Volume : 3 | Issue : 2 | February 2014 • ISSN No 2277 - 8179

Research Paper

Medical Science

KEYWORDS : Bradycardia, hypotension, spinal anaesthesia

Development of Intra-Operative Severe Bradycardia Under Spinal Anaesthesia: A Case Report

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ABSTRACT

Bradycardia and hypotension are often and benign complication of regional anaesthesia.The causes for development of bradycardia after spinal anaesthesia are not completely understood, yet circulatoryetiology is considered to be primary one. Severe bradycardiaunder spinal anesthesia occurs more frequently in healthy, young ASA I patients while hypotension occurs more frequently in the elderly. So, the knowledge of the risk factors as well as their pathophysiology and immediate and aggressive treatment, during the occurrence of severe bradycardia and hypotension is must in order to prevent fatalities and to achieve best long term outcome.

Introduction:

Bradycardia and hypotension are the known but preventable side -effects of spinal anesthesia. However sudden unexpected bradycardia and cardiac arrest under spinal anesthesia is considered as rare and uncommon manifestation.The causes for the development of bradycardia after spinal anaesthesia are not completely understood.[1]Decreased heart rate may occur during anaesthesia from many causes,including cardiac pathology, pre-operative or anaesthetic drugs and hypoxia. Sudden bradycardia may also be produced through 'paradoxical' cardiovascular reflexes. These are activated especially during periods of reduced venous return to the heart, over-riding the effect of baroreflex suppression which acts to maintain blood pressure in thissituation by increasing heart rate and vasoconstriction.[2] Bradycardia with hypotension occurs in 5% of spinal anaesthetics,[3] but the precise incidence of sudden severe bradycardia is not available. However, the latest review of incidence and potential risk factor of hypotension and bradycardia during spinal anesthesia were reported in 1992 (33% and 13%, respectively).[3,4] Changing anestheticpractice in the last decade have modified these incidences to some extent.

We are reporting a case of a young healthy patient posted for lower limb surgery, who developed sudden severe bradycardia intra-operatively following spinal anaesthesia.

The sudden hemodynamic disturbance in the perioperative period can occur because of various surgical and anesthetic reasons but hemodynamic collapse due to noxious stimulus of periosteum stripping has not been described. We report two cases of severe hypotension and bradycardia during periosteum stripping in orthopedic surgery under subarachnoid block even though the block level was adequate. In our patients, hemodynamic collapse occurred specifically at a moment when surgeons manipulated periosteum and fall in blood pressure and heart rate was sudden in onset. The hemodynamic disturbance did not appear to be related to vagally mediated or due to blockade of sympathetic fibers but appeared to be related to periosteal nociceptors.

Keywords:

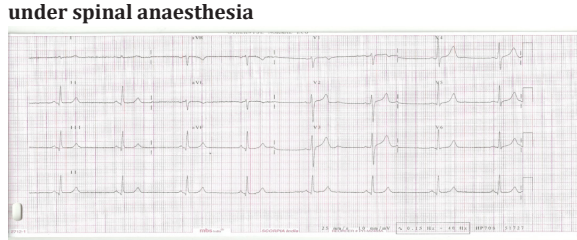
Bradycardia, Hypotension, Nociceptors, Periosteum, Spinal anesthesia

CASE REPORT:

A 38year old physical status ASAI male weighing 67 kg and height 180 cm with varicose veins was scheduled for elective surgery. Nothing was significant in his pre-operative check-up. On arrival in operation theatre, IV line was set with 18 G cannula. Intra-operative haemodynamicmonitoring initiated, including ECG, NIBP,HR and SpO_2at ten minutes interval till the end of surgery. His pre-operative blood pressure was 124/74 mmHg and pulse rate was 70 beats/minutes.ECG tracing and SpO_2 were within normal limits. The case was planned to be done under spinal anaesthesia.

Pre-loading was started with Ringer's lactate 10 ml/Kg, 20 minutes before administering spinal anaesthesia. Patient was kept in left lateral decubitus position and subarachnoid block was given with 26 G BD Quincke spinal needle in between L3-4 interspace under aseptic conditions. A total of 2.5 ml of 0.5% Bupivacaine (heavy) was given to achieve a sensory level of T10. Patient was turned to supine position as per surgeon's requirement following 10 minutes of subarachnoid block and surgery started.Patient's hemodynamic parameters tracing was running normally, suddenly forty minutes following surgery, patient developed bradycardia of 48/ min (**Figure 1…….about here**)

Figure 1: Showing slowing of heart rate intro-operatively under spinal anaesthesia



but his blood pressure was almost within physiological normal limit(108/64 mmHg). Intravenous atropine was administered in an increment of 0.3 mg until desired heart rate restoredand rapid IV fluid, Ringer's lactate was administered simultaneously. Oxygen administered with nasal prongs at 8L/min. Patient had no complaints clinically. Patient respondedwell to intravenous atropine and IV fluid therapy and remained stable throughout the surgery and thereafter.

DISCUSSION:

Hypotension and bradycardia of various severity are frequent and often benign complications of regional anaesthesia. The incidence of intraoperative severe bradycardia requiring therapy has been reported and cardiac arrest has occurred.[5]Severe bradycardia is defined as HR< 50beat/min requiring treatment with atropine 0.5 mg i.v, repeated as necessary. Severe hypotension is defined as a decrease in systolic arterial pressure to less

than 70% of the patient's initial systolic blood pressure before induction of spinal anaesthesia, requiring treatment with vasopressors. The mechanisms of these events complex and usually of as primary circulatory etiology.[6]

Most of these effects are directly or indirectly related to the blockade of sympathetic efferents during spinal anaesthesia. For example,the level of sympathetic blockade during spinal anaesthesia is often two to six levels higher than the sensory level, so a patient with a T_4 sensory block may have completely blocked cardiac accelerator fibres that originate from T1 to T4. Blockade of these fiberscan result in a variety of bradyarrhythmias. [7] But, because of the too low level (L_{3-4})of spinal anaesthesia, this effect was most likelynot relevant in our case.

Mackey et al **[8]** have suggested three working reflexes: (a) The pacemaker stretch reflex: the rate of firing of the pacemaker cells within the myocardium is proportional to the degree of stretch. Decreased venous return results in decreased stretch and a slower heart rate. (b) Low pressure baroreceptors in the right atrium and vena cava when stimulated may cause bradycardia. It is known that baroreceptor activation secondary to increases in BP,decreases sympathetic outflow and increases parasympathetic activity leading to vasodilation and bradycardia and that with a decrease in BP, baroreceptor activity ceases. (c) The Bezold-Jarisch reflex: mechanoreceptors in the left ventricle when stimulated may cause bradycardia. The origin of severe bradycardia in our case could not be explained very well on this basis alone as blood loss in such surgery was insignificant and preloading with 10 ml/Kg seemed to be adequate.

Although reflex cardiovascular depression with bradycardia is usually triggered by reduced venous return, it can be also triggered through affective stimuli as pain or fear initiating the famous vasovagal mechanism**[2]**but sometimes also via various non-cardiac baroreceptors which may become paradoxically active by stimulating the limbic sympatho-inhibitory centers. **[2]**But, no such history suggestive of the development of such events was associated with our patient who was a young apparently healthy, quite and co-operative person.

Heart rate does not change significantly during spinal anesthesia in most patients. However, clinically significant bradycardia occasionally occurs with a reported incidence of 10% to 15% .**[9]** Carpenter et al reported that ASA physical status I young patients have three-fold increased risk of developing moderate bradycardia of < 50beats/min during spinal anaesthesia, while hypotension was more frequent in the elderly. **[3]***Pollard* has recently summarized the risk factors for bradycardia <50 beats/min during spinal anaesthesia in the form of baseline heart rate < 60 beats/min, ASA physical status I, age < 50 years, sensory level block above T6, prolonged P-R interval and the use of β-blocking drugs.**[10]** But, no obvious clinico-pathological correlation was evident for the occurrence of sudden severe bradycardia in this case except there was a history of lateral decubitus position used for spinal needle insertion and turning into supineposition for surgical position as a precipitating factor to influence the orthostatic stress. Anticipating an impending decrease in venous return, placing the patient in head down position and rapidly infusing IV fluids can be helpful,**[11]**when time may not be sufficient. If this is not possible or if it does not rapidly reverse vagal symptoms, the administration of atropine or a vasopressor may be appropriate. Two prospective studies examined the prevalence and risk factors for bradycardia and other intraoperative complications during spinal anaesthesia, in a total of 2700 patients.Bradycardia occurred in 10%, hypotension in 22% and nausea in 10%.**[2]**

The mechanism that triggers severe bradycardia and cardiac arrest under. spinal anesthesia remains controversial and unclear. Over sedation, respiratory arrest, unintentional total spinal, myocardial infarction and local anesthetic toxicity were hypothesized as the causative factors.[12] But, these all conditions could not be implemented in our case. However, sometimes, contribution of intrinsic cardiac mechanisms and autonomic imbalance with the background of parasympathetic predominance may provide more convincing and physiological explanation for the occurrence of abrupt severe bradycardia and cardiac arrest under spinal anesthesia, that might otherwise be more benign, transient, or possibly unnoticed. There exist a number of risk factors (Table1 about here……..)

Table 1: Showing various risk factors for bradycardia and cardiac arrest during spinal anaesthesia

1. Age <50 years
2. Baseline heart rate <60/min
3. ASA physical status I and II
4. Use of beta blockers
5.Sensory level blockade above T6
6. Prolonged PR interval
7. Vagotonia

with variable impact on the occurrence of severe bradycardia and cardiac arrest under spinal anesthesia.

Presence of two or more listed factors may place these patientsat highrisk for bradycardia and cardiac arrest under spinal anesthesia. But, due to inconsistent reporting, riskfactor association with the occurrence of bradycardia and cardiac arrest under spinal anesthesia still remains uncertain and contradictory. **[12]*Pollard*** holds the opinion that the presence of a single risk factor out of these six, does not make it certain that a patient will experience severe bradycardia or cardiac arrest. However, when two or more of these six factors are present, the patient may be considered at high-risk for bradycardia and cardiac arrest during spinal anaesthesia.**[10].** Following reviewing literatures, it could be speculated that in our patient a combination of Bezold-Jarisch reflex with a raised vagal tone and postural effects might have been contributing factors for the development of severe bradycardia and hypotension.

Conclusion:

Although various factors can contribute to bradycardia during spinal anaesthesia but it is clear from literature that in most cases a vagally mediated reflex response to decreased venous return play a key role. So, while selecting spinal anaesthesiafor a patient, requiring intra-operative postural change , maintenance of adequate preload is a key to decreasing the risk of bradycardia and cardiac arresttogether with the knowledge of pathophysiological risk factors, vigilance and skills to avoid fatalities and for patient's survival.

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