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Calm recovery, safe rhythm: Preventing bradycardia after surgery

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Abstract
Bradycardia and hypotension are recognised complications of spinal anaesthesia, often attributed to autonomic imbalance and reflex mechanisms. We report a case of sudden, profound bradycardia in a healthy 35-year-old male immediately following bilateral inguinal hernia repair under spinal anaesthesia. The episode occurred as the patient attempted to move and observe the surgical field, suggesting a vasovagal response triggered by anxiety and physical straining. This case highlights the importance of vigilant postoperative monitoring and patient counselling to mitigate autonomic disturbances, even in American society of anaesthesiologist's class I individuals.

Keywords: Bradycardia, Autonomic nervous system, Vigilance

Introduction
Bradycardia and hypotension are not uncommon post spinal anesthesia due to various mechanisms through either direct or indirect effects and the reflexes activated by our autonomic system varies within individuals due to sympathovagal imbalance. This attributes to strict vigilance of monitoring hemodynamic alterations throughout intraoperative course as well as postoperatively. Anxiety and straining of patients in the ware off period of spinal anesthesia in order to move the limbs causes activation of parasympathetic system due to mechanisms that differ within individuals' autonomy causing bradycardia and hypotension.
We report such case where a 35-year-old healthy male, developed bradycardia immediately post-surgery, for bilateral inguinal hernia done under spinal anesthesia, before shifting the patient to PACU (Post anesthesia care unit)

Case Report
A 35-year-old healthy male belongs to American society of anaesthesiologist's (ASA) class 1, was posted for bilateral inguinal hernia surgery.
Preoperative investigations nil significant. After obtaining written, informed consent shifted the patient to Operation theatre. All standard ASA monitors were connected. Baseline vitals were recorded- Heart rate (HR) of 74 beats per minute, Blood pressure (BP) of 130/82 mm Hg, pulse oximetry showing saturation of 100% at room air, Electrocardiography (ECG)- normal sinus rhythm.
Under aseptic precautions, Sub arachnoid block (SAB) was performed with 25 Gauge(G) Qunicke spinal needle, free flow of cerebrospinal fluid (csf) noted and 2.8 ml of 0.5% Bupivacaine (heavy) with 25mcg fentanyl was administered intrathecally. Patient was made to lie down in supine position and checked for the spinal anesthesia level by appreciating abolition of cold sensation which was noted till T6 dermatome. Surgery went uneventful, throughout the surgery patient had stable hemodynamics. Surgical dressing was applied over the area of operation and we informed patient that operation was completed.
Immediately patient made attempts to look over to the surgical field and strained to make an effort to lift his lowerlimbs, suddenly the heart rate decreased down gradually from 70 to 45 and further even to 28 within 30 seconds, ecg showed bradycardia and blood pressure recorded at that time showed 80/48 mm hg. We have administered injection Atropine 0.6 mg and asked the patient to stop moving and made him relax. Heart rate picked up and blood pressure was normalised and shifted the patient to PACU.

Discussion

Bradycardia following spinal anaesthesia is a well-recognised complication, often attributed to autonomic imbalance, particularly in young, healthy individuals with high baseline vagal tone. The mechanisms are multifactorial, involving both direct neural blockade and reflex-mediated cardiovascular responses.

Sympathetic blockade from spinal anaesthesia (typically T1–L2) leads to vasodilation, reduced venous return, and decreased cardiac preload, which can activate cardiac mechanoreceptors and trigger reflex bradycardia. This is classically described as the Bezold–Jarisch reflex (BJR), a cardioinhibitory reflex mediated by unmyelinated vagal C fibres in the wall of left ventricle [1]. However, not all cases of bradycardia post-spinal anaesthesia can be explained by BJR alone.

Study conducted by Ponhold *et al*. states that higher incidence of bradycardia in patients with Trendelenburg position than patients in the horizontal or hammock position [2]. They contradicted the theory for bradycardia stated by Greene *et al* that reduced venous return due to peripheral vasodilatation that is sympathetic blockade activates right atrial cardiac receptors and by reflex mechanisms slow down the heart rate.[2] In our case, throughout intraoperative course there were stable hemodynamics and the level of SAB at the end of surgery was low thoracic and therefore decreased venous return might not be the primary reason for developing bradycardia making BJR less likely. Addition of fentanyl low dose (25 mcg) to the hyperbaric bupivacaine is for additive analgesic purpose and to increase the onset of the spinal blockade. So, the drug dosing and the volume cannot contribute as a factor for bradycardia.

Instead, the timing and context—straining to move limbs, attempting to visualise the surgical field—suggest a vasovagal mechanism. A study by Vahabi *et al*. reported a significant incidence of vasovagal responses during spinal anaesthesia, particularly in patients with heightened emotional reactivity or inadequate anxiolysis [3].

Higher centres involving cortex and hypothalamus have a descending pathway to the centers in medulla which trigger the response. Emotional stress, anxiety and pain can cause vasovagal response. Animal experiments showed two important centers present at higher level that regulates the cerebral circulatory control. First one is the defence reaction area that is lying in the limbic lobe and latter is the limbic sympatho inhibitory center. Excitation of the former one causes increase in heart rate and blood pressure that arouses human reactions resembling to anxiety and stimulation of latter causes bradycardia and furtherly hypotension [4].

This case reinforces the importance of anticipating autonomic disturbances in the immediate postoperative period. Patient counselling to avoid straining, adequate anxiolysis, and vigilant monitoring in PACU are essential preventive strategies. Furthermore, PACU staff should be trained to recognise early signs of bradycardia and initiate prompt treatment, even in patients deemed low risk.

Conclusion

Bradycardia following spinal anaesthesia may result from parasympathetic overactivity triggered by emotional stress and physical straining during recovery. Early recognition, patient reassurance, and vigilant postoperative monitoring are essential to prevent adverse autonomic events.

Conflict of Interest

Not available

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References

1. Bekele Z, Abebe F. Cardiac Arrest Due to Severe Vasovagal Response After Spinal Anaesthesia in Caesarean Section: Case Report. IJS Oncology. 2022 Jan 1;7(1):15-9.
2. Ponhold H, Vicenzi MN. Incidence of bradycardia during recovery from spinal anaesthesia: influence of patient position. Br J Anaesth. 1998 Nov;81(5):723-6. doi: 10.1093/bja/81.5.723. PMID: 10193283.
3. Vahabi S, Beiranvand S, Radpay B. The Incidence of Vasovagal Response in Spinal Anesthesia during Surgery.
4. Van Lieshout JJ, Wieling W, Karemaker JM, Eckberg DL. The vasovagal response. Clinical science (London, England: 1979). 1991 Nov 1;81(5):575-86. van Lieshout JJ, Wieling W, Karemaker JM, Eckberg DL. The vasovagal response. Clinical science (London, England: 1979). 1991 Nov 1;81(5):575-86.
5. Kinsella SM, Tuckey JP. Perioperative bradycardia and asystole: relationship to vasovagal syncope and the Bezold–Jarisch reflex. British Journal of Anaesthesia. 2001 Jun 1;86(6):859-68.
6. Chowdhury T, Schaller B. Key to Prevention of Bradycardia: Be Relax Postoperatively: A Case Report. Medicine (Baltimore). 2016 May;95(22):e3733. doi: 10.1097/MD.0000000000003733. PMID: 27258501; PMCID: PMC4900709

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