# Regional Anesthesia in Anesthetized or Heavily Sedated Patients

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The American Society of Regional Anesthesia and Pain Medicine (ASRA) Practice Advisory on Neurologic Complications in Regional Anesthesia and Pain Medicine includes an evidence- and expert opinion-based section on performing procedures on anesthetized or heavily sedated patients. This practice advisory is based on existing scientific literature, pathophysiological principles, and expert opinion. The advisory panel examined the ability of anesthetized or heavily sedated patients to recognize and report intravascular injection of local anesthetic or impending neurologic injury. The advisory panel also considered whether or not the ability to recognize and report symptoms could actually affect the occurrence of nerve injury or local anesthetic systemic toxicity. The advisory contains recommendations pertaining to both adult and pediatric patients. *Reg Anesth Pain Med 2008;33:449-460.*

**Key Words:** Nerve injury, Regional anesthesia, Pain medicine, Local anesthetic toxicity, Peripheral nerve block.

When properly performed, regional anesthesia is a safe clinical practice with a risk of serious complication that is not significantly different than that of general anesthesia. This report focuses on an area of particular controversy—whether or not it is safe to perform regional anesthesia or pain medicine procedures on patients who are anesthetized or heavily sedated. We define the *anesthetized patient* as one who is under general anesthesia. A *heavily sedated patient* is one who is sedated to the point of being unable to recognize and/or report any sensation that the physician would interpret as atypical during block placement. Given the variability in response to sedative/hypnotics and analgesics that might be used for sedation, it is impossible to provide dosage guidelines or drug recommendations that clearly draw a line between "light" and "heavy" sedation.

Those who routinely perform regional blocks in anesthetized or heavily sedated patients argue that this practice increases safety by decreasing the chance that the patient will move suddenly and cause the block needle to impale a vital structure. In addition, they point out that anesthesia or heavy sedation increases patient acceptance and therefore increases the number of patients who will potentially benefit from regional anesthesia/analgesia. Many clinicians who perform regional anesthesia in infants and children often invoke this latter reasoning, noting that regional blocks would be impractical in the pediatric patient population without anesthesia or heavy sedation.

Those who eschew the practice of performing blocks in anesthetized or heavily sedated patients assert that doing so removes important early warning signs that help prevent both local anesthetic systemic toxicity and neurological injury. Their basic assumption is that the awake or minimally sedated patient will be able to report developing symptoms of systemic local anesthetic toxicity before a toxic dose is injected or will be able to recognize and report pain or other atypical symptoms from an errant needle before neurological injury occurs. While this reasoning seems logical, it is as unproven as are the assertions of those who advocate performing blocks in anesthetized or heavily sedated patients.

In this article, we review the available literature in

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an effort to come to a consensus as to whether the risk of complications from regional anesthesia procedures is increased or decreased by anesthesia or heavy sedation. The data reviewed necessarily consist of case reports, large and small observational human studies, and experimental animal studies, because there are no prospective, randomized, controlled clinical trials aimed at evaluating the impact of anesthesia or heavy sedation on the risk of complications from regional anesthesia or pain medicine procedures. Indeed, such studies may never be conducted because of logistical difficulties associated with performing a properly controlled study that examines an event as rare as anesthesia-related nerve injury. Consequently, we are left to draw conclusions from indirect sources.

## Systemic Local Anesthetic Toxicity

Systemic toxicity from local anesthetics is manifest either within the central nervous system (CNS) or the cardiovascular system, with CNS toxicity occurring at significantly lower plasma concentrations than cardiovascular toxicity.

### Central Nervous System Toxicity

The reported incidence of seizures during regional anesthesia varies between approximately 0.1 and 1 per thousand, with the lower incidence reported by the French SOS Regional Anesthesia Hotline Service,[1] which used a voluntary reporting methodology, and the higher incidence obtained from a retrospective chart review from the Mayo Clinic.[2] Local anesthetic plasma concentrations high enough to cause seizures can be reached either by unintentional intravascular (venous or arterial) injection, systemic absorption from the perineural or epidural injection site, or a combination of both.

Human studies of local anesthetic CNS toxicity demonstrate that if plasma concentrations rise slowly, subjects will progress through a fairly stereotypic series of CNS symptoms prior to developing seizures.[3,4] The early CNS symptoms of rising local anesthetic plasma concentration include tongue or circumoral numbness followed by "lightheadedness" and then visual or auditory disturbances. Consequently, one could reasonably argue that an awake patient attuned to the symptoms of early local anesthetic CNS toxicity would be able to warn a clinician of developing CNS toxicity prior to seizures and that if the local anesthetic is being injected slowly enough, the injection could be aborted before a dose large enough to cause seizures (or worse) is administered. Consistent with this argument are studies demonstrating that an unpremedicated subject can detect an intravenous bolus of lidocaine (1.5 mg/kg), 2-chloroprocaine (90 mg), or bupivacaine (25 mg) with 100% sensitivity, but that the sensitivity decreases to between 60% and 80% with even small doses of sedatives or opioid analgesics (e.g., 1.5-2.8 mg midazolam and 60-96 $\mu$g fentanyl).[5,6]

The argument that aware patients could meaningfully detect an intravascular injection of local anesthetic is appealing, but is not universally applicable. For example, seizures that result from systemic absorption of local anesthetic generally occur after most or all of the local anesthetic has been injected; thus premonitory symptoms typically occur too late to prevent a toxic dose from being administered. Moreover, seizures that occur as a result of unintentional local anesthetic injection into the carotid or vertebral arteries during stellate ganglion or interscalene blocks have occurred after as little as 1.5 mL of local anesthetic were injected.[7] Similarly, patients in whom local anesthetic is unintentionally and rapidly injected intravenously may develop seizures before they have time to recognize and report CNS symptoms and prevent the administration of a toxic dose. In all of these groups of patients, the fact that they may be unanesthetized and unsedated provides no discernable benefit and in some cases may actually increase the risk of CNS and potentially cardiovascular toxicity (see below).

Several studies have demonstrated that the use of an appropriate local anesthetic "test-dose" (e.g., epinephrine, isoproterenol) can help identify unintentional intravascular local anesthetic injection.[8] Importantly, the dose of epinephrine or isoproterenol and the diagnostic criteria for considering a cardiovascular response to be positive may be different in anesthetized versus awake patients (and in "elderly" patients[9]) but the sensitivity is still high if the appropriate test dose and criteria are used.[10-15] In contrast, patient report of CNS symptoms can never be 100% sensitive because of the large number of patients incapable of either sensing or adequately communicating their symptoms (e.g., young children, demented patients, patients with a language barrier).

Therefore, although experimental reports indicate that unsedated/unanesthetized patients who are verbal and fully cognizant can detect and report symptoms of intravascular injection of local anesthetics, this situation is not universally applicable in clinical practice. Because an appropriate test dose that is properly applied and monitored is virtually 100% effective at detecting intravascular injection in patients regardless of their premedication, the test dose, and not patient report, should be considered a more reliable method to detect or prevent intravascular injections that might lead to systemic toxicity.

Moreover, appropriate sedation can actually decrease the risk of seizures.[16-18] Sedative hypnotics

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