

# PSYCHOGENIC ABRUPT ASYSTOLE DURING SPINAL ANESTHESIA: REPORT OF A CASE

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*Abstract - Psychogenic abrupt asystole during spinal anesthesia is an uncommon occurrence. A case is presented with literature review.*

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**Key Words:** *Psychogenic abrupt asystole, spinal anesthesia*

## INTRODUCTION

Sinus bradycardia and cardiac arrest have been observed during numerous pediatric surgical procedures, including strabismus repair, laryngoscopy, and neurosurgery. There have been reports of adult cases of bradycardia when vecuronium was used as a muscle relaxant. Most of these incidents occurred during laparoscopic abdominal operations(1). Although bradycardia, a fall in blood pressure and hypoxia are commonly encountered during neuraxis blockade (2,3,4), the occurrence of psychogenic abrupt asystole during spinal anesthesia is extremely rare (5, 6). Recently a few cases of abrupt asystole during spinal anesthesia have been reported, and one of them occurred when the patient was awake and psychologically dispaired. In this paper, a case of intraoperative abrupt asystole is being reported in a patient who became overexcited before the occurrence of asystole. The patient was successfully resuscitated without any sequelae.

## CASE REPORT

A 68-yr-old man, weighing 65 kg, with benign prostatic hypertrophy was scheduled for transurethral retropubic resection. The patient's medical condition was significant for

hypertension, which was well controlled by propranolol. All preoperative laboratory values and the chest roentgenogram were within normal limits except for the electrocardiogram which showed left ventricular hypertrophy. Before anesthesia was started, electrocardiograph, automated arterial blood pressure, and pulse oximetry measurements were monitored. The initial blood pressure was 120/80 mm Hg, the heart rate was 75 beats/min with sinus rhythm, and the Saturation via pulse oximetry (SpO<sub>2</sub>) on room air was 95%. After a rapid infusion of crystalloid solution, spinal anesthesia was performed. Two and a half ml of 0.5% bupivacaine in dextrose was injected into the subarachnoid space at the level of L2-3 interspace. Ten minutes later, when sensory level to pinprick was noted to be T<sub>10</sub> bilaterally, the operation was started. Oxygen 3 lit/min was started via a nasal cannula. During the initial 20 minutes of operation the blood pressure remained approximately 100/60 mm Hg, heart rate 90 bpm, and SpO<sub>2</sub> 95%. The patient was comfortable without receiving any sedative medication. Thirty minutes after the initiation of spinal anesthesia, while the patient was relating an emotional memory to the anesthesiologist, he suddenly complained of feeling "unwell". The heart rate fell abruptly to 30 bpm, followed by asystole and loss of consciousness. Resuscitative measures, including immediate ventilation with 100 percent oxygen, administration of 1 mg atropine intravenously, and cardiac massage were started. Within one minute, spontaneous sinus rhythm with a heart rate of 100/min resumed. Blood pressure was 90/70 mm Hg. The patient started to

breath spontaneously. Immediately following the return of stable pulse and blood pressure, the patient's sensory block level to pinprick was checked again and found to be at the level of T<sub>10</sub> bilaterally. The remainder of the anesthetic course and hospital stay were unremarkable. A postoperative ECG was normal.

## DISCUSSION

Cardiac arrest with cemented total hip arthroplasty has been reported in 0.6-10% of patients and is thought to be the result of intraoperative hypotension secondary to methyl methacrylate and cement (7). Traction of abdominal contents has been incriminated to cause sinus bradycardia and asystole mediated primarily via the vagal fibers distributed to the AV nodal tissue (1). A 10-25% incidence of probe-patent foramen ovale in the general population implies that many patients undergoing total hip replacement are at risk for paradoxical emboli and subsequent cardiovascular collapse presumably resulting from paradoxical coronary artery occlusion (8).

Sporadic intraoperative cardiac changes are infrequently seen and the causes are innumerable, ranging from light anesthesia to drug overdosage or interactions, respiratory problems such as hypoxia, and hypercapnia and at times undiagnosed electrolyte abnormalities. During spinal anesthesia, hemodynamic changes such as bradycardia and a fall in blood pressure are not uncommon (2,3,4). The theories regarding these hemodynamic changes involve indirect effects of the technique on the heart. These effects are caused by central sympathetic efferent blockade that may lead to a decrease of venous return to the heart (4,5,6). In the patient presented here the level of analgesia was T<sub>10</sub> and despite the fact that the cardioaccelerator fibers were not blocked, asystole occurred abruptly 30 minutes after spinal anesthesia. It is noteworthy that the blood pressure and pulse rate of the patient remained

stable, immediately preceding the episode.

According to closed claims analysis by Caplan and coworkers(3), cardiac arrest during spinal anesthesia usually resulted from hypoxia and hypercapnia associated with unappreciated respiratory insufficiency (hypoxia and lack of vigilance). However, the case presented here did not receive any sedative drugs and his SpO<sub>2</sub> was greater than 92% at the time of asystole. Therefore, his asystole could not be due to respiratory insufficiency.

According to Mackey and coworkers (5) the cause of abrupt onset of asystole in spinal anesthesia in their patients, whose level of analgesia was T<sub>4</sub>, was probably due to paradoxical Bezold-Jarisch reflex, in which a rapid decrease in ventricular volume could actually stimulate and increase the activity of the mechanoreceptors located in the ventricle presumably due to vigorous ventricular contractions around an almost empty chamber. In our case, the level of analgesia was T<sub>10</sub> and the patient was normovolemic, therefore paradoxical stimulation of the ventricular receptor due to the empty heart initiating asystole could be safely ruled out. There are some reports in the literature of vasovagal syncope and sudden death which are produced by psychological stress (9). Fredrichs and coworkers (6) presented a case of abrupt asystole in an awake vagotonic patient who received a high level of epidural anesthesia. Asystole occurred immediately after the patient was burdened with an emotional stress. They believe that emotionally produced vasovagal response was exaggerated in the patient whose vagal response was already exaggerated by a high level of epidural anesthesia.

Vasovagal response can be responsible for the asystole in our case, because, the patient was fully awake and just preceding the asystole, he was expressing one of his delightful memories with strong emotions. Furthermore, the patient responded to cardiac resuscitation and institution of atropine 1 mg intravenously. Therefore, centrally evoked vasovagal reflexes due to

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over-excitement coupled with a beta-blocked heart due to propranolol consumption can justify the psychogenic nature of asystole in this particular case. This case underscores the importance of close vigilance during the entire period of anesthesia in a patient who is under spinal anesthesia, especially if he is receiving beta blocker therapy. Moreover, benzodiazepines could be of value in patients who are under neuraxis blockade to tame their strong emotions.

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