# Cardiac arrest during transurethral resection of the prostate: the overzealous restriction of intravenous fluids – a possible cause?

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## Abstract
Cardiac arrest during spinal anaesthesia has been widely reported in literature, with several mechanisms being described. There have so far, however, been no reports of cardiac arrest after spinal anaesthesia during transurethral resection in prostate surgery. We report on a case of near-cardiac arrest during transurethral resection in prostate surgery, and possible mechanisms and strategies to prevent the same.

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## Introduction

Sudden cardiac arrest during spinal anaesthesia is a rare but catastrophic event. The reported incidence in literature is about 6.4 ± 1.2 per 10 000 spinal anaesthetics.[1] Various possible causes, which we review in the discussion, have been suggested. We also report on a case of sudden near-cardiac arrest during the transurethral resection of the prostrate (TURP) following spinal anaesthesia in a patient with no prior comorbidity. We cannot attribute any definite cause for this complication, except for the possibility of the overzealous restriction of intravenous fluids in anticipation of preventing the occurrence of TURP syndrome.

### Case report

A 60-year-old male patient with no comorbidity was scheduled for the transurethral resection of his prostrate. The surgery was the fifth case on the operative list and was to take place in the afternoon. On the day prior to surgery, the patient was evaluated by the resident anaesthesiologist and premedicated with 0.25 mg of oral alprazolam the previous evening. He was also asked to be nil per mouth from midnight for solid foods, and 06:00 on the day of surgery for clear fluids.

Surgery was planned under spinal anaesthesia. The patient was moved to the operating room at 12:30. Baseline monitoring included 5-lead ECG monitoring, non-invasive blood pressure (BP) monitoring and pulse oximetry. Initial vital parameters were normal, with a regular heart rate of 75 beats per minute, BP 130/70 mmHg and arterial saturation with the pulse oximeter ($SpO_2$) 100%. An 18G intravenous (IV) cannula was secured in the left upper limb and connected to a Ringer's lactate infusion. Preloading was not done, as is routine practice at our institution for TURP.

As planned, spinal anaesthesia was administered with a 25G Quincke Babcock spinal needle and 2 ml of 0.5% heavy bupivacaine administered at the L3-L4 interspace (Sensorcaine®, Astra Zeneca), with the patient in the right lateral position. Needle placement was atraumatic and successful with the first attempt. A sensory block reaching T10 was achieved within eight minutes and the patient was positioned in lithotomy for the surgery.

Twenty-five minutes into the procedure, the patient complained of dizziness and general weakness. The non-invasive BP was 90/40 mmHg and the heart rate 50 per minute. Only 150 ml of Ringer's Lactate had been infused up to this point. The patient was reassured after reconfirming the sensory block level at T10. Vasopressors had not been used at that point and the lower BP was accepted with the intention of reducing intraoperative bleeding.

Within the next minute or two, however, the heart rate suddenly dropped to near zero. Surgery was immediately stopped. The patient had become increasingly drowsy but had not lost consciousness. Administration of 1.2 mg of IV atropine did not have any response. An IV adrenaline bolus of 200 µg immediately restored the heart rate and the next BP recording was 150/100 mmHg.

Surgery was uncomplicated and blood loss was around 100 ml to 150 ml in total when the bradycardia developed. BP dropped again after about five minutes when the effect of the adrenaline bolus weaned. Central venous access via the right subclavian vein was secured with a 7Fr triple lumen catheter and the measured venous pressure was -2 mmHg. The patient required a bolus of one litre of Ringer's Lactate to raise the central venous pressure to 4 mmHg with a strong pulse volume and a stable BP thereafter.

Surgery was completed in the next ten minutes and the patient was transferred to the recovery room where he was monitored for the next three hours. He did not require any additional vasopressors or fluid boluses. Postoperative haemoglobin, random blood sugar and electrolytes were normal.

**Discussion**

Cardiac arrests during spinal anaesthesia are described as "very rare" and "unexpected" but they are actually relatively common.[2–4] Auroy et al[1] reported that all but one of the 26 cardiac arrests that have occurred during spinal anaesthesia were related to anaesthetic causes. Advanced age and a high ASA physical status could have contributed to these arrests but these factors were conspicuously absent in our case.

Half of the patients who experienced cardiac arrest in the operating room during spinal anaesthesia were young adults with no comorbidity.[5] The fact that many of these arrests occurred in healthy young adults during minor surgery raises the possibility that many were avoidable. Cardiac arrests that occurred after spinal anaesthesia were not closely linked with sedation or the known effects of spinal anaesthesia on respiratory drive.[6]

Baron et al[7] found that cardiac vagal tone is enhanced primarily through reduced venous return. The effect of spinal anaesthesia on venous return can be profound: reductions in the right atrial pressure of 36% after low spinal levels (below T4), and of 53% after higher levels of blockade, have been reported.[8] With IV fluid losses or restriction in IV fluid volume, as in our case, these effects can be even more dramatic. Such decreases in preload may initiate reflexes that cause severe bradycardia. Three such reflexes have been suggested.[9] The first reflex involves the pacemaker stretch. The rate of the firing of these cells within the myocardium is proportional to the degree of stretch. Decreased venous return results in decreased stretch and a slower heart rate. The second reflex may be attributable to the firing of low-pressure baroreceptors in the right atrium and vena cava. The third reflex is a paradoxical Bezold-Jarisch reflex, in which mechanoreceptors in the left ventricle are stimulated and cause bradycardia.[10]

Jacobsen et al[11] studied the effect of epidural anaesthesia on the left ventricular diameter, with echocardiography, in eight unpremedicated young volunteers. Two developed bradycardia and hypotension after 25 minutes with anaesthetic levels at T8 and T9. This was associated with a reduction of up to 22% in the left ventricular diameter. In both cases, the changes were reversed by the head-down position and the rapid infusion of IV fluids.

Vagal reflexes decreasing in preload may not be restricted to just bradycardia. Studies of the haemodynamic effects of graded hypovolaemia have demonstrated progressive vagal symptoms, including sweating, nausea and syncope.[12] In a study by Bonica et al,[13] two healthy subjects experienced vagal arrests after they had 10 ml/kg of blood withdrawn to simulate acute blood loss with sensory epidural block levels of T4 to T6. These studies demonstrated that decreases in preload can precipitate not only classic vagal symptoms but also full cardiac arrest.

When spinal anaesthesia is selected for a patient, maintaining adequate preload is key to decreasing the risk of bradycardia and cardiac arrest during the case.[10]

When bradycardia is profound or full cardiac arrest occurs after spinal anaesthesia, the early administration of epinephrine can be critical.[10] Currently, epinephrine is administered during only 25% to 40% of cardiac arrests after spinal anaesthesia, up to 25% of these arrests being fatal.[1] The earlier and more consistent use of epinephrine has been recommended.[14] Even though Mark et al[15] suggested IV vasopressor agents rather than IV fluids for hypotension during TURP surgeries, we feel that, before vasopressors are used to correct hypotension, consideration should be given to assessing IV volume deficits and ongoing blood losses and to treating these accordingly before vasopressors are used. To avoid excessive

fluid absorption, procedural guidelines such as limiting resection time to less than one hour and suspending the irrigating fluid bag no more than 30 cm above the operating table at the beginning, and 15 cm in the final stages, of resection should also be considered[16] rather than compromising on IV volume maintenance and correction.

Conflict of interest: None

## References


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