RESEARCH PAPER

Severe Bradycardia Associated with Dexmedetomidine Administration During Surgery: A Case Series

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Abstract

Background

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist widely used in anesthetic practice because of its sedative, analgesic, anxiolytic, and opioid-sparing properties while preserving spontaneous respiration. Despite its favorable pharmacological profile, dexmedetomidine may produce significant cardiovascular adverse effects, particularly bradycardia and hypotension. Although most episodes are mild and transient, severe bradycardia requiring immediate intervention may occur during the perioperative period. This case series describes the clinical presentation, management, and outcomes of five patients who developed severe dexmedetomidine-induced bradycardia during surgery.

Case Series

Five patients undergoing elective surgical procedures under general anesthesia or regional anesthesia with dexmedetomidine sedation developed severe intraoperative bradycardia following dexmedetomidine administration. The onset of bradycardia occurred after either the loading dose or during continuous infusion and was associated with hypotension in four patients. Heart rates ranged from 26 to 34 beats/min. Immediate discontinuation of dexmedetomidine, administration of intravenous atropine or glycopyrrolate, fluid resuscitation, and vasopressor therapy when indicated resulted in rapid restoration of hemodynamic stability. None of the patients required cardiopulmonary resuscitation, temporary cardiac pacing, or prolonged intensive care. All patients recovered completely without postoperative cardiovascular or neurological complications.

Conclusion

Severe bradycardia is an uncommon but clinically important adverse effect of dexmedetomidine that may occur even in patients without significant pre-existing cardiac disease. Continuous electrocardiographic and hemodynamic monitoring, individualized dosing strategies, early recognition of cardiovascular deterioration, and prompt therapeutic intervention are essential to ensure patient safety. This case series reinforces the importance of careful patient selection and adherence to evidence-based monitoring and management protocols for the safe perioperative use of dexmedetomidine.

Keywords: Dexmedetomidine; Severe bradycardia; General anesthesia; Regional anesthesia; Intraoperative complications; α2-Adrenergic agonist; Hemodynamic instability;

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.Introduction

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist that has become an integral component of modern anesthetic practice because of its sedative, analgesic, anxiolytic, and sympatholytic properties. Unlike many conventional sedative agents, dexmedetomidine provides cooperative sedation with minimal respiratory depression, making it particularly useful during procedural sedation, awake fiberoptic intubation, intensive care unit sedation, and as an adjunct to general and regional anesthesia. In addition, it reduces perioperative opioid requirements, attenuates stress responses, and facilitates smooth emergence from anesthesia.[1-4]

Despite its favorable pharmacological profile, dexmedetomidine is associated with dose-dependent cardiovascular adverse effects, particularly bradycardia and hypotension. These hemodynamic effects result primarily from central sympatholysis, reduced norepinephrine release, enhanced vagal activity, and diminished sympathetic outflow. While mild reductions in heart rate are common and generally well tolerated, severe bradycardia may lead to profound hypotension, sinus arrest, atrioventricular block, or even cardiac arrest, particularly in susceptible patients.[1,2,5]

The reported incidence of dexmedetomidine-induced bradycardia varies widely depending on patient characteristics, dosage, infusion rate, concomitant medications, and surgical procedures. Patients with high baseline vagal tone, advanced age, pre-existing conduction abnormalities, hypovolemia, beta-blocker therapy, calcium channel blocker use, neuraxial anesthesia, or procedures associated with vagal stimulation are at greater risk of developing clinically significant bradycardia.

Rapid loading doses and higher maintenance infusion rates have also been associated with increased cardiovascular instability.[3,6,7]

The mechanism underlying dexmedetomidine-induced bradycardia is multifactorial. Activation of central α2-receptors suppresses sympathetic nervous system activity while enhancing parasympathetic tone, resulting in reduced sinoatrial node automaticity and delayed atrioventricular nodal conduction. Peripheral α2B receptor stimulation may initially produce transient vasoconstriction and hypertension, followed by prolonged hypotension due to central sympatholysis. These physiological changes may become clinically significant in patients with limited cardiovascular reserve or during periods of surgical vagal stimulation.[1,4,8]

Early recognition of severe bradycardia is essential because delayed intervention may result in reduced cardiac output, impaired organ perfusion, and cardiovascular collapse. Management includes immediate reduction or discontinuation of dexmedetomidine infusion, administration of intravenous fluids when indicated, atropine or glycopyrrolate for symptomatic bradycardia, vasopressor support for persistent hypotension, and epinephrine or temporary cardiac pacing in refractory cases. Continuous electrocardiographic and hemodynamic monitoring throughout dexmedetomidine administration is therefore recommended.[2,5,9-11]

Although dexmedetomidine is widely regarded as a safe anesthetic adjunct, reports of severe bradycardia continue to appear in the literature, emphasizing that this complication may occur even in patients without significant cardiovascular disease. Increased awareness of predisposing factors, careful patient selection, individualized dosing strategies, and prompt recognition of adverse cardiovascular effects are essential for improving patient safety.[6,12-14]

This case series describes five patients who developed severe bradycardia during surgery following dexmedetomidine administration. The report highlights the clinical presentation, contributing risk factors, management strategies, and favorable outcomes achieved through early diagnosis and prompt intervention. These cases emphasize the importance of vigilant perioperative monitoring and adherence to evidence-based recommendations for the safe use of dexmedetomidine in anesthetic practice.[5,10,15]

Case series

Case 1

A 65-year-old male (ASA II) with controlled hypertension underwent laparoscopic cholecystectomy under general anesthesia. Following induction, a loading dose of dexmedetomidine (1 μg/kg over 10 minutes) was administered, followed by a maintenance infusion of 0.5 μg/kg/h. Approximately 20 minutes after skin incision, the patient's heart rate gradually decreased from 78 to 34 beats/min, accompanied by hypotension (82/48 mmHg). The dexmedetomidine infusion was immediately discontinued, intravenous atropine (0.6 mg) was administered, and rapid crystalloid infusion was initiated. Heart rate increased to 68 beats/min within three minutes, blood pressure normalized, and surgery proceeded uneventfully. The patient recovered without postoperative cardiovascular complications.

Case 2

A 72-year-old female (ASA III) with type 2 diabetes mellitus and ischemic heart disease was scheduled for total knee arthroplasty under combined spinal anesthesia with dexmedetomidine sedation. After receiving a loading dose of dexmedetomidine (0.8 μg/kg), the patient developed profound sinus bradycardia (heart rate 30 beats/min) associated with hypotension (74/42 mmHg). Dexmedetomidine infusion was discontinued immediately, oxygen therapy was initiated, intravenous atropine (0.6 mg) and ephedrine (6 mg) were administered, resulting in rapid improvement of heart rate and blood pressure. The remainder of the procedure was completed successfully without recurrence, and postoperative ECG showed no conduction abnormalities.

Case 3

A 48-year-old male (ASA I) underwent tympanoplasty under general anesthesia with dexmedetomidine infusion for controlled hypotensive anesthesia. Thirty-five minutes after commencement of the infusion, manipulation near the vagally innervated middle ear resulted in sudden severe bradycardia with a heart rate of 26 beats/min and transient sinus pause. The surgical stimulus was immediately stopped, dexmedetomidine infusion was discontinued, and intravenous glycopyrrolate (0.2 mg) was administered. Heart rate rapidly recovered to 72 beats/min without the need for cardiopulmonary resuscitation. Surgery was completed successfully, and the patient had an uneventful postoperative recovery.

Case 4

A 59-year-old female (ASA II) underwent thyroidectomy under general anesthesia. Dexmedetomidine was administered as an adjunct for attenuation of sympathetic responses during surgery. Approximately 45 minutes after initiation of the infusion, she developed severe bradycardia (heart rate 32 beats/min) associated with hypotension (78/46 mmHg). The infusion was stopped immediately, intravenous atropine (0.6 mg) and fluid boluses were administered, followed by a low-dose phenylephrine infusion because hypotension persisted briefly. Hemodynamic stability was restored within five minutes, and the remainder of the surgery was completed without

further complications. She was discharged on the third postoperative day in stable condition.

Case 5

A 55-year-old male (ASA II) underwent lumbar spine decompression surgery under general anesthesia with dexmedetomidine infusion for opioid-sparing analgesia. Approximately one hour after surgery began, the patient developed progressive sinus bradycardia (heart rate 28 beats/min) followed by hypotension (70/40 mmHg). Dexmedetomidine infusion was discontinued immediately, intravenous atropine (0.6 mg), ephedrine (6 mg), and crystalloid infusion were administered. The patient's heart rate improved to 74 beats/min, and blood pressure normalized within several minutes. No further episodes occurred, postoperative cardiac evaluation was unremarkable, and the patient was discharged without residual cardiovascular complications.

Parameter	**Case 1**	**Case 2**	**Case 3**	**Case 4**	**Case 5**
Age/Sex	65/M	72/F	48/M	59/F	55/M
ASA Status	II	III	I	II	II
Surgical Procedure	Laparoscopic cholecystectomy	Total knee arthroplasty	Tympanoplasty	Thyroidectomy	Lumbar spine decompression
Anesthetic Technique	General anesthesia	Spinal anesthesia with sedation	General anesthesia	General anesthesia	General anesthesia
Dexmedetomidine Administration	Loading 1 µg/kg followed by 0.5 µg/kg/h infusion	Loading 0.8 µg/kg for sedation	Continuous infusion for controlled hypotension	Continuous intraoperative infusion	Continuous infusion for opioid-sparing analgesia
Lowest Heart Rate (beats/min)	34	30	26	32	28
Lowest Blood Pressure (mmHg)	82/48	74/42	Stable	78/46	70/40
Clinical Presentation	Severe bradycardia with hypotension	Bradycardia with hypotension	Isolated severe bradycardia	Bradycardia with hypotension	Bradycardia with marked hypotension
Management	Dexmedetomidine stopped; atropine 0.6 mg IV; IV fluids	Infusion stopped; atropine 0.6 mg IV; ephedrine 6 mg IV	Infusion discontinued; glycopyrrolate 0.2 mg IV	Infusion stopped; atropine; IV fluids; phenylephrine	Infusion stopped; atropine; ephedrine; IV fluids
Outcome	Complete recovery	Uneventful recovery	Heart rate normalized	Stable recovery	Discharged without sequelae
Anesthetic Technique	General anesthesia	Spinal anesthesia with sedation	General anesthesia	General anesthesia	General anesthesia

Discussion

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist that has gained widespread acceptance in anesthetic practice

because of its sedative, anxiolytic, sympatholytic, and opioid-sparing properties without causing significant respiratory depression. It is commonly used as an adjunct during general anesthesia, regional anesthesia, and procedural sedation. Despite these advantages, dexmedetomidine is associated with predictable cardiovascular adverse effects, particularly bradycardia and hypotension, resulting from decreased central sympathetic outflow and enhanced vagal activity. Although mild reductions in heart rate are common, severe bradycardia requiring pharmacological intervention or temporary cessation of the drug is relatively uncommon but may become life-threatening if not recognized promptly.[1–4]

In the present case series, all five patients developed severe intraoperative bradycardia temporally related to dexmedetomidine administration. The episodes occurred during both general anesthesia and regional anesthesia with sedation, demonstrating that clinically significant bradycardia may occur irrespective of the anesthetic technique. The onset of bradycardia ranged from shortly after the loading dose to approximately one hour after continuous infusion, consistent with previous reports describing both early and delayed cardiovascular effects of dexmedetomidine.[2,5]

The principal mechanism of dexmedetomidine-induced bradycardia involves stimulation of central α2-receptors within the locus coeruleus, resulting in inhibition of sympathetic nervous system activity and increased parasympathetic tone. Activation of presynaptic α2-receptors suppresses norepinephrine release, producing reduced heart rate, decreased cardiac output, and hypotension. In addition, a transient peripheral α2-mediated vasoconstrictive response following rapid loading doses may activate baroreceptor-mediated reflex bradycardia, further contributing to profound reductions in heart rate. Concomitant vagal stimulation during surgical manipulation, advanced age, pre-existing cardiovascular disease, hypovolemia, or concurrent use of beta-blockers may further increase susceptibility to severe bradycardia.[1,3,6]

Previous studies have identified advanced age, baseline low heart rate, diabetes mellitus, cardiac conduction abnormalities, high loading doses, rapid drug administration, and prolonged infusion as important predictors of dexmedetomidine-associated bradycardia. In our series, two patients were elderly with significant cardiovascular comorbidities, whereas the remaining patients developed severe bradycardia despite having no major underlying cardiac disease. One patient experienced bradycardia during middle ear surgery, where surgical stimulation of the vagus nerve likely potentiated the chronotropic effects of dexmedetomidine. These findings emphasize that severe bradycardia may occur even in relatively healthy individuals and that multiple perioperative factors often contribute simultaneously.[4,7,8]

Prompt recognition and immediate management are essential to prevent progression to cardiac arrest. In all five patients, discontinuation of dexmedetomidine infusion together with intravenous atropine or glycopyrrolate resulted in rapid restoration of heart rate. Vasopressors such as ephedrine or phenylephrine and intravenous fluid administration were used in patients with associated hypotension. None of the patients required cardiopulmonary resuscitation, temporary cardiac pacing, or prolonged postoperative intensive care. These observations are consistent with current recommendations that emphasize early discontinuation of dexmedetomidine, correction of contributing factors, administration of anticholinergic agents, and use of vasopressors when required.[3,5,9]

The literature indicates that dexmedetomidine-related bradycardia is usually reversible when recognized early. However, isolated reports of asystole and cardiac arrest have been described, particularly in patients receiving high loading doses or those with enhanced vagal tone. Therefore, continuous electrocardiographic and hemodynamic monitoring remains mandatory throughout dexmedetomidine administration. Dose adjustment, slow infusion rates, avoidance of unnecessary loading doses in high-risk patients, and careful patient selection may significantly reduce the incidence of severe cardiovascular complications.[2,6,10]

Our case series highlights the importance of individualized dexmedetomidine dosing based on patient age, cardiovascular status, surgical procedure, and concomitant medications. Elderly patients and those with underlying cardiac disease should receive lower loading doses or maintenance infusions with close hemodynamic surveillance. Furthermore, anesthesiologists should remain vigilant during procedures associated with increased vagal stimulation, as these may potentiate dexmedetomidine-induced bradycardia.[5,8,11]

The major limitation of this case series is its small sample size and descriptive design, which preclude assessment of incidence or risk factors. Nevertheless, the consistent temporal association between dexmedetomidine administration, development of severe bradycardia, prompt response to standard treatment, and complete recovery in all patients strengthens the clinical relevance of these observations. Reporting such cases contributes to greater awareness among anesthesiologists regarding early recognition and appropriate management of this potentially serious adverse effect.[9,12]

Summary

Dexmedetomidine is widely used in contemporary anesthetic practice because of its sedative, analgesic,

anxiolytic, and opioid-sparing properties while preserving spontaneous respiration. However, its potent sympatholytic action may result in clinically significant bradycardia and hypotension, particularly in susceptible patients. This case series describes five patients who developed severe intraoperative bradycardia following dexmedetomidine administration during both general anesthesia and regional anesthesia with sedation. The severity of bradycardia ranged from isolated sinus bradycardia to profound bradycardia associated with hypotension. Early recognition, immediate discontinuation of dexmedetomidine infusion, administration of anticholinergic agents, intravenous fluid resuscitation, and vasopressor support when required resulted in complete recovery in all patients without permanent cardiovascular complications. These cases highlight the importance of vigilant hemodynamic monitoring, individualized dosing, and prompt intervention to prevent progression to life-threatening cardiovascular events.

Conclusion

Dexmedetomidine-induced severe bradycardia is an uncommon but clinically significant perioperative complication that requires immediate recognition and timely management. Although dexmedetomidine remains a valuable anesthetic adjunct because of its favorable sedative and analgesic profile, careful patient selection, dose optimization, avoidance of rapid loading doses in high-risk individuals, and continuous electrocardiographic and hemodynamic monitoring are essential to ensure safe administration. Early discontinuation of the drug, prompt treatment with anticholinergic agents and vasopressors when indicated, and correction of contributing factors usually result in rapid recovery and excellent clinical outcomes. This case series reinforces the need for increased awareness among anesthesiologists regarding dexmedetomidine-associated bradycardia and emphasizes adherence to evidence-based perioperative monitoring and management strategies to optimize patient safety.

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