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Orthostatic intolerance during early mobilization after fast-track hip arthroplasty

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Editor's key points

- Haemodynamic responses during orthostatic intolerance (OI) after hip surgery were studied.
- Up to 20% patients had OI 24 h after surgery.
- Patients with intolerance showed impaired haemodynamic responses to changes in position.
- The study shows how common orthostatic hypotension after hip surgery is, and it attempts to evaluate the underlying mechanisms.

Background. Early postoperative mobilization is a cornerstone in fast-track total hip arthroplasty (THA), but postoperative orthostatic intolerance (OI) may delay early recovery or lead to fainting, falls, and prosthesis dislocation or fracture. However, the prevalence and pathophysiology of OI has not been established after THA. This study evaluated the cardiovascular response and tissue oxygenation to mobilization before and after surgery in relation to OI in fast-track THA patients.

Methods. OI and the cardiovascular response to standing were evaluated with a standardized mobilization protocol, before, 6, and 24 h after surgery in 26 patients undergoing THA with spinal anaesthesia and an opioid-sparing analgesic regime. Haemoglobin, fluid balance, and opioid use were recorded. Systolic (SAP) and diastolic (DAP) arterial pressure, heart rate (HR), stroke volume (SV), cardiac output (CO), and systemic vascular resistance were measured non-invasively (Nexfin®) and cerebral (Sc_{O_2}) and muscle tissue oxygenation by non-infrared spectroscopy.

Results. No patients demonstrated OI before surgery, whereas 11 (42%) and five (19%) patients experienced OI 6 and 24 h after surgery, respectively. OI was associated with decreased orthostatic responses in SAP, DAP, SV, CO, and Sc_{O_2} compared with orthostatic tolerant patients ($P<0.05$). There was no difference in postoperative haemoglobin concentrations or opioid use between orthostatic intolerant and tolerant patients.

Conclusions. Early postoperative OI is common in patients undergoing THA and is associated with an impaired cardiovascular orthostatic response and decreased cerebral oxygenation.

Keywords: arthroplasty, replacement, hip; orthostatic intolerance; postoperative period

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Early postoperative mobilization is essential for rapid functional recovery after surgery and is considered a cornerstone in the so-called fast-track multimodal perioperative approach which includes early ambulation and oral nutrition, multimodal opioid-sparing analgesia, and optimized fluid therapy. This strategy has improved patients outcome after surgery and reduced length of hospital stay.[1] However, postoperative orthostatic intolerance (OI) may delay ambulation and is characterized by symptoms of dizziness, nausea, blurred vision, or syncope due to failed orthostatic cardiovascular regulation, a decrease in arterial pressure, and cerebral hypoperfusion during standing.[2] Transient inability to ambulate has been observed after ambulatory surgery and remains a cause of prolonged hospital stay.[3–5] Additionally, a high (50%) incidence of OI during early postoperative mobilization has been reported in patients undergoing radical prostatectomy,[6] whereas intact orthostatic cardiovascular regulation was observed 30 min after mastectomy in a fast-track setting.[7]

Patients undergoing fast-track total hip arthroplasty (THA) are often mobilized on the day of surgery and are required to participate in physiotherapy sessions in the early postoperative period.[8] Thus, OI may delay recovery and cause symptoms of cerebral hypoperfusion and syncope, leading to potential serious complications such as falls with a risk of prosthesis dislocation and fracture in this often elderly group of patients.[9] However, the incidence of OI during early mobilization in orthopaedic surgery is unknown and the predisposing factors and cardiovascular mechanisms involved in failed orthostasis are not fully understood.[6]

The primary aim of this study was to determine the incidence of OI during early postoperative mobilization in

fast-track THA and secondary aims were to describe the changes in cerebral oxygenation and cardiovascular variables involved in the pathophysiology of OI. In addition, we examined whether bleeding, postoperative anaemia, or opioid use was associated with OI.

Methods

Twenty-six patients (17 females) undergoing unilateral primary THA were included in the period of March–July 2010. Exclusion criteria were history of OI, diabetes mellitus, atrial fibrillation, ASA score ≥III, or a history of alcohol abuse (>40 units week^{-1}). All patients gave informed consent and the trial was approved by the local ethics committee (H-D-2009-067) and registered by the Danish data protections agency and on ClinicalTrials.gov under the US national library of medicine (NCT01089946).

Anaesthesia, pain management, and surgery

Patients were anaesthetized with spinal anaesthesia (12.5–15 mg bupivacaine) and received propofol sedation at the discretion of the attending anaesthesiologist.

The preoperative fasting period was 6 and 2 h before surgery for solid food and clear fluids, respectively.[10] To cover basal and surgical fluid losses, a fixed volume fluid regimen of 12 ml kg^{-1} isotonic saline the first hour of surgery was administered, followed by 6 ml kg^{-1} h^{-1} until end of surgery.[11] Blood loss was replaced 1:1 with 6% hydroxyl ethyl starch (Voluven; 130/0.4 Fresenius Kabi AB, Uppsala, Sweden). For the first 6 h after surgery, patients received saline 2 ml kg^{-1} h^{-1} with no restrictions on oral fluid intake. Criteria for blood transfusion were haemoglobin (Hb) <7.5 g dl^{-1} or Hb <10.0 g dl^{-1} in patients with severe ischaemic heart disease according to guidelines published by the Danish National Board of Health.[12]

Perioperative pain management was standardized as follows: acetaminophen 2 g and gabapentin 600 mg before operation, continuing with gabapentin 900 mg and acetaminophen 4 g daily for the duration of hospital stay. During the first 24 h after surgery, high volume local infiltration analgesia with ropivacaine 2% was administered intraoperatively and 8 and 24 h after surgery using an 18 G epidural catheter placed in the incision by the surgeon.[13 14] Pain scores were graded on a verbal rating scale (0–10) and if they exceeded 3 at rest or 5 during movement, patients received supplemental oxycodone.

Orthostatic challenge

A standardized mobilization procedure was performed ~1 h before surgery and was repeated 6 and 24 h after the operation, defined from the time of wound closure. Mobilization included supine rest (5 min), followed by 30° passive leg raise (3 min), supine rest (5 min), sitting on the bed with the feet on the floor (3 min), followed by standing while the patient was verbally encouraged to stand on the toes and shift body weight from one leg to the other in order to activate the muscle pump and attenuate venous pooling in the legs (3 min).[15] The mobilization procedure ended with recovery at supine rest (5 min).[6] The procedure was terminated if the patients reported symptoms of OI (dizziness, nausea, blurred vision) or upon a decrease in systolic arterial pressure (SAP) >30 mm Hg. During the test, the muscle and cerebral (frontal lobe cortex) oxygenation were assessed with intervals of 10 s by near-infrared spectroscopy (NIRS, Somanetics, INVOS®, cerebral oximeter, Troy, OH, USA) with optodes placed on the biceps brachii muscle and on the forehead. NIRS has been validated as a measure of tissue oxygenation in both surgical and non-surgical settings.[16–18] Continuous arterial pressures were measured by a finger cuff applied on the middle part of the third finger. From the arterial pressure wave, an aortic flow waveform is computed by simulating a non-linear, time-varying model of the aortic input impedance (Nexfin®, BMeye, Amsterdam, The Netherlands) and integrating the computed aortic flow waveform per beat provides stroke volume (SV). Cardiac output (CO) was calculated as SV times heart rate (HR) and total peripheral resistance (TPR) from the ratio of mean arterial pressure to CO.[19] During each postoperative mobilization test, fluid status and Hb were recorded and pain was graded for each body position. Before the 6 h test, remaining motor blockade was ruled out using a modified Bromage scale.[20]

Orthostatic classification

During the mobilization challenge, patients were classified as having OI if they experienced signs of cerebral hypoperfusion such as dizziness, nausea, blurred vision, feeling of heat or syncope, or a decrease in SAP >30 mm Hg. Regardless of symptoms, patients with a >20 mm Hg decrease in SAP or a >10 mm Hg decrease in diastolic arterial pressure (DAP) upon standing were classified as having orthostatic hypotension.[21]

Data analyses

The finger arterial pressure curve and the derived cardiovascular values were analysed using the Nexfin@PC 1.0 software package (BMeye, Amsterdam, The Netherlands). Each curve was visually inspected for artifacts, and such data were excluded. For both Nexfin and NIRS variables, estimates representing the supine rest periods were averaged over 5 min, while estimates representing the mobilization periods of sitting and standing were averaged over the last 10 s before termination of each posture, both for patients completing the mobilization procedure and patients terminating the mobilization procedure prematurely due to OI.

Normally distributed data are presented as mean (SD), while data not normally distributed are presented as median (inter-quartile range, IQR). A mixed-model analysis of variance for repeated measures was used for comparison of cardiovascular and oxygenation variables between and within each test session and between orthostatic tolerant (OT) and intolerant patients. CO and TPR were included in the model as covariates in order to explain differences in