Dexmedetomidine as a sole sedating agent with local anesthesia in a high-risk patient for axillofemoral bypass graft: A case report

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The α_2-agonist dexmedetomidine is indicated for sedation of patients receiving mechanical ventilation in the intensive care unit. It has additional off-label uses for coadministration with local, regional, and general anesthesia. This report describes the use of dexmedetomidine as a sole sedating agent in conjunction with local anesthesia for major vascular surgery.

A PubMed literature search produced no previous report of the use of dexmedetomidine as a sole sedating agent used in conjunction with local anesthesia. The anxiolytic, hypnotic-sedative, anesthetic-sparing, and analgesic actions of the drug along with the lack of significant respiratory depressant effects are described. The patient required no airway management with the exception of supplemental mask oxygen. He tolerated the procedure well and was discharged without sequelae on the third postoperative day.

Dexmedetomidine should be used judiciously, and understanding the potential adverse effects and how to treat them is of paramount importance. However, with vigilant intraoperative monitoring of blood pressure, heart rate, and level of consciousness, it can be administered safely, thus lessening the anesthetic requirements and possibly improving the surgical outcome of the high-risk patient. This report describes the indications, dosing, off-label uses, pharmacodynamics, pharmacokinetics, and common adverse effects of dexmedetomidine.

Key words: Conscious sedation, dexmedetomidine, difficult airway, total intravenous anesthetic agent (TIVA), Troop elevation pillow.

The α_2-agonist dexmedetomidine belongs to a new class of sedating agents that have been shown to be safe and effective.[1,2] It is indicated for sedation in the intensive care unit; however, it has additional off-label uses that can be beneficial during the administration of local, regional, and general anesthesia (Table 1).[2]

Mantz[3] reported that in addition to its cardiovascular properties, dexmedetomidine possesses anxiolytic, hypnotic-sedative, anesthetic-sparing, and analgesic actions and is devoid of significant respiratory depressant effects. He added that because of these properties, dexmedetomidine may be particularly useful during the perioperative period, as well as for sedation of patients in critical care settings.[3] The stress response to surgery and the hemodynamic response to intubation and extubation are decreased as a consequence of the α_2-mediated decrease in sympathetic tone of dexmedetomidine.[1,4-7]

A loading dose of dexmedetomidine of 1 µg/kg over 10 minutes (Table 2) usually is followed by a maintenance dose of 0.2 to 0.7 µg/kg per hour.[2] However, Ramsay and Luterman[9] recently reported that maintenance doses of up to 10 µg/kg per hour were tolerated without hypotension or bradycardia when used as a total intravenous anesthetic agent in certain patients. They demonstrated how dexmedetomidine preserved the respiratory drive so well that virtually no airway management intervention was required (with the exception of a chin lift) during anesthesia care for 3 patients with difficult-to-manage airways.[9] Although this use of high dose dexmedetomidine is controversial,[10,11] Ramsay[12] demonstrated that among other factors, dosing parameters for dexmedetomidine when used as a sole sedating agent should also be dependent on the clinical situation and the experience of the practitioner.

Table 1. Indication and off-label uses of dexmedetomidine[2]

Indication
Indicated as a continuous infusion for short-term sedation (< 24 h) of intubated and mechanically ventilated patients in an intensive care unit (ICU) setting
Off-label uses
Adjunct to regional anesthesia
Adjunct to general anesthesia
Bridge to ICU sedation and analgesia
Supplement to regional block in patients undergoing carotid endarterectomy
Use in craniotomy when the patient must remain awake

Table 2. Loading infusion guide for dexmedetomidine* using a standard solution of 4 µg/mL[8]

Patient's weight (kg)	Loading infusion rate (mL/h)	Total volume to be infused (mL)
50	75.0	12.5
55	82.5	13.8
60	90.0	15.0
65	97.5	16.3
70	105.0	17.5
75	112.5	18.8
80	120.0	20.0
85	127.5	21.3
90	135.0	22.5
95	142.5	23.8
100	150.0	25.0
105	157.5	26.3
110	165.0	27.5
115	172.5	28.8
120	180.0	30.0

*** Dexmedetomidine generally is initiated with a loading infusion of 1.0 µg/kg over 10 minutes.**

A recent review described the pharmacodynamics and pharmacokinetics of dexmedetomidine.[2] It is pharmacologically related to clonidine, an α_2-agonist but has 8 times more affinity for α_2 receptors than does clonidine. It produces sedation and anxiolysis by binding to α_2 receptors in the locus ceruleus, which diminishes the release of norepinephrine and inhibits sympathetic activity, thus decreasing heart rate and blood pressure. It produces analgesia by binding to adrenoreceptors in the spinal cord.[2]

Dexmedetomidine has an onset of action of 30 minutes, which is slower than that of midazolam (3-5 minutes) and propofol (10-50 seconds). Use of a standard loading dose (1 µg/kg infused over 10 minutes[2]; see Table 2) will decrease the onset of action. Dexmedetomidine has a duration of action of 4 hours, which is longer than that of midazolam (3-80 minutes) and propofol (3-10 minutes). It has an offset time of 5 minutes, whereas offset in midazolam is 2 to 6 hours and in propofol is 3 to 8 minutes. Complete biotransformation is accomplished by glucuronidation and cytochrome P-450–mediated metabolism. The effects of dexmedetomidine may be significantly increased when it is coadministered with anesthetics, sedative hypnotics, or opioids.[2]

At my institution, dexmedetomidine is used to decrease the requirement for general anesthesia in some high-risk patients for various major surgical procedures. It also is used as a sedating agent in high-risk patients for cases such as carotid endarterectomy with cervical plexus block and in endographic abdominal aortic aneurysm repair with local anesthesia supplementation. In addition, it has been used here as a sole intravenous anesthetic agent for general anesthesia in some patients with difficult-to-manage airways.[9] It provides a pharmacological bridge for postoperative mechanical ventilation (eg, abdominal aortic aneurysm repair [see Table 1]) following general endotracheal anesthesia. However, it is not without adverse effects and requires close perioperative monitoring[1] in the operating room, postanesthesia care unit, and intensive care unit (Tables 3 and 4). A recent article associated the use of dexmedetomidine with the occurrence of severe bradycardia (see Table 3) progressing to asystole.[14]

Case report

A high-risk patient with a potentially difficult-to-manage airway underwent a left axillofemoral bypass graft receiving dexmedetomidine as the sole sedating agent with local anesthesia supplementation. His only option beyond axillofemoral bypass graft was a leg amputation, which he adamantly refused. The vascular surgeon requested that no general anesthesia be used because of the patient's debilitated state and difficult postoperative course following a previous surgery. The consensus was that he would be extremely difficult to wean from postoperative ventilation, and should it be required, he most likely would not survive.

The patient was a 70-year-old man with congestive heart failure and severe cor pulmonale, severe chronic obstructive pulmonary disease with three pillow orthopnea, hypertension, coronary artery disease, atrial fibrillation with runs of nonsustained ventricular tachycardia, pacemaker, severe peripheral vascular disease, and end-stage renal disease with hemodialysis. His ejection fraction was 35%. He weighed 72 kg and was 182 cm tall. Airway assessment revealed a potentially difficult-to-manage airway with a Malampati classification of 3 and no range of motion of the atlanto-occipital joint.[15] He underwent dialysis the morning of surgery. Medications included amlodipine, losartan, aspirin, furosemide; fluticasone/salmeterol; famotidine; calcium acetate; and prednisone.

Vital signs were as follows: blood pressure, 160/90 mm Hg; heart rate, 88 beats per minute. Laboratory results were as follows: hemoglobin, 9.7 mg/dL; hematocrit, 32.3%; sodium, 135 mg/dL; potassium, 4.2 mg/dL; chloride, 103 mg/dL; serum urea nitrogen,

Table 3. The most common adverse events (from most to least frequently occurring) with intravenous dexmedetomidine[2]

Hypotension*	Vomiting
Hypertension*	Hypoxia
Nausea	Tachycardia
Bradycardia*	Anemia
Fever	

*Can develop with rapid infusion.

Table 4. The Ramsay Sedation Scale[13]

Ramsay sedation score	Explanation
1	Patient is anxious and agitated or restless, or both
2	Patient is cooperative, oriented, and tranquil
3	Patient responds to commands
4	Patient exhibits brisk response to light glabellar tap or loud auditory stimulus
5	Patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus
6	Patient exhibits no response

42 mg/dL; and creatinine, 3.4 mg/dL. The arterial blood gas values were as follows: FIO_2, 0.21; pH, 7.33; pCO_2, 44 mm Hg; pO_2, 93 mm Hg; SaO_2, 98%; acid base index, –3; HCO_3^-, 23 mg/dL.

Routine monitors were applied. A Doppler was used to facilitate placement of a radial artery catheter because the right radial pulse was not palpable.[16] Because of the patient's inability to lie flat, he rested in a low-Fowler position on a Troop elevation pillow (Mercury Medical, Clearwater, Fla)[17] and a standard intubation pillow to support his head. A Capnoxygen mask (Southmedic, Barrie, Ontario, Canada) with oxygen at 5 L/min was used to monitor exhaled carbon dioxide. After administering a standard loading dose (see Table 2) of 1 µg/kg of dexmedetomidine over 10 minutes, a maintenance infusion of up to 0.7 µg/kg per hour was administered. An adequate level of sedation was attained (Ramsay score, 4)[13] (see Table 4) within 10 minutes, enabling the surgeon to painlessly inject 1% lidocaine before skin incision. Further injections of local anesthetic were used without complaint during the remainder of the case.

A remifentanil infusion was added in an attempt to ensure complete comfort during the surgical creation of the tunnel. However, despite the use of a low-dose infusion (0.025 µg/kg per minute) over 2 minutes, there was a precipitous drop in blood pressure (systolic blood pressure, 76 mm Hg); however, it was quickly attenuated with two 100-µg doses of phenylephrine and immediate cessation of the remifentanil. No further use of vasopressors or narcotics was required, and surgical tunneling produced no noticeable discomfort. The patient was arousable and appropriately responsive[13] (Ramsay score, 4) throughout the surgical procedure, which lasted 101 minutes with a total anesthesia time of 145 minutes. His blood pressure averaged 110 to 140 over 63 to 78 mm Hg; his respiratory rate was 20 to 25 breaths per minute, and exhaled carbon dioxide measured 28 and 33 mm Hg. Following an uneventful stay in the postanesthesia care unit, he was transferred to the nursing unit from which he was discharged without complications on the third postoperative day.

Discussion

Dexmedetomidine was chosen instead of propofol because of the respiratory depression that may occur with maintenance infusions of propofol.[18] As experienced with this patient, one should be alert for hypotension if dexmedetomidine is coadministered with anesthetics, sedative hypnotics, or opioids.[2] Even without coadministration, a rapid infusion of dexmedetomidine may cause significant hypotension (see Table 3).[2] Knowing the duration of action of dexmedetomidine to be 4 hours, one should be vigilant to monitor for hypotension well into the postoperative period.

When dexmedetomidine is used as a bridge (see Table 1) to enable the patient to tolerate tracheal intubation into the postoperative period following general anesthesia, a maintenance infusion (without a loading dose) should be started about 30 minutes before the end of the general anesthetic. Another consideration in using dexmedetomidine is cost because it is significantly more expensive than generic propofol.[2] However, Ramsay and Luterman[9] noted that even though the cost is potentially higher than that of conventional anesthetics, the advantage of no loss of respiratory drive and good analgesia without the need for opioids may justify the increased expense. In addition, obviating the need for general anesthesia and the possible need for postoperative ventilation also might justify the increased cost of dexmedetomidine.

A PubMed literature search of 524 titles did not reveal a previous case describing the use of dexmedetomidine as a sole sedating agent with local anesthesia. However, previous reports of its use in conjunction

with regional and general anesthesia exist (see Table 1).[2] Understanding the potential adverse effects of dexmedetomidine (see Table 3) and how to treat them is of paramount importance. It should be used judiciously but should receive consideration for use as a sedating agent in appropriate high-risk candidates in whom general anesthesia may not be beneficial. It should also receive consideration as a bridge after general anesthesia for high-risk patients requiring postoperative tracheal intubation with or without controlled ventilation. With vigilant perioperative monitoring of blood pressure, heart rate, and level of consciousness (see Tables 3 and 4), it can be administered safely, thus lessening anesthetic requirements and possibly improving the surgical outcome for high-risk patients.

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