

OPEN

Key to Prevention of Bradycardia: Be Relax Postoperatively

A Case Report

Tumul Chowdhury, MD, DM, FRCPC and Bernhard Schaller, MD, PhD, DSc

Abstract: Hypotension and bradycardia are commonly observed after the spinal anesthesia and various mechanisms have been postulated for these hemodynamic changes.

A middle-aged otherwise healthy male Caucasian patient developed several episodes of bradycardia postoperatively after the umbilical hernia repair under subarachnoid block (SAB) while trying to lean forward and move his legs. Episodes were aborted when patient was advised to relax in supine position.

The common mechanism of bradycardia and hypotension under SAB is postulated as sympathetic blockade, decrease venous return, and parasympathetic over-dominance leading to a decrease in right arterial pressure and pressure in the great veins as they enter the right atrium. But over time, the parasympathetic inhibition is usually withdrawn first, leading to the risk of severe bradycardia that is probably favored by the reverse Trendelenburg position as described in our case.

Postoperative severe hemodynamic changes can occur even under stable spinal anesthesia; however, can be prevented by vigilant monitoring and simple maneuver which includes maintenance of relax posture on the bed.

(*Medicine* 95(22):e3733)

Abbreviations: BP = blood pressure, HR = heart rate, PACU = postanesthesia care unit, SAB = subarachnoid block, T = thoracic.

INTRODUCTION

Hemodynamic perturbations are common under anesthesia. Hypotension and bradycardia are commonly observed after the spinal anesthesia and various mechanisms have been postulated for these hemodynamic changes.¹ In addition, surgical manipulation of different neural structures also causes similar changes.^{2,3} Notably, majority of cases had been occurred just after initiation of the anesthesia or intraoperatively during surgical manipulation.

In this case, we have reported (written informed consent has been obtained) the postoperative severe hemodynamic

changes in otherwise stable patient and further tried to elucidate the cause, mechanism, and management.

CASE REPORT

A middle-aged male Caucasian patient was scheduled for umbilical hernia repair under subarachnoid block (SAB). This patient was otherwise healthy and his laboratory investigations were unremarkable as well. All standard anesthetic monitors were applied. His baseline vitals included heart rate (HR) of 70 bpm and blood pressure (BP) of 140/82 mm Hg. In sitting position and under all aseptic precautions, SAB was performed with 25 G Whitacre spinal needle (first attempt) and 1.3 ml of 0.75% bupivacaine with 15 µg fentanyl was administered. Patient was lied down supine and abolition of ice-cold perception was noted till T8 dermatome. Monitored anesthesia care plan was instituted with intravenous injection of midazolam 2 mg and propofol infusion at 25 to 50 µg/kg/min. Surgery was uneventful and throughout the procedure, vitals remained fairly stable (HR –60 to 80 bpm and BP –130/80 to 110/60 mm Hg). The blood loss was minimal and overall fluid intake was 700 ml. At the conclusion of surgery, propofol infusion was stopped and patient was shifted to postanesthesia care unit (PACU). After 15 minutes of observation at PACU, the patient developed several episodes of transient severe bradycardia (30–35 bpm) accompanied with hypotension (73/40 mm Hg) while he was trying to lift his legs and to look around. Electrocardiographic strip obtained from the monitor showed sinus bradycardia. Attending nurse immediately altered to anesthesiologist and checked the block level that was coinciding with T12 level (ice-cold sensation). It was clearly evident that each episode of bradycardia was coincided with patient's upper body movement in trying to lift his legs. It was also observed that the recovery bed was in slight reverse Trendelenburg position. Anesthesiologist advised the patient that he needed to relax and not to try moving, bending forward and exerting and kept the bed flat. Injection 0.4 mg glycopyrrolate intravenously was also administered. Patient's hemodynamics reverted back to normal and the rest of the PACU course was uneventful.

DISCUSSION

Hemodynamic changes from mild to severe type may occur intraoperatively due to various mechanisms. One of the most frequent causes remains the anesthetic one.^{2,3} Both general and spinal anesthesia can incite various cardiovascular perturbations; fortunately, most of them are moderate and do not require any specific treatment. The common mechanism of bradycardia and hypotension under SAB is postulated as sympathetic blockade (inhibition of cardioaccelerator fibers T1–T4), decrease venous return and parasympathetic over-dominance leading to a decrease in right arterial pressure and pressure in the great veins as they enter the right atrium.⁴ Frequently, these manifestations are associated with mid thoracic levels (T4–T6); however, it can also be noted in lower level blocks, therefore

Editor: Maria Kapritsou.

Received: March 11, 2016; revised: April 26, 2016; accepted: April 27, 2016.

From the Department of Research, University of Southampton, Southampton, UK (BS), and Department of Anesthesiology and Perioperative Medicine, University of Manitoba, Winnipeg, Manitoba, Canada (TC).

Correspondence: Tumul Chowdhury, Department of Anesthesiology and Perioperative Medicine, University of Manitoba, Winnipeg, Manitoba, Canada (e-mail: tumulthunder@gmail.com).

TC and BS are Academic Editors of *Medicine*.

The authors have no conflicts of interest to disclose.

Copyright © 2016 Wolters Kluwer Health, Inc. All rights reserved.

This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial License 4.0 (CCBY-NC), where it is permissible to download, share, remix, transform, and buildup the work provided it is properly cited. The work cannot be used commercially.

ISSN: 0025-7974

DOI: 10.1097/MD.0000000000003733

explains the other possible mechanisms. Interestingly, in the present case, the level of SAB was low thoracic level and patient's intraoperatively course was uneventful; though the blockade level was high. Therefore, a decrease in venous return most probably was not the primary cause for severe bradycardia during spinal anesthesia in our patient. The dosing (volume and concentration) of intrathecal bupivacaine is also a potential contributing factor for negative hemodynamic changes; however, addition of low dose intrathecal fentanyl (10–30 µg) decreases the total dose of bupivacaine, increases the onset of blockade and enhances the analgesic effects. Therefore, it should not be a factor in the present case as well.⁵

We demonstrate here, a case with modulation of sympathovagal balance after spinal anesthesia by reverse Trendelenburg position. At the beginning of spinal anesthesia, there was a decrease in sympathetic and parasympathetic activity in our patient, as it is generally known. Nevertheless, over time, probably favored by the reverse Trendelenburg position, the usual predominant parasympathetic inhibition withdrawn has been overshooting leading to the above-described severe bradycardia. As acetylcholine released at the vagal nerve endings diminishes the release of norepinephrine from neighboring sympathetic terminals, decreased cardiac sympathetic activity is explained as direct and indirect effects.^{6–8} This pathophysiological explanation underlines also that atropine has and cannot have any effect in such cases^{8–11} and that relaxing is favorable. Since the mechanisms of bradycardia during perioperative period are multifactorial and may be difficult to evaluate accurately. Therefore, objective examinations including HR variability or Valsalva maneuver can be performed to clarify the mechanisms of bradycardia. As this patient was otherwise healthy and showed relatively stable hemodynamics intraoperatively, we did not perform such tests.

Our observation is of particular importance as not only it adds important knowledge about the autonomous nerve system to the literature but also as such patients would have been discharged from the PACU before the occurrence of the severe bradycardia in normal clinical practice. It is common phenomenon in the postoperative period, that patients want to look around, to move their legs, try to sitting up, that can cause similar dreaded responses; therefore, reminding them the simple maneuvers (such as just relax on the bed) in the postoperatively can prevent these catastrophic consequences.

CONCLUSION

In conclusion, severe hemodynamic perturbations in postoperative period can be anticipated even under stable spinal anesthesia intraoperatively; however, vigilant monitoring and maintaining a relax posture on the bed may prevent such episodes.

REFERENCES

1. Kinsella SM, Tuckey JP. Perioperative bradycardia and asystole: relationship to vasovagal syncope and the Bezold–Jarisch reflex. *Br J Anaesth*. 2001;86:859–868.
2. Meuwly C, Chowdhury T, Sandu N, et al. Anesthetic influence on occurrence and treatment of the trigemino-cardiac reflex: a systematic literature review. *Medicine (Baltimore)*. 2015;94:e807.
3. Karaman T, Demir S, Dogru S, et al. The effect of anesthesia depth on the oculocardiac reflex in strabismus surgery. *J Clin Monit Comput*. 2015. [Epub ahead of print].
4. Schaller B. Physiology of cerebral venous blood flow: from experimental data in animals to normal function in humans. *Brain Res Rev*. 2004;46:243–260.
5. Gwirtz KH1, Young JV, Byers RS, et al. The safety and efficacy of intrathecal opioid analgesia for acute postoperative pain: seven years' experience with 5969 surgical patients at Indiana University Hospital. *Anesth Analg*. 1999;88:599–604.
6. Schaller BJ. Ketamine and decrease of oculocardiac reflex. *Acta Neurochir (Wien)*. 2007;149:737–738.
7. Meuwly C, Chowdhury T, Schaller B. Topical lidocaine to suppress trigemino-cardiac reflex. *Br J Anaesth*. 2013;111:302.
8. Chowdhury T, Mendelowitz D, Golanov E, et al. Trigemino-cardiac reflex: the current clinical and physiological knowledge. *J Neurosurg Anesthesiol*. 2015;27:136–147.
9. Schaller B, Sandu N, Filis A, et al. Peribulbar block or topical application of local anaesthesia combined for paediatric strabismus surgery. *Anaesthesia*. 2008;63:1142–1143.
10. Ponhold H, Vicenzi MN. Incidence of bradycardia during recovery from spinal anaesthesia: influence of patient position. *Br J Anaesth*. 1998;81:723–726.
11. Meuwly C, Golanov E, Chowdhury T, et al. Trigeminal cardiac reflex: new thinking model about the definition based on a literature review. *Medicine (Baltimore)*. 2015;94:e484.