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Forced cough for witnessed extreme bradycardia in hip arthroplasty: a maneuver in extremis

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A 35-year-old man (weight, 80 kg; height, 165 cm) with chronic persistent left hip pain, without any known comorbidities, was diagnosed with avascular necrosis of the femoral head of the left hip joint and was scheduled for uncemented left hip total arthroplasty. His preoperative vital signs were unremarkable: blood pressure, heart rate, and oxygen saturation on room air were 110/74 mmHg, 76 /min, and 99%, respectively. The patient had a normal airway with no major risk factors. Spinal anesthesia with standard anesthetic monitoring was planned. In order to administer anesthesia, pre-procedure ultrasound-guided marking of the desired interspace was performed under aseptic conditions. Using a 25 gauge pencil-point standard spinal needle, 2.7 ml of 0.5% bupivacaine heavy and 300 μg of preservative-free morphine were administered into the intrathecal space at the L4–5 interspace using a paraspinal approach. The level of spinal anesthetic block was checked after 5 minutes using the loss of sensation to touch, and it was determined to be at the T7 dermatomal level. The patient was then positioned in the right lateral decubitus position for surgery. His vital signs continued to be stable until approximately 90 minutes after administration of spinal anesthesia, during surgical reaming of the femur, when he developed a sudden extreme bradycardia/asystole. This was seen on an electrocardiogram (ECG) monitor within a few seconds. The total blood loss at this stage of the surgery was approximately 200 ml. A brief check to rule out disconnection of leads was performed while the patient remained partly responsive and a quick pulse check revealed an indeterminate pulse. Recognizing the need for immediate intervention, we instructed the patient to cough forcefully and continuously until he could be turned onto the supine position for chest compressions or intravenous (IV) medication to be administered as per standard cardiopulmonary resuscitation (CPR) protocols. Remarkably, the patient complied with our instruction to cough repeatedly (every 3 seconds). The monitor then showed a return of the ECG trace, with a display of a slow heart rate of about 30 beats/min (Table 1); however, ECG waveforms were not printed. He remained partly responsive with symptoms of retching. Considering the unusual situation of a slow heart rate with continued responsiveness, oxygen via facemask, IV atropine 0.6 mg, and IV epinephrine 100 μg were administered. This resulted in the return of a palpable peripheral pulse with stable recordable blood pressures. Without the need for any further resuscitation, the surgery was completed. After this event, the level of spinal anesthesia was judged to be at the T9 dermatomal level.

A proposed mechanism for bradyarrhythmias under spinal anesthesia involves the Bezold-Jarisch reflex or the 'reverse' Bainbridge reflex activated by preload reduction [1]. In the presence of adequate preload, only a 10% reduction in heart rate compared to baseline levels may occur. This emphasizes reduced preload as a more important cause of bradycardia than sympathetic denervation ('high spinal') in spinal anesthesia [1]. The blood volume lost initially was not considered significant

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Table 1. Tabular Trend of Vital Signs Captured from the Patient's Monitor

Vital signs	Time (h:min)/Event*							
	10:24	10:25	10:26	10:27 (1) (2)	10:28 (2)	10:29 (3)	10:30 (4)	10:31
Heart rate (/min)	67	63	61	†	33	31	105	125
NIBP-systolic (mmHg)	88	-	-	92	-	-	184	-
NIBP-diastolic (mmHg)	68	-	-	55	-	-	137	-
NIBP-mean (mmHg)	75	-	-	63	-	-	144	-
SpO_2 Saturation (%)	100	100	100	89	‡	97	99	100
SpO_2 pulse rate (/min)	67	63	58	192†	‡	‡	105	126
Respiratory rate (/min)	16	7	33	108†	12†	23	29	27

NIBP: non-invasive blood pressure, SpO_2: peripheral oxygen saturation. -: no values recorded. *Events indicate possible time of transition to extreme bradycardia (1), forced cough (2), administration of IV medication (3) and return of recordable vital signs (4). †Near zero values, ‡Indeterminate values recorded.

enough, but a sudden loss of blood volume could have occurred during reaming of the femur. It is not known if the manual reaming could have forced particulate emboli into circulation at the time. Postoperative echocardiography did not reveal abnormalities of right ventricular contractility or elevation of pulmonary arterial pressures.

Standard CPR protocols define cardiac arrest as unresponsiveness accompanied by abnormal breathing in the patient. In this case, the patient was partly responsive and he responded to instructions to cough forcefully every 3 seconds [2]. This maneuver was attempted due to reports of its utility in briefly maintaining perfusion (for up to 90 seconds, until standard CPR could be initiated), for witnessed arrhythmias in the cardiac catheterization lab, including ventricular fibrillation [2–4]. The mechanism of forced cough-induced maintenance of coronary and cerebral perfusion could involve the changes in intrathoracic pressure that drive intravascular volume [3]. A recent double-body plethysmography study on hemodynamic effects of forced cough in healthy participants demonstrated a significant thoracic blood shift via thoracic and abdominal pump mechanisms [5]. Forced coughing has even been reported in some cases to generate systemic blood pressures higher than conventional chest compressions [2–4]. The maneuver may have helped in briefly maintaining coronary and cerebral perfusion pressures in this patient, as indicated by continued responsiveness and the return of slow heart rhythm on the monitor, i.e., from extreme bradycardia to about 30 beats/min (Table 1) [4].

Atropine and epinephrine were administered intravenously in accordance with the algorithm for severe symptomatic bradycardia. Response to IV epinephrine was observed at a lesser dose (i.e., 100 μg) than that described for cardiac arrest.

The forced cough maneuver may be briefly attempted in extremis, such as in witnessed cases of extreme bradycardia, until standard CPR algorithms can be instituted.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

Author Contributions

John George Karippacheril (Conceptualization; Data curation; Formal analysis; Writing – original draft; Writing – review & editing)
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