Case 2019.2: Epinephrine and β blockers – Watch Out!

**Situation:** A 55 y/o 65kg female presented for extraction of two carious mandibular molars (#18 and 31) under intravenous moderate sedation and local anesthesia. She reported a history of hypertension which was well managed with carvedilol (Coreg™). Preoperative vital signs included HR 74/min, BP 122/82, SpO2 98 on room air. Sedation was initiated with 40% nitrous oxide and oxygen and 3mg midazolam IV. A bite block was inserted and 1.8cc 2% lidocaine with 1:100,000 epinephrine (36mg lidocaine/18µg epinephrine) was injected to block the left mandible.

Three minutes after injection and before the right mandible was anesthetized, a vital sign check revealed HR 50/min, BP 188/105, SpO2 99. The patient denied any symptoms and responded appropriately to verbal command. The blood pressure returned to baseline 5 minutes later and the first tooth (#18) was extracted without complication. Following extraction of the first tooth, the mandibular right side was anesthetized using 0.9cc 2% lidocaine with 1:100,000 epinephrine (18mg lidocaine/0.9µg epinephrine) and 1.8cc 3% mepivacaine plain (54mg) without a rise in blood pressure. The second procedure was completed without incident.

**What we learned:** This case demonstrates the profound dose-related drug interaction that can occur with epinephrine and non-selective β blockers. While it is common practice to check blood pressure every 5 minutes during moderate sedation, pressures are rarely rechecked when sedation is not administered – and in such a case, this potentially life-threatening interaction would be undiscovered and ignored. Furthermore, as it is common practice to anesthetize all areas of the mouth to be treated at the beginning of the procedure, this dose-related interaction could have been much more severe, had it not been noticed.

Non-selective β blockers block both β2 vasodilation and β1 mediated increase in heart rate. Epinephrine stimulates vasoconstriction (α1), heart rate (β1), and vasodilation (β2). In the presence of non-specific β blockers, epinephrine triggers unopposed vasoconstriction (α1); while compensatory vasodilation and heart rate increase are blocked. The resulting hypertension triggers reflex bradycardia leading to the classic hypertension and bradycardia characteristic of this drug interaction.

**Recommendations:** Continued monitoring of vital signs of patients who are receiving local anesthetics and taking non-selective beta blockers is essential and unrelated to the use of sedation or general anesthesia. Use of non-epinephrine containing local anesthetics or local anesthetics with reduced concentrations of epinephrine should be considered.

**Additional reading:**

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