# Suspected Sinus Arrest After Sugammadex Administration: A Case Report

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We describe a case of profound bradyarrhythmia after sugammadex administration during ambulatory anesthesia. The patient was a 21-year-old man with autism spectrum disorder undergoing planned general anesthesia for dental treatment. After treatment completion, sugammadex was administered upon awakening, and sudden bradyarrhythmia appeared immediately. The patient's heart rate decreased to approximately 30 beats/min but quickly recovered to roughly 80 beats/min after the administration of intravenous atropine. Electrocardiography suggested sinoatrial block or sinus arrest. Although the exact mechanism is unknown, severe electrocardiographic changes can occur within a few minutes of sugammadex administration.

**Key Words:** Sugammadex; Bradyarrhythmia; Sinoatrial block; Sinus arrest.

Electrocardiographic abnormalities associated with the administration of sugammadex have been reported and include bradycardia, tachycardia, QT prolongation, severe atrioventricular block, ventricular fibrillation, ventricular tachycardia, and cardiac arrest. We report the case of a male patient undergoing intubated general anesthesia for dental treatment who experienced acute onset bradyarrhythmia following administration of sugammadex to reverse rocuronium-induced neuromuscular paralysis.

## CASE PRESENTATION

The patient was a 21-year-old man (height, 162 cm; weight, 48 kg; and body mass index, 18.2 kg/m$^2$) with autism spectrum disorder who underwent planned dental treatment under general anesthesia. He had no other significant medical history, family history, allergies, or regular medications, and routine preoperative examination revealed no remarkable findings. The planned dental treatment consisted of restorations on 3 teeth and taking a dental impression.

After application of appropriate anesthetic monitors (electrocardiogram [ECG]; lead II), noninvasive blood pressure cuff, and pulse oximeter), slow mask induction was performed with sevoflurane (0.5%-5%), oxygen, and nitrous oxide. Intravenous (IV) access was secured once the patient was induced, and general anesthesia was maintained with an IV target-controlled infusion (TCI) of propofol at 3 μg/mL (estimated rate, 0.7 mg/kg bolus followed by infusion at 8 to 6 mg/kg/h) along with a remifentanil infusion of 0.3 μg/kg/min. An IV bolus of 50 mg rocuronium was administered, after which nasal intubation was performed without difficulty. General anesthesia was then maintained using air, oxygen, propofol at a TCI of 3 μg/mL, and 0.15 μg/kg/min of remifentanil. A total of 1.8 mL of 2% lidocaine (36 mg) with 1:80 000 epinephrine (0.0225 mg) was administered for local anesthesia via infiltration prior to starting the dental treatment.

During the operation, the patient's systolic blood pressure was 90 to 100 mm Hg, diastolic blood pressure was 40 to 50 mm Hg, and heart rate was 60 to 80 beats/min. The ECG waveform at this time showed a normal sinus rhythm. The duration of the dental treatment was 1 hour 50 minutes. All IV anesthetics were stopped at the end of treatment, and 200 mg of sugammadex was administered to facilitate reversal of rocuronium-induced neuromuscular paralysis. At that time, his systolic blood pressure was 100 mm Hg, diastolic blood pressure was 50 mm Hg, and heart rate was 120 beats/min.

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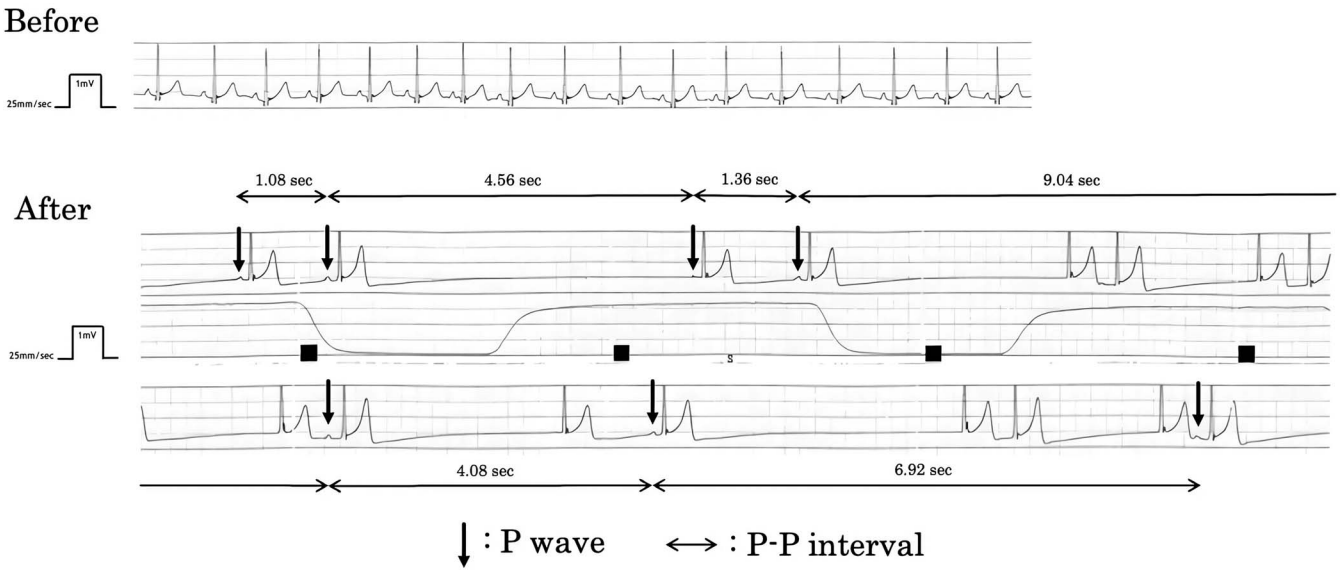
**Figure.** ECG (Lead II) Before and After Sugammadex Administration




Profound bradyarrhythmias noted immediately after IV administration of 200 mg of sugammadex.

Immediately after IV administration of sugammadex, the patient became profoundly bradycardic (~30-40 beats/min), the R-R interval became irregular, and an atrioventricular junctional escape rhythm without P waves appeared. The longest period during which no P waves were observed was approximately 9 seconds (Figure). The patient's blood pressure at this time was 110/70 mm Hg. An IV bolus of 0.5 mg atropine was administered roughly 1 minute after the ECG changes were noted, and approximately 30 seconds later, the patient's heart rate recovered to 80 beats/min with a normal sinus rhythm.

After confirming consciousness, adequate spontaneous breathing, and no other ECG changes, the patient was extubated and transferred to the recovery room. He was discharged home after 2 hours once full recovery from general anesthesia was confirmed.

## DISCUSSION

The bradyarrhythmia observed following sugammadex administration in this case was strongly suspected to be sinus arrest because (1) the P wave disappeared with QRS; (2) the P-P interval was abruptly prolonged, indefinite, and not an integral multiple of the normal P-P interval; and (3) the P-P interval was prolonged by more than 3 seconds or 3-fold the normal P-P interval (Figure). Pühringer et al. reported a 1.9% incidence of bradyarrhythmia as a side effect of sugammadex.[1] Fierro et al. reported that bradyarrhythmias caused by sugammadex suggest a direct effect on the cardiovascular system rather than an effect secondary to anaphylaxis.[2] However, Kojima et al.[3] reported that sevoflurane at induction decreased sinus node automaticity but did not significantly alter clinical heart rate and that propofol, even at the effective blood concentration in clinical use, was partly responsible for bradyarrhythmias due to current suppression in sinus node cells. In clinical practice, bradycardia induced by remifentanil was reportedly caused by vagus nerve stimulation.[3] In the present case, the suppression of sinus function probably became apparent due to the interaction of sugammadex, which has bradyarrhythmia as a potential side effect, and the propofol and remifentanil that were administered throughout the case and stopped immediately before the sugammadex was given.

Although the bradyarrhythmia observed in this case may have been related to the sugammadex administration in a dose-dependent manner, it remains unclear if it was caused by rocuronium-sugammadex inclusion complexes or by sugammadex alone.[4] Therefore, the administration of sugammadex ideally should be based on information obtained from muscle relaxation monitoring (Table). The time elapsed since the last dose of rocuronium, the current depth of neuromuscular blockade, and the potential for adverse effects should be taken into consideration when administering sugammadex to reverse neuromuscular paralysis.

Even when sugammadex is used properly with the guidance of muscle relaxation monitoring, various ECG changes that require emergent management may occur within a few minutes after administration. Preparations should be made to manage potential adverse effects following sugammadex

**Table.** Sugammadex Dosing for Muscle Relaxation States

| *Muscle relaxation state* | *Sugammadex dosing (mg/kg)* | *Ratio of T4/T1 time to recovery to 0.9 (min)* |
|---|---|---|
| Shallow muscle relaxation (reappearance of T2) | 2.0 | 1.5 |
| Deep muscle relaxation (appearance of 1-2 PTCs) | 4.0 | 2.9 |
| Emergency (CICV) (3 min after rocuronium administration) | 16.0 | 3.2 |

Abbreviations: CICV, cannot intubate cannot ventilate; PTC, posttetanic count.

administration including preparing for significant bradyarrhythmias and even cardiac arrest.

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## REFERENCES

1. Pühringer FK, Rex C, Sielenkämper AW, et al. Reversal of profound, high-dose rocuronium-induced neuromuscular blockade by sugammadex at two different time points: an international, multicenter, randomized, dose-finding, safety assessor-blinded, phase II trial. *Anesthesiology*. 2008;109(2):188–197.

2. Fierro C, Medoro A, Mignogna D, et al. Severe hypotension, bradycardia and asystole after sugammadex administration in an elderly patient. *Medicina (Kaunas)*. 2021;57(1):79.

3. Kojima A, Imashuku Y, Kitagawa H, Matsuura H. Ionic mechanism of sinoatrial node automaticity and its modulation by anesthetics [in Japanese]. *Cardiovasc Anesth*. 2018;22(1):19–23.

4. Shimazu R, Nishimura A, Shinohara A, Kato Y, Kawauchi A, Iijima T. Three cases of suspected sugammadex-induced hypersensitivity reactions [in Japanese]. *J Jpn Dent Soc Anesthesiol*. 2018;46(1):1–5.