CASE REPORT

Bezold Jarisch reflex as a cause of haemodynamic alterations during surgery in prone position

Sanjay Agrawal, MD, PDCC (NeuroAnaesthesia)*,
Vineet Srivastava, MD, PDCC (NeuroAnaesthesia)**

**Associate Professor; **Consultant (Neuroanaesthesia)*
Department of Neuroanaesthesia , Apollo Hospital, Bilaspur

Correspondence: Dr Sanjay Agrawal, Associate Professor, Department of Anesthesiology, Himalayan Institute of Medical Sciences, Swami Ram Nagar, Dehradun (India); Email: drumstix1972@yahoo.co.in

ABSTRACT

Haemodynamic dysfunction and cardiac conduction abnormality in patients undergoing spine surgery in prone position is multifactorial. Here we present a case report of an elderly male patient undergoing multiple level dissectomy under general anaesthesia, who developed bradycardia and hypotension after prolonged surgery and was managed successfully. The case description and review of literature is presented.

Keywords: Prone position; General anaesthesia; Bezold Jarisch reflex; Hypotension; Bradycardia

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INTRODUCTION

Reflex cardiovascular depression with vasodilation and bradycardia has been termed as Bezold Jarisch reflex. This is mediated through a neural mechanism rather than any cardiac dysfunction. Bradycardia and vasodilation are the principal changes triggered either centrally or peripherally[1,2]. Here we present a case of an elderly male patient undergoing dissectomy at multiple levels under general anaesthesia, who developed bradycardia and hypotension after two and a half hours of surgery. Active intervention was required to manage the dysfunction successfully. We present here the case description and review of literature.

CASE REPORT

A 77 years old male patient presented to neurosurgery outpatient with complaints of severe pain in his lower back radiating through the both of his lower limbs. On clinical examination and radiological investigation, he was found to have multiple level disc protrusion and he was planned for L1-5 laminectomy with L3-4, L4-5 and L5-S1 dissectomy.

During the preoperative assessment, the patient gave a history of being a hypertensive, controlled with tablet(tab) telmisartan 40 mg OD and tab amlodipine 5 mg OD. He had no history of palpitations, syncopy or chest pain and had a moderate exercise tolerance. Preoperative ECG was normal. The patient was accepted as ASA grade II patient and was advised tab alprazolam 0.5 mg in night and on morning of surgery along with his usual antihypertensive drugs with a sip of water two hours prior to his being shifted to operating room.

On the day of surgery, in the operating room, intravenous access was established and routine monitoring e.g. NIBP, 5 lead ECG, and pulse oximeter was established. Induction of anaesthesia was done with fentanyl 2u kg and propofol 1mg/kg IV and intubation was facilitated with inj. vecuronium bromide 0.1 mg/kg. Anaesthesia was maintained with isoflurane in 66% nitrous oxide in oxygen, intermittent boluses of vecuronium and fentanyl. Ventilation was maintained to achieve the endtidal carbon dioxide ($EtCO_2$) at 32-35 mm Hg. Invasive blood pressure (IBP) monitoring was established by cannulating the left radial artery. The patient was then turned prone with a soft wedge under his chest and at pubic region, so as to keep the abdomen free from pressure. The patient behaved very well for the next two and a half hours, after which it was noticed that his heart rate decreased from 66 beats/minute to 55 beats/minute and IBP fell from 120/70 mmHg to 96/54 mmHg. There was a spontaneous restoration of the vitals to their previous reading after rapid infusion of crystalloids. However, just after five minutes there was a second episode of bradycardia (42 beats/minute) and hypotension (70/38mm Hg); SpO_2 and $EtCO_2$ (34 mmHg) remained within normal limits. The surgeon was informed about this happening, inhalational agent and N_2O were switched off and the patient was given 100%

oxygen. Fast infusion of ringer lactate and inj. atropine 0.6 mg in a bolus was given. A 6 mg bolus of inj. mephenteramine was also pushed. The surgeon was asked to check for any active bleeding as well as to flood the operative site with normal saline. The haemodynamic were restored to the IBP of 105/66 mmHg and a heart rate of 58-60 beats/minute. The surgery was commenced. A third episode of bradycardia and hypotension occurred after thirty minutes of second episode. This time, it was accompanied with ST segment depression of -1.5 mV. Ionotropic support was initiated with infusion of inj. dopamine 15 μg. ECG trace on the cardiac monitor showed a junctional rhythm with the rate of 45/minute. Dopamine infusion restored the haemodynamics to 140/70 mmHg of IBP and a heart rate of 62/min, but the ST segment depression persisted. Possibility of intraoperative MI was considered and inj. nitroglycerine was started at the rate of 3 μg/kg/min. A cardiology consultation was sought, who on viewing the ECG on monitor, was of the opinion that the changes might be due to junctional bradycardia and may be due to either surgical stimulus or increased vagal tone. The surgeon was asked to continue with the surgery. Haemodynamics were maintained with the infusion of dopamine and low dose nitroglycerine. At the completion of the surgery the patient was shifted to ICU and put on controlled mode of ventilation with the ionotropic support. A 12 lead ECG, bedside echocardiograpy and Trop-T test done in ICU, were all negative for recent MI. As the patient was stable haemodynamically, the vasopressor support was gradually withdrawn, he was weaned off the ventilator over the next 8 hours and extubated. After extubation, the IBP remained stable at 140/90 mmHg and heart rate at 65/minute. He was kept under observation for the next 12hours and then shifted to high dependency unit (HDU) from where he was shifted to the ward to be finally discharged home after full recovery.

DISCUSSION

Intraoperative haemodynamic alterations are multifactorial. Possible causes may be may include position of the patient, use of anaesthetic drugs or preoperative condition of the patient as well as the preoperative use of ACE inhibitors. The use of prone position may result in decreased venous return from pooling of blood in the legs and decreased left ventricular compliance secondary to increased intrathoracic pressure for spine surgery, causing a decrease in both arterial pressure and tissue perfusion[3]. On comparing different prone positions and the use of frames or bolsters for positioning, Sreenivasa et al found that least cardiac function deterioration occurred with the use of Jackson spine table or with use of longitudinal bolsters[4]. This may be due to least changes in preload due to least hampering of venous return.

The causes of intraoperative junctional rhythm include surgical stress response, vagal hypertonicity, hypothermia, endotracheal tube cuff pressure or venous air embolism. Occurrence of bradycardia as a result of atrial stretch receptors (Bezold Jarisch response) also plays an important role in pathogenesis of these arrhythmias and haemodynamic variables. This reflex is common in patients with contracted blood volume[1]. Another theory suggested the etiology of these haemodynamic changes in hypertensives- the "collapse firing theory", to be due to is a decrease of sympathetic outflow and increased parasympathetic activity in hypertensives. With decrease in blood pressure, baroreceptor activity ceases but with progressive hypovolemia there may be a paradoxical recurrence of baroreceptor discharge and bradycardia[5].

Surgical stimulus as a cause of bradycardia is probably due to stimulation of afferent parasympathetic nerve endings causing a reflex celiac (vasovagal) reaction. This parallels the Buer Lockhard reflex seen in cases where anal/perineal stretching cause bradycardia or even cardiac arrest[6].

Drug therapy e.g. inj. atropine are often employed as first line treatment of a slow heart rate during anaesthesia. It is, however, not useful in conditions where bradycardia is accompanied with vasodilation. Hypotension during vasovagal response may persist after relief of bradycardia[7]. Sympathomimetic drugs, by contrast, are more useful in such cases as these will counteract the vasodilation in both arterial and venous circulation[8].

In our patient, the possible cause of the haemodynamic changes may have been the Bezold Jarisch reflex. As the patient was elderly and hypertensive, his preop blood volume may have been contracted. The use of anaesthetic agent and blood loss may have further lead to hypovolemia and activation of atrial stretch receptor. The transient response to atropine as well as sustained response to dopamine infusion, also support this hypothesis. The normal end tidal concentration of carbon dioxide rules out the presence of venous air embolism as a possible cause.

CONCLUSION

In conclusion, we highlight the fact that hypotension and bradycardia in prone position may not always be due to a decrease in cardiac index but the activation of physiological reflexes may also contribute to intraoperative changes and an anesthesiologist must keep these facts in mind whenever such patients are operated in prone position.

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Corrigendum

In Vol 14, No. 1 (June 2010) issue of Anaesthesia, Pain & Intensive Care, the names of the authors of the original article entitled, 'Comparative study of acute normovolaemic haemodilution and acute hypervolaemic haemodilution in major surgical procedures' were erroneously printed as;

R.V.Shidhaye, M.D. D.A., D.S. Divekar, M.D. D.A., Vijaya Lakhkar, M.D. D.A., Vijaya Lakhkar, M.D., Rahul Shidhaye, M.D. M.H.S.

The correct names of the authors were;

R.V.Shidhaye, M.D. D.A., D.S. Divekar, M.D. D.A., Vijaya Lakhkar, M.D. D.A., Sandeep Sinha, M.D., Rahul Shidhaye, M.D. M.H.S.

Anaesthesia, Pain & Intensive Care deeply regrets the mistake.

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