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Acute Bradycardia Following Propofol and Succinylcholine Induction in a Healthy Patient: A Case Report

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ABSTRACT

This case report examines an episode of acute bradycardia in a healthy 34-year-old female during induction for an elective bilateral salpingectomy. The patient received fentanyl, lignocaine, propofol, and succinylcholine for anesthesia induction. Despite a lack of prior cardiac history, her heart rate dropped from 88 to 40 beats per minute shortly after succinylcholine administration. The bradycardia was promptly resolved with intravenous atropine, allowing surgery to proceed uneventfully. This case highlights the potential for bradycardia with the use of propofol and succinylcholine in anesthesia induction, particularly in the absence of premedication with anticholinergic agents. The case underscores the importance of preoperative assessment and the need for vigilance in monitoring heart rate, especially when using these agents in combination.

Keywords: Propofol; Succinylcholine; Bradycardia; Anesthesia; Atropine; Induction agents; Case report

INTRODUCTION

Propofol and succinylcholine are commonly utilized anesthetic agents around the globe. Propofol (2,6-diisopropylphenol) is favored as an induction agent due to its characteristics, including quick and smooth onset and recovery with minimal adverse effects. It lowers blood pressure, cardiac output, and systemic vascular resistance by inhibiting sympathetic vasoconstriction and disrupting the baroreceptor reflex [1]. Succinylcholine remains the sole depolarizing muscle relaxant used in clinical practice. Its sustained use, despite associated risks, can be attributed to its rapid onset (achieving deep block within 60 seconds), short duration, formation of non-toxic metabolites, and affordability [2]. However, as a mimic of acetylcholine, succinylcholine can stimulate both muscarinic and nicotinic receptors, leading to potentially serious complications such as

bradycardia, asystole, muscle twitching, and increased pressures in the stomach, eyes, and brain. It can also cause prolonged paralysis in patients with plasma pseudocholinesterase deficiency and may trigger malignant hyperthermia [3]. Nonetheless, its short action time makes it a suitable choice for cases involving difficult intubation. While both drugs have the potential to cause bradycardia, this effect is not universally experienced and can often be mitigated through premedication. This report presents a case of abrupt bradycardia in a patient with no prior history of cardiac issues.

CASE PRESENTATION

A 34-year-old female, para 4, was admitted for an elective bilateral salpingectomy for sterilization. She had no significant medical or surgical history and no co-morbidities. Her preoperative evaluation showed normal vital signs: temperature, pulse at 81 beats per minute, and blood pressure at 120/80 mmHg. Her BMI was 28. Systemic examinations of the respiratory, cardiovascular, and central nervous systems were unremarkable. Both her complete blood count and EKG results were normal.

In the operating room, her heart rate increased to 88 beats per minute, and her blood pressure was recorded at 140/90 mmHg. After three minutes of pre-oxygenation, she was induced with fentanyl 175 mcg, 5 ml of 1% lignocaine, and 150 mg of propofol, followed by 100 mg of succinylcholine. Suddenly, her heart rate fell to 40 beats per minute. Atropine 1 mg was administered intravenously, successfully increasing her heart rate back to 75 beats per minute.

She was intubated using a 7 mm Portex endotracheal tube with a 3 mm blade. Bilateral air entry was confirmed, and the tube was secured at the 20 cm mark. The patient was placed on pressure-controlled ventilation with volume guarantee (PCV-VG), set for a tidal volume of 450 ml, a respiratory rate of 12 breaths per minute, and an inspiration-to-expiration ratio of 1:2, with 60% oxygen and a flow rate of 1 L/min using sevoflurane as the anesthetic agent. Rocuronium 40 mg was given as a muscle relaxant, and phenylephrine 300 mcg was administered to manage a decrease in blood pressure.

The surgery was conducted without complications. At the conclusion, the neuromuscular block was reversed with sugammadex 200 mg, and the patient was extubated, recovering normally from anesthesia. She was then moved to the post-anesthesia care unit, where her vital signs were stable, including a blood pressure of 120/80 mmHg, heart rate of 81 beats per minute, respiratory rate of 15 breaths per minute, and a pulse oximetry reading of 96% on room air.

DISCUSSION

In this case study, a 34-year-old female with no significant medical history underwent an elective bilateral salpingectomy. During the procedure, she experienced a transient drop in heart rate to 40 beats per minute after induction with fentanyl, lignocaine, propofol, and succinylcholine. This was promptly treated with atropine. The surgery was uneventful, and she recovered with stable vital signs postoperatively.

Baraka noted that using a sequence of propofol followed by succinylcholine can lead to significant bradycardia in patients not premedicated with atropine. Administering atropine beforehand can help prevent this complication. Unlike thiopental, propofol does not have central vagolytic properties and may instead exhibit a central vagotonic effect, which could amplify the muscarinic effects of succinylcholine. Cullen and Sorensen highlighted the necessity of adequate anticholinergic premedication when utilizing this sequence for anesthesia

induction, especially when combined with centrally vagotonic opioids like fentanyl or following multiple doses of succinylcholine [4-6].

Williams et al. found that succinylcholine-induced cardiac changes in humans are mediated by both sympathetic and parasympathetic nerves [7]. Galindo and Davis elaborated that these changes stem from sympathetic post-ganglionic stimulation, leading to reflex cardiac responses via baroreceptors and the vagus nerve [8]. This aligns with Beretervide's findings in dogs and rabbits, where the use of pentobarbital abolished the reflex response [9].

In young children, cardiac output relies more on heart rate compared to adults, making them particularly vulnerable to bradycardia during anesthesia, especially those with poorer ASA physical status [10].

Keenan et al. demonstrated that propofol significantly decreases heart rate in children under two, with a bradycardia incidence of 1.27% in the first year of life, which is higher than in older, healthier individuals. Although bradycardia can occur with various anesthetic agents, thiopental rarely causes it, though it may lead to arrhythmias. The rapid redistribution of thiopental likely accounts for its brief protective effect, which is why we chose propofol for a smoother induction in this patient [10].

In elderly patients with multiple comorbidities, including cardiac issues, fluctuations in blood pressure and heart rate can occur after propofol administration. Changes induced by succinylcholine are also common in individuals with hyperkalemia or pseudocholinesterase enzyme deficiency [2]. However, our patient was a healthy young adult with no comorbidities who experienced acute severe bradycardia.

CONCLUSION

This case report highlights the rare yet significant occurrence of severe bradycardia following the administration of propofol and succinylcholine in a young, healthy patient with no prior cardiac history. Although the mechanisms behind such hemodynamic changes involve complex interactions between the drugs' vagotonic and parasympathetic effects, careful premedication with anticholinergics like atropine may mitigate this risk. This case emphasizes the importance of vigilant monitoring and preparedness for prompt intervention during anesthetic induction, even in low-risk patients, to ensure patient safety and optimal outcomes.

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