α. resulting elevated peripheral vascular resistance. Therefore, when these receptors are blocked, or the α2 receptors stimulated, the opposite effect takes place. For this paper, the α2-adrenoreceptors are discussed, with dexmedetomidine being its agonist or stimulator.

Sympatholytic drugs block the sympathetic adrenergic system on 3 levels: centrally, peripherally, and postganglionically. They are primarily adrenergic antagonists, inhibitors of adrenergic receptors, or presynaptic adrenergic agonists inhibiting further impulse transmissions to the sympathetic receptors. The centrally acting drugs block sympathetic outflow within the brain. These medications block the activity of the sympathetic system by binding to and activating the presynaptic α2-adrenoceptors, resulting in a decrease in norepinephrine release, leading to a decreased heart rate and contractility as well as vasodilation and hypotension.

α-Receptors are found presynaptically, postsynaptically, and extrasynaptically. Presynaptic receptors, α2’s, regulate the amount of norepinephrine and adenosine triphosphate through a negative feedback mechanism. Specifically, these α2 receptors are found in the peripheral nervous system and CNS, platelets, and many organs including the kidneys, liver, pancreas, and eyes. The physiological responses mediated by these receptors therefore vary with location.

From an anesthesiologic point, the α2 receptors activate guanine-nucleotide regulatory binding proteins

### TABLE 4 α and β Receptors

| α1 | Arteriolar constriction in skin, mucous membranes, and viscera, causing an increase in peripheral resistance, dilated pupils, and contracted bladder |
| α2 | Reduced sympathetic outflow resulting in peripheral vasodilation |
| β1 | Elevated heart rates (chronoscopic), increased force of cardiac contraction (inotropic), and speed of conduction (dromotropic) |
| β2 | Bronchodilation, dilated arterioles in skeletal muscles resulting in decreased peripheral resistance, increased glycoegenolysis and gluconeogenesis in the liver and bladder, relaxation resulting in decreased urine output |
These activated proteins induce intracellular signaling pathways through a second messenger system, which modulates ion-channel activity in the nerve terminal. The activated second messenger system inhibits adenylate cyclase, an enzyme that decreases the formation of 3,5-cyclic adenosine monophosphate. The modulation of the ion-channel activity leads to hyperpolarization of the cell membrane and an efflux of potassium, resulting in a suppression of neuronal firing. Calcium conductance into cells is reduced from the \( \alpha \)-adrenergic agonist by direct regulation of calcium entry through N-type voltage-gated calcium channels, independent of 3',5'-cyclic adenosine monophosphate and protein phosphorylation. Therefore, this calcium suppression affects analgesia by inhibiting the secretion of neurotransmitters.

Activating the \( \alpha_2 \) receptors in the CNS inhibits neuronal firing and causes hypotension, bradycardia, sedation, and analgesia. Other areas when stimulated result in decreased salivation and decreased bowel motility, contraction of vascular and smooth muscle, inhibition of renin release with an increased glomerular filtration, increased sodium and water retention in the kidneys, decreased intraocular pressure, and decreased insulin secretion.

### PHARMACOLOGY OF DEXMEDETOMIDINE

Dexmedetomidine is a specific and relatively selective \( \alpha_2 \)-adrenoceptor agonist. The drug inhibits the release of norepinephrine at the presynaptic \( \alpha_2 \) receptors, which in turn terminates the beginning of pain signals while postsynaptically activating the \( \alpha_2 \) receptors in the CNS and inhibiting the SNS's elevation of blood pressure and heart rate. The net result of both these pathways produces sedation, anxiolysis, and analgesia.

The suppressed neuronal firing effect produced by the hyperpolarization with the G protein–gated potassium channels and the reduced calcium entry inhibiting the neurotransmitters being released represent 2 specific ways of effecting analgesia. One stops the nerve cell from ever firing, and the other cannot send its signal to another cell.

The sedative effect of dexmedetomidine comes from the largely dense postsynaptic \( \alpha_2 \) receptors found in the locus coeruleus part of the brain (see Figure 1). This is considered the hypnotic or wakefulness modulator area in the brain. The locus coeruleus site is also the origin for nociceptive neurotransmission, where \( \alpha_2 \)-adrenergic and opioidergic systems have common effector mechanisms (see Figures 2 and 3). Here, the dexmedetomidine mechanism of action inhibits the G protein that would have increased conduction through potassium channels, thus resulting in sedation.

Being easily arousable while effectively sedated becomes another unique feature of the drug. This is not found with the use of other sedatives.

All of the effects of producing analgesia, sedation, and anxiolysis make dexmedetomidine unique by producing less side effects than from using multiple agents. Even while intubated, patients are calm, comfortable, and cooperative.

### Administration

The recommended maintenance dose ranges from 0.2 to 0.7 µg/kg. For conscious intubations, 0.5 µg/kg per hour is typically used. The initial loading dose, if required, is 1 µg/kg over 10 minutes. Normal response is within 6 minutes following IV administration. An IV infusion pump should be used to control the dosage to the desired level of clinical sedation. There is no difference in the pharmacokinetics of dexmedetomidine in young versus elderly and female versus males.

Dexmedetomidine is compatible with lactated Ringer’s solution, D5% W, mannitol, etomidate, vecuronium, succinylcholine, glycopyrrolate, phenylephrine, atropine, midazolam, morphine, and fentanyl, among others. Dexmedetomidine infusions should not be continued after 24 hours because its safety and effectiveness have not been evaluated past this time period. Like long-term use of clonidine, dexmedetomidine shares a comparable withdrawal syndrome. Adverse effects include hypotension, hypertension, nausea, bradycardia, atrial fibrillation, and hypoxia. Overdosage may cause first- or second-degree heart blocks. These effects are seen during or briefly after loading the drug.

![Figure 1. Region of dense \( \alpha_2 \) receptors.](image-url)
Distribution
Dexmedetomidine has a distribution half-life of approximately 6 minutes. It has a 93.7% average protein binding with no renal impairment effect. Patients with hepatic impairment may experience changes in protein binding, resulting in lowered clearance of the drug. There is no significant change in protein binding when in the presence of other typical ICU medications such as digoxin, lidocaine, warfarin, phenytoin, and propranolol.

Elimination
The drug is excreted in the urine (95%) and feces (4%) after extensive biotransformation by the liver. Elimination half-life for dexmedetomidine is approximately 2 hours.

Adverse Effects of Dexmedetomidine
Bradycardia, along with various atrioventricular blocks and reduction of sympathoadrenergic hypertension, is the most prominent adverse effect associated with dexmedetomidine. Cardiovascular events are predictable because of the known effects. These effects may cause severe problems during ventilator weaning and during transition to the wake state.

THE RESPONSIBILITY OF THE ACUTE CARE NURSE PRACTITIONER AND CRITICAL CARE NURSE
First, the drug dexmedetomidine should be administered only by clinicians skilled in the management of the ICU patient. These patients must be continuously monitored. Transient hypertension has been seen during the loading dose with its peripheral vasoconstrictive effects. Generally, treatment is not required, but reducing the loading dose rate can be advantageous. Caution should be exercised in patients with any history of advanced heart blocks when infusing dexmedetomidine. Clinicians should be prepared to intervene if hypotension or bradycardia occurs with the infusion. Treatment should include reducing or stopping the rate of infusion while administering fluids or vasopressors. Pretreating with glycopyrrolate, a potent antimuscarinic, is a safe management tool in preventing a potential adverse bradycardia. Sometimes, having available atropine or pacing at the bedside is required for unfavorable bradycardia.

Although compatibility has not been established, nothing should be administered with blood products. The drug must be diluted in 0.9% sodium chloride before administration. Clearance can be slower with hepatic impairment. Therefore, it may be necessary to reduce the dose in patients with liver disease. If dexmedetomidine is administered continually then abruptly discontinued, withdrawal
symptoms, which include nervousness, agitation, rapid hypertension, and headaches, can occur.35

CONCLUSION

Oversedation can cause respiratory depression, psychological distress, metabolic abnormalities, a depressed immune system, and a prolonged difficult weaning from mechanical ventilation. Certain physiological processes are masked or overlooked. On the other end, under-sedation will cause its own share of problems from disorientation, ventilator intolerances, and hemodynamic instability such as hypertension and tachycardia, thus increased oxygen consumption and demand.

Dexmedetomidine will enable intubated and ventilated patients to be more arousable and responsive while reducing analgesic and anesthetic use. There is no respiratory depression, hemodynamic stability is maintained, and oxygen demand is minimized.37

The benefits of dexmedetomidine, being a selective and specific α2-adrenergic receptor agonist, are clinically effective for sedation in the ICU. Dexmedetomidine represents a significant advance in managing and supporting ICU patients.

References


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