

Vecuronium and Danger of Vagal Induced Cardiac Arrest during Laparotomy: A Case Report and Literature Review

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Vecuronium bromide, a relatively new nondepolarizing neuromuscular blocker, is frequently cited as a good option for muscular relaxation, since it provides good neuromuscular block and is free from cardiovascular activity¹. However, bradycardia² and even cardiac arrest³ were reported after its use. We report herein a case of a healthy woman who developed asystole a few minutes after the administration of vecuronium and made complete recovery after resuscitation.

Case Report

A 53-year-old Caucasian woman was admitted to our hospital for elective transabdominal hysterectomy. She denied previous chronic diseases and both the physical and the laboratory examinations were normal.

After premedication with meperidine 1 mg·kg⁻¹ and promethazine 0.5 mg·kg⁻¹ administered 30 min before the scheduled surgery time, anesthesia was induced by the intravenous administration of sodium thiopentone 4 mg·kg⁻¹ and 0.1 mg of fentanyl. Fol-

lowing sleep induction, as indicated by at least two of the listed criteria: a) Centralized eyeballs; b) Absent eyelash reflex; c) Reduced jaw tone⁴, vecuronium 0.1 mg·kg⁻¹ was administered for facilitation of endotracheal intubation. The patient was carefully ventilated through a face mask, and after two minutes, when the neuromuscular monitor device (RelaxographTM, Datex Instrumentarium Corp., Helsinki, Finland) showed appropriate muscular relaxation, an 8 mm I.D. endotracheal tube was uneventfully inserted through her mouth into the trachea. Tube position was asserted by bilateral listening of breath sounds and the tube was fixed at place by adhesive tape.

Controlled ventilation was then initiated (Ventimeter[®] Controller II, Air Shields Vickers, Hatboro, PA 19040, USA) with a tidal volume of 10 ml·kg⁻¹ and a frequency set to maintain an end tidal CO₂ of 35 mmHg. Anesthesia was maintained with a mixture of N₂O 4 l·min⁻¹, O₂ l·min⁻¹ and isoflurane 1.5%.

Heart rate, blood pressure, hemoglobin saturation (SaO₂), esophageal temperature and expiratory CO₂ were monitored continuously by the CardiacapTM (Datex Instrumentarium Corp., Helsinki, Finland). For oxygen concentration monitoring, the oxygen

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monitor 143 (Medix LTD, Rehovot, Israel) was attached to the inspiratory limb of the anesthetic circuit.

After induction, the blood pressure decreased from 140/90 to 110/75 mmHg and the pulse rate from 90 to 75 beats·min⁻¹, whereas the SaO₂ remained 99%. The skin and fascia incision was performed without detected changes in the cardiorespiratory parameters, but after opening the peritoneum, during insertion of peritoneal retractors and traction on the wound edges, a sudden profound bradycardia, rapidly followed by cardiac arrest occurred. At this moment, no evidences of hypoxemia (SaO₂=99%) or CO₂ abnormalities (end tidal CO₂=35 mmHg) were observed in the monitor apparatus.

Prompt institution of CPR, ventilation of the lungs with 100% oxygen and intravenous administration of 1 mg atropine, caused return of normal sinus rhythm and blood pressure 15 seconds thereafter.

The surgery continued uneventfully and the postoperative course was without complications. The patient was discharged to her home on the 4th postoperative day without evidences of cardiovascular or neurological deficits. During her hospital stay serial CPK isoenzymes measurements and ECG recording showed no signs of myocardial infarction.

Discussion

Vecuronium is a monoquaternary homologue of pancuronium with a relatively short nondepolarizing muscle relaxant activity and apparently devoid of significant cardiovascular changes when administered in clinical doses^{1,5,6}. Nevertheless, vecuronium can induce bradycardia⁷⁻⁹ and some cases of cardiac arrest after its use were related in the literature^{3,10,11}. The reasons for this reduction in heart rate are poorly understood¹². Cozantis⁸ and Miller¹³

suggest that vecuronium per se is devoid of intrinsic bradycardic activity. They postulate that because of the lack of vagolytic activity of this neuromuscular agent any vagal mediated bradycardia caused by drugs (fentanyl for example) or by surgical stimuli (such this from peritoneal traction) can easily occur. In contrast, Inoue and colleagues¹³ as well as Salmenpera and co-workers¹⁴ suggested that vecuronium possesses an intrinsic bradycardic activity. Whatever the cause is, its incidence appears to be low if the anesthetic technique and the surgery are free from vagal activity¹⁵. Moreover, vagolytic preanesthetic medication apparently protects from this undesirable effect⁷.

Gravlee and colleagues¹⁷ had demonstrated that the association of vecuronium with fentanyl caused a significant decrease in heart rate and cardiac index, although those changes were more evident with sufentanil than with fentanyl. Since the combination of pancuronium with fentanyl provided the greatest overall hemodynamic and electrocardiographic stability, those authors recommend the use of this association in cardiac surgical patients.

In our case, fentanyl which is known to have a strong vagal activity¹⁶ was administered at the beginning of the anesthesia. Furthermore, cardiac arrest occurred during peritoneal traction, which could cause an additional vagal stimulation. As described above, since vecuronium lacks vagolytic activity or has an intrinsic bradycardic action, the vagal stimulus could be strong enough to induce cardiac arrest.

Since vecuronium induced cardiac arrest was previously described one can wonder about the value of this report. However, vecuronium is becoming increasingly popular and most reports postulate that it is safe and free from adverse cardiovascular activity, which can lead many anesthetists

to use it with an exaggerate sense of security. Therefore, it is our intention to remind our readers that the association of vecuronium and fentanyl together with the surgical stimuli can induce a potential lethal complication: cardiac arrest.

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