J Anesth (2013) 27:521–527
DOI 10.1007/s00540-013-1568-x

ORIGINAL ARTICLE

Vigilance of hemodynamic changes immediately after transferring patients is crucial

Zen'ichiro Wajima · Toshiya Shiga · Kazuyuki Imanaga · Tetsuo Inoue

Received: 25 April 2012 / Accepted: 25 January 2013 / Published online: 1 March 2013
© Japanese Society of Anesthesiologists 2013

Abstract
Purpose A decrease in blood pressure is sometimes observed when a postsurgical patient is transferred to another bed after recovering from anesthesia. However, the mechanism behind this hypotension has not been completely elucidated. The purpose of this study was to investigate and compare changes in hemodynamic properties for possible causes of hypotension before and after transfer to another bed of postsurgical patients receiving general anesthesia, combined epidural and general anesthesia, or combined spinal and general anesthesia.

Methods We studied 69 patients undergoing elective surgery who were randomized to receive anesthesia by one of the three methods. After surgery, the tracheal tube was removed, and each patient was transferred to another bed. Hemodynamic data recorded immediately before and after transfer of the patient to another bed were compared.

Results After transfer of patients receiving general anesthesia or combined epidural and general anesthesia, systolic arterial pressure (SAP), diastolic arterial pressure (DAP), and cardiac output (CO) decreased; heart rate (HR) and systemic vascular resistance (SVR) did not change. However, after transfer of patients receiving combined spinal and general anesthesia, SAP, DAP, HR, and CO decreased, but SVR did not change.

Conclusion The decrease in blood pressure observed after transfer of a postsurgical patient to another bed after general, combined epidural and general, and combined spinal and general anesthesia was associated with a decrease in CO and no change in SVR, but HR decreased after combined spinal and general anesthesia, whereas it was unchanged after general and combined epidural and general anesthesia. The decrease in blood pressure is assumed to be caused by a decrease in venous return, and several reflexes might participate in this decrease of blood pressure, especially after combined spinal and general anesthesia.

Keywords General anesthesia · Epidural anesthesia · Spinal anesthesia · Postoperative complications · Hypotension

Part of this work was presented at the American Society of Anesthesiologists 2010 Annual Meeting (San Diego, CA, USA).

Z. Wajima (✉)
Department of Anesthesiology, Shioya Hospital, International University of Health and Welfare, Tomita 77, Yaita-shi, Tochigi 329-2145, Japan
e-mail: HFB01245@nifty.com

Z. Wajima
Department of Anesthesiology, International University of Health and Welfare Hospital, Tochigi 329-2763, Japan

T. Shiga
Department of Anesthesiology, Kaken Hospital, Chiba 272-0827, Japan

K. Imanaga
Division of Cardiovascular Anesthesia, Department of Anesthesia, New Tokyo Hospital, Chiba 271-0077, Japan

Present Address:
K. Imanaga
Department of Anesthesia, Shonan Kamakura General Hospital, Kanagawa 247-8533, Japan

T. Inoue
Department of Anesthesia, Chiba-Hokusoh Hospital, Nippon Medical School, Chiba 270-1694, Japan

Introduction

A decrease in blood pressure is sometimes observed when a postsurgical patient is transferred to another bed after

recovering from anesthesia [1, 2]. As far as we are aware, however, the mechanism behind this hypotension has not been completely elucidated. We hypothesized that such properties as cardiac output (CO) and systemic vascular resistance (SVR) might provide clues to the mechanism behind this hypotension occurring during post-anesthesia bed transfer.

The purpose of this study was to investigate and compare changes in CO, SVR, and other hemodynamic properties for possible causes of hypotension during bed transfer, and their dependence on the type of anesthesia administered: general anesthesia, combined epidural and general anesthesia, and combined spinal and general anesthesia.

Patients and methods

Subjects

After approval of this prospective study by the local institutional review board for human studies, written informed consent was obtained from all patients. The patients comprised 69 patients aged 30–78 years who were scheduled for elective lower abdominal or lower extremity surgery. The patients were classified as ASA physical status class 1 or 2. All patients were randomized by use of computer-generated numbers to undergo one of three methods of anesthesia: general anesthesia, combined epidural and general anesthesia, or combined spinal and general anesthesia. Exclusion criteria included known cardiac disease, pulmonary disease, diseases affecting intravascular fluid volume or balance (e.g., anemia, gastrointestinal obstructive or inflammatory diseases), diabetes mellitus, and diseases affecting the autonomic nervous system. No patients were premedicated. Our target stroke volume variation (SVV) values during surgery were 10–15 % in this study for all anesthesia groups.

General anesthesia group

Induction of anesthesia was performed with propofol (initial effect-site concentration 4 μg/ml) and a total of 1 μg/kg of intravenous remifentanil, with intravenous rocuronium 0.6 mg/kg administered to facilitate tracheal intubation. After tracheal intubation, each patient's lungs were mechanically ventilated by means of a semi-closed circle system at a fresh gas flow of 6 l/min (O_2, 2 l/min and air, 4 l/min). Controlled ventilation was set at 10 breaths/min, with a tidal volume of 8 ml/kg and inspiratory:expiratory ratio of 1:2. Later, intermittent administration of fentanyl, continuous infusion of remifentanil, and the effect-site concentration of propofol (administered by a plasma target-controlled infusion method) were adjusted to achieve $40 < BIS < 60$ and stable circulatory variables (systolic arterial blood pressure (SAP) and continuous heart rate (HR) observed on the monitor both maintained within ±20 % of perioperative values), with intermittent administration of rocuronium if needed.

At the beginning of wound closure, the remifentanil and propofol infusions were discontinued. If detected by the anesthesiologist, residual paralysis was antagonized with intravenous neostigmine 2.0 mg and atropine 1.0 mg, and extubation of the trachea was then performed.

Combined epidural and general anesthesia group

An epidural catheter was placed in one intervertebral space ranging from Th8–9 to L3–4, at a distance of 4 cm inside the space cephaladly, before induction of general anesthesia. The epidural space was identified by the loss-of-resistance technique using physiological saline [3, 4]. Anesthesia consisted of 1 % ropivacaine epidural anesthesia, and the level of analgesia was determined by a pinprick 15 min after administration of the epidural ropivacaine. If the SAP decreased to <90 mmHg or if mean arterial pressure decreased >20 % from baseline, ephedrine 4–8 mg was given intravenously. If the HR decreased to <45 bpm, intravenous atropine 0.3–0.5 mg was administered during anesthesia.

After establishing a level of analgesia to at least that of skin incision level, induction of general anesthesia and anesthesia maintenance were performed as described for the general anesthesia group, with the exception that epidural 1 % ropivacaine and intravenous fentanyl were given intermittently with intermittent administration of rocuronium if needed. Only remifentanil, if needed, was administered as supplemental analgesic during surgery.

The timing of discontinuation of the remifentanil and propofol infusion, administration of intravenous neostigmine and atropine, and extubation of the trachea was similar to that for the general anesthesia group.

Combined spinal and general anesthesia group

Spinal anesthesia was performed through the L3–4 or L4–5 interspace with the patient in the lateral decubitus position. After dural puncture with a 25G Quincke needle, a hyperbaric solution of 0.5 % bupivacaine was injected intrathecally. Bupivacaine dose was determined on the basis of height (height <155 cm, dose 16 mg; 155–170 cm, dose 17 mg; 170–185 cm, dose 18 mg).

The level of analgesia was evaluated by a pinprick 15 min after intrathecal administration of bupivacaine. If SAP decreased to <90 mmHg or if mean arterial pressure decreased >20 % from baseline, ephedrine 4–8 mg was

given intravenously. If HR decreased to <45 bpm, intravenous atropine 0.3–0.5 mg was administered during anesthesia.

After establishing a level of analgesia to at least that of skin incision level, induction of general anesthesia and anesthesia maintenance were performed as described for the general anesthesia group, with the exception that only remifentanil as supplementary analgesic was administered with intermittent administration of rocuronium during surgery if they were required. Intravenous fentanyl was not given.

The timing of discontinuation of the remifentanil and propofol infusion, administration of intravenous neostigmine and atropine, and extubation of the trachea was similar to that of the general anesthesia group.

Measurements

A catheter was inserted in the left or right radial artery after tracheal intubation. A dedicated sensor (FloTrac™; Edwards Lifesciences, Irvine, CA, USA) was connected to the radial arterial line on one side and to the Vigileo™ system (Edwards Lifesciences; software version 01.14) on the other, and the sensor was zeroed to atmospheric pressure [5]. Throughout the perioperative period, SAP, diastolic arterial pressure (DAP), and HR were continuously monitored with a standard patient monitor (S/5 Anesthesia Monitor; GE Healthcare, Finland), and CO, SVR, and SVV were continuously monitored with the FloTrac/Vigileo™ system.

When the patient awoke after surgery, the tracheal tube was removed, and the FloTrac™ sensor was fixed on the patient's shoulder. If no major problem was experienced, the patient was transferred to another bed. The above-mentioned hemodynamic data recorded immediately before transfer (baseline) and immediately after transfer were compared. Observers unaware of the method of anesthesia judged the hemodynamic data.

Immediately after transfer of the patient, artifacts were induced on the arterial pressure contour and ECG waveform. Therefore, SAP, DAP, and HR were measured 5 s after the artifacts disappeared, and CO and SVR were measured 20 s after that, because the S/5 Anesthesia Monitor performs its calculations on the most recent 5 s of data, and the Vigileo™ samples the pressure waveform at 100 Hz over a 20-s period, capturing 2,000 data points for analysis and calculations, which are provided at the end of every 20-s period [5–8].

Statistical analysis

All data are presented as mean ± SD. The sample size was estimated from preliminary data obtained from 10 patients and an assumption that a 0.2 l/min reduction in CO between measurements made at baseline and after transfer would be clinically relevant. The power analysis suggested that a minimum of 20 patients would be needed for beta = 0.1 and alpha = 0.05. To compensate for potential dropouts, we enrolled 23 patients in each group. This analysis was performed using GraphPad StatMate 2.00 (GraphPad Software, La Jolla, CA, USA).

Patient characteristics were analyzed statistically by one-way analysis of variance (ANOVA) or use of Fisher's exact probability test. Paired *t* tests were used to compare differences in SAP, DAP, HR, CO, and SVR between measurements at baseline and after transfer. Unpaired *t* tests were used to compare differences in the end of upward analgesia level between the combined epidural and general anesthesia group and the combined spinal and general anesthesia group. A P value of <0.05 was considered statistically significant. These analyses were performed with GraphPad Prism 5.02 (GraphPad Software).

Results

No patients were taking β-blocker medications chronically. The values of SVV immediately before beginning spontaneous breathing were similar in the three groups (Table 1). No patients suffered massive bleeding during surgery, and neither severe hypotension nor bradycardia were observed for any patient after transfer. All patients received atropine and neostigmine.

In the combined epidural and general anesthesia group, the mean interspace level for catheter insertion was Th12–L1 (SD was 2 vertebral levels and range of insertion was Th8–9 to L3–4). The upper level of analgesia (mean level of right and left sides) reached Th5 ± 2, and the lower level reached the sacral area after surgery. In the combined spinal and general anesthesia group, the upper level of analgesia (mean level of right and left sides) reached Th7 ± 3, and the lower level again reached the sacral area after surgery. The upper level of analgesia reached in the combined epidural and general anesthesia group was significantly higher than that reached in the combined spinal and general anesthesia group after surgery ($P = 0.0025$).

After patient transfer in both the general anesthesia group (Fig. 1) and the combined epidural and general anesthesia group (Fig. 2), SAP and DAP decreased and HR did not change, and CO decreased but SVR did not change. In the combined spinal and general anesthesia group, SAP, DAP, HR and CO decreased after patient transfer, but SVR did not change (Fig. 3). The decrease in blood pressure recovered after a short period (data not shown). Because SVV values measured immediately before spontaneous breathing were similar in all three groups and were normal

Table 1 Characteristics of the study population

	General anesthesia group ($n = 23$)	Combined epidural and general anesthesia group ($n = 23$)	Combined spinal and general anesthesia group ($n = 23$)
Gender (female/male)	15/8	14/9	18/5
Age (years) (range)	54 ± 14 (30–77)	52 ± 12 (35–78)	50 ± 13 (33–76)
Weight (kg)	59 ± 11	60 ± 9	55 ± 7
Height (cm)	159 ± 8	160 ± 8	159 ± 8
Body mass index (kg/m^2)	1.6 ± 0.2	1.6 ± 0.2	1.6 ± 0.1
SVV immediately before spontaneous breathing (%)	8.2 ± 2.1	7.4 ± 2.3	7.6 ± 2.2

Values are mean ± SD (min–max)

SVV stroke volume variation

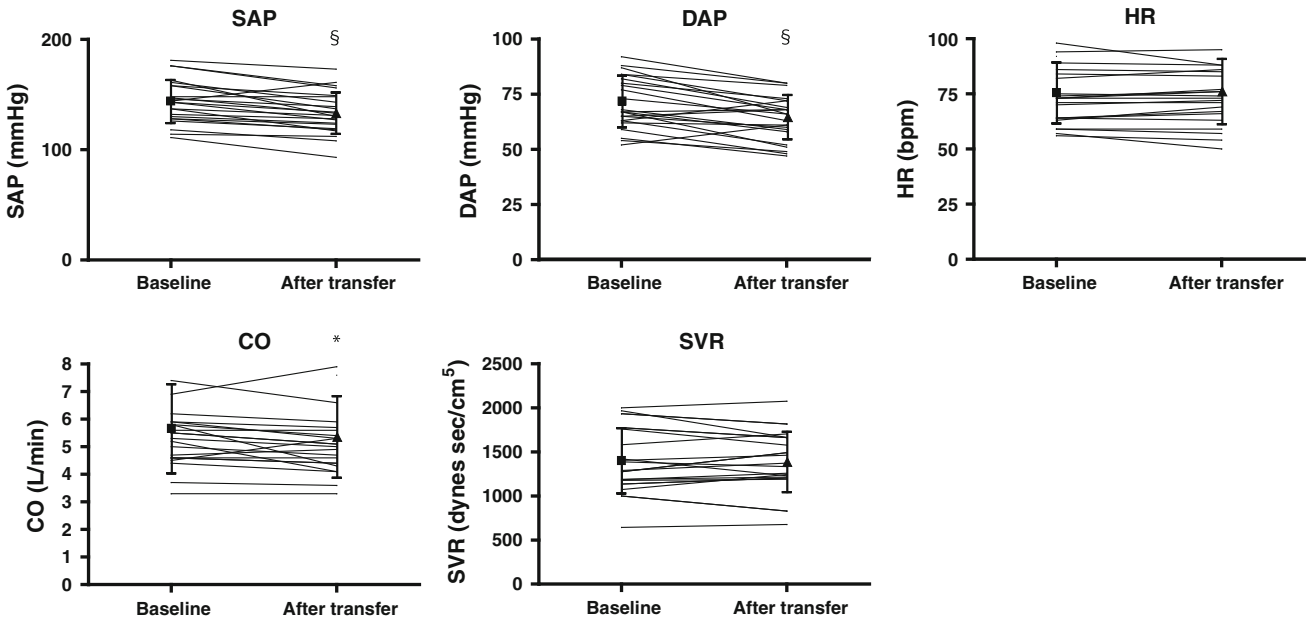

Fig. 1 Changes of hemodynamic data in the general anesthesia group. *SAP* systolic arterial pressure, *DAP* diastolic arterial pressure, *HR* heart rate, *CO* cardiac output, *SVR* systemic vascular resistance. *$P < 0.05$ versus baseline. §$P < 0.0001$ versus baseline

[9, 10], the intravascular volume status of the patients was considered to be normal.

Discussion

The main findings of this study are that the hemodynamic changes occurring after patient transfer in the general anesthesia group and combined epidural and general anesthesia group were similar but were different from those occurring in the combined spinal and general anesthesia group. HR was unchanged in both the general anesthesia and combined epidural and general anesthesia groups whereas it decreased in the combined spinal and general anesthesia group, and this is the most important finding of this study.

In all groups, CO decreased, and SVR was unchanged after transfer. Therefore, we suspected that the decrease in blood pressure was caused by a decrease in venous return. Although the mechanism responsible for the decrease in venous return is not clear from the analysis of the results of this study, we speculate that when the patients were lifted and transferred from the operating table to the bed, their intravascular volume shifted to vasculature with greater capacitance (i.e., the vasculature of body surfaces in contact with the operating table that had been compressed during surgery), and that this shift may have caused a decrease in venous return.

Bandi et al. [2] investigated hemodynamic changes after spinal anesthesia during the post-Cesarean transfer period after transfer of the patient to stretcher and on arrival at the recovery room and found that after transfer of the patients on to the stretcher there was a 5 % incidence of hypotension in the supine position (not head-up position) and a 2 % incidence of intervention (ephedrine IV) to sustain blood pressure in the supine position. They commented that

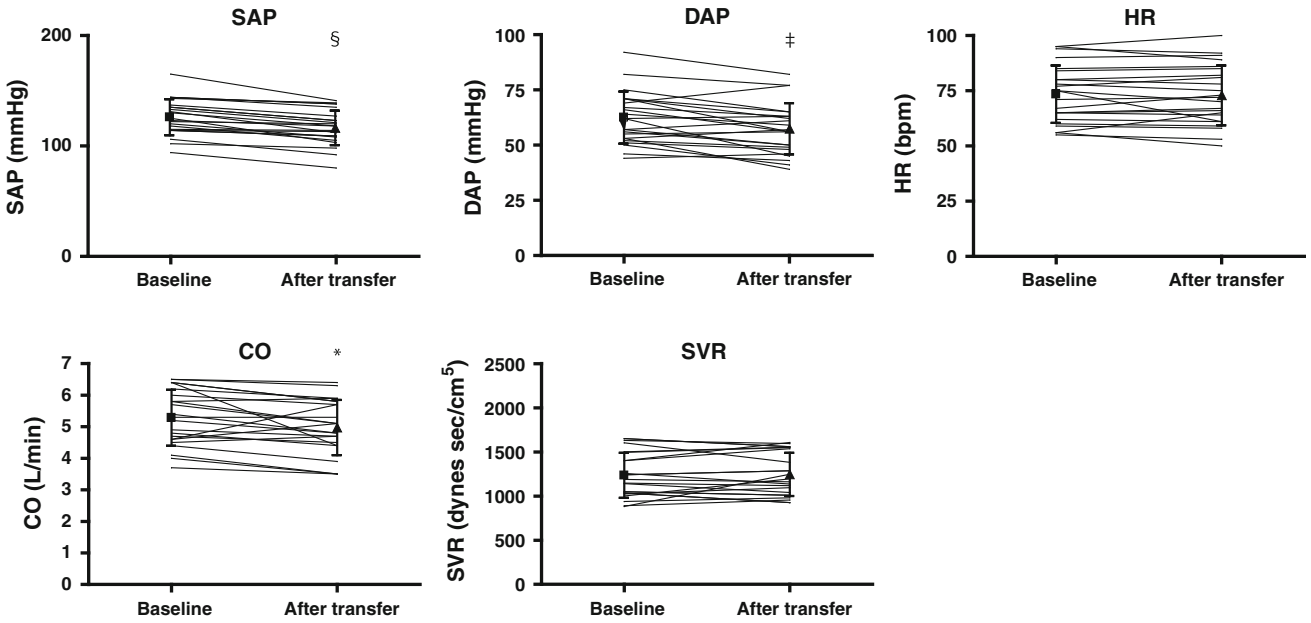

Fig. 2 Changes of hemodynamic data in the combined epidural and general anesthesia group. Abbreviations as in Fig. 1. $^{*}P < 0.05$ versus baseline. $^{\ddagger}P < 0.005$ versus baseline. $^{\S}P < 0.0001$ versus baseline

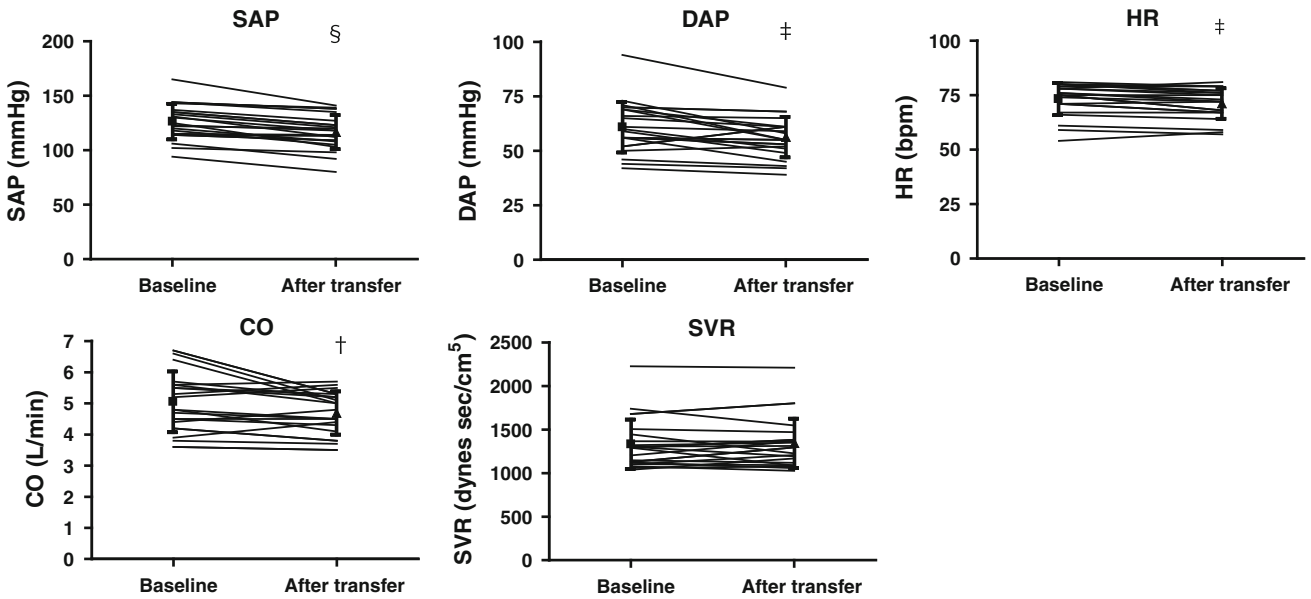

Fig. 3 Changes of hemodynamic data in the combined spinal and general anesthesia group. Abbreviations as in Fig. 1. $^{\dagger}P < 0.01$ versus baseline. $^{\ddagger}P < 0.005$ versus baseline. $^{\S}P < 0.0001$ versus baseline

hypotension may be caused by postural fluid shifts during transfer from the operating table. Although the patients in our study were awake, Cassorla and Lee [11] noted that because of pooling of blood into the lower body during general anesthesia, patients are particularly prone to hypotensive episodes after position change, and incremental positioning can reduce the extent and duration of hypotension. Our study revealed a decrease in CO with no change in SVR. Multiple factors can induce a decrease in CO in addition to a reduction in venous return, and other interpretations, for example reduced cardiac contraction are possible.

Curatolo et al. [12] conducted an observational study on 1,050 nonpregnant patients to identify patient, anesthesia, and surgery-related factors affecting the probability of hypotension and bradycardia (heart rate $\leq$45 bpm) after

epidural blockade and found that the probability of bradycardia was lower in women. Their suggested explanation was that men were more likely to react with vagal activation to the reduction of central blood volume caused by epidural block. However, the characteristics of our study population were similar in the three anesthesia groups. Moreover, Lesser et al. [13] evaluated the contributions of patient characteristics and intraoperative factors to the occurrence, severity, and clinical course of bradycardia during spinal and epidural anesthesia that did not also involve general anesthesia by use of 57,240 automated anesthesia records (obstetrical patients and patients <12 years of age were excluded) and found that spinal anesthesia was associated with an increased frequency of bradycardia episodes compared with epidural anesthesia. They also found that a baseline HR <60 bpm and male sex contributed significantly to the risk of a severe bradycardia episode. A baseline HR <60 bpm, age <37 years, male sex, nonemergency status, chronic β-blocker medications, and case duration contributed to the risk of a moderate bradycardia episode. Time of occurrence of the bradycardia event was distributed widely across the entire duration of a case [13]. They commented that mechanism resulting in moderate and severe bradycardia during neuraxial anesthesia may be different, but in any event remain poorly understood [13].

Although the decrease in HR in the combined spinal and general anesthesia group in this study can be regarded as clinically small, the change was different from that in the other two groups; therefore, we believe that particular attention should be paid to the decrease in HR that occurred in this group. Moreover, the upper analgesia level reached in the combined spinal and general anesthesia group was significantly lower than that reached in the combined epidural and general anesthesia group after surgery. Many theoretical and real factors may be associated with the development of bradycardia during spinal anesthesia [14]. Scavone et al. [15] commented in their textbook as follows: "Heart rate is a complex function of the balance between sympathetic and parasympathetic tone, as well as cardiac filling, and reflex responses to decreased preload. During spinal anesthesia restricted to low thoracic dermatomes or below, reflex increases in sympathetic activity above the level of blockade, as well as decreases in vagal activity trend to increase heart rate, helping to preserve cardiac output." Although the true mechanism of the decrease in HR in the combined spinal and general anesthesia group in this study is still unclear, attention should be paid to the decrease in HR when hypotension occurs during post-anesthesia bed transfer. Furthermore, a decrease in venous return to the heart leads to reduced stretch in the right side of the heart, which subsequently leads to a decrease in HR (Bainbridge reflex). A paradoxical form of the Bezold–Jarisch reflex is also believed to occur rarely during spinal anesthesia, leading to severe bradycardia and asystole [14, 16–18]. Because HR decreased in the combined spinal and general anesthesia group in this study, these reflexes might have been involved in the decrease of blood pressure, especially that observed in the combined spinal and general anesthesia group, although the decrease in HR was clinically small.

A study of the recovery period after spinal anesthesia of non-obstetric patients revealed reduced incidence of bradycardia in the hammock position (legs and trunk both elevated 30°) compared with the supine and Trendelenburg positions [19]. Bandi et al. [2] investigated hemodynamic changes during the post-Cesarean transfer period after spinal anesthesia and found that for 10 % of patients transport to the recovery room is associated with the development of substantial hypotension that is unaffected by position. They recommended routine monitoring of blood pressure and HR after transfer to the trolley and recording of the sensory level of block at the end of Cesarean section [2]. We also agree with their recommendations, and further, we strongly recommend routine monitoring of blood pressure and HR of patients after transfer to another bed, even after general anesthesia and epidural anesthesia.

For this study we chose patients without coexisting disease and who were not hypovolemic; nevertheless, when we transferred the patients, their arterial blood pressure decreased significantly. Thus, further study is needed to understand the hemodynamics of bed transfer after anesthesia for patients with coexisting disease and/or hypovolemia.

Our study had several limitations: first, although we naturally wanted to compare three different methods of anesthesia (general anesthesia only, epidural anesthesia only, and spinal anesthesia only), this study compared three anesthesia regimens: general anesthesia only, combined epidural and general anesthesia, and combined spinal and general anesthesia, and we cannot deny that the effects of general anesthetics (propofol and opioids) might modify, to some extent, the results obtained in the combined epidural and general anesthesia and combined spinal and general anesthesia groups. Second, we gave atropine and neostigmine to all patients; thus, there was a likelihood that these drugs affected autonomic activity and possibly also modified the effects of general and regional anesthetics. In a future study, use of sugammadex, instead of atropine and neostigmine, should be considered.

In conclusion, the decrease in blood pressure observed after transfer of a postsurgical patient to another bed after general, combined epidural and general, and combined spinal and general anesthesia was associated with a decrease in CO with no change in SVR, but HR decreased

after combined spinal and general anesthesia whereas it remained unchanged after general and combined epidural and general anesthesia. We assumed that this decrease in blood pressure was caused by a decrease in venous return, and several reflexes might be involved in the decrease of blood pressure, especially after combined spinal and general anesthesia. We strongly recommend routine monitoring of blood pressure and HR after transfer of a postsurgical patient to another bed immediately after awakening from anesthesia, and particular attention should be paid when patients are transferred, especially patients receiving combined spinal and general anesthesia, because a decrease in blood pressure and HR may occur. Further investigation of critically ill patients (e.g., those with massive bleeding, heart attack, or diseases affecting the autonomic nervous system) will be required.

References

1. Verdeyen J, Ory JP, Wyckmans W, Vandermeersch E, Jamaer L, Van Assche A. Prevention of postoperative hypotension following spinal anesthesia for TURP: a double-blind randomized controlled trial comparing ephedrine with placebo. Acta Anaesthesiol Belg. 2008;59:73–8.
2. Bandi E, Weeks S, Carli F. Spinal block levels and cardiovascular changes during post-Cesarean transport. Can J Anaesth. 1999; 46:736–40.
3. Wajima Z, Shitara T, Ishikawa G, Inoue T, Ogawa R. Analgesia after upper abdominal surgery with extradural buprenorphine with lidocaine. Can J Anaesth. 1998;45:28–33.
4. Wajima Z, Shitara T, Ishikawa G, Kaneko K, Inoue T, Ogawa R. Analgesia after upper abdominal surgery using extradural administration of a fixed dose of buprenorphine in combination with lignocaine given at two infusion rates: a comparative study. Acta Anaesthesiol Scand. 1997;41:1061–5.
5. Biais M, Vidil L, Sarrabay P, Cottenceau V, Revel P, Sztark F. Changes in stroke volume induced by passive leg raising in spontaneously breathing patients: comparison between echocardiography and Vigileo/FloTrac device. Crit Care. 2009;13:R195.
6. Wajima Z, Shiga T, Imanaga K, Inoue T. Assessment of the effect of rapid crystalloid infusion on stroke volume variation and pleth variability index after a preoperative fast. J Clin Monit Comput. 2010;24:385–9.
7. de Leeuw MA, Slagt C, Hoeksema M, Zuurmond WW, Perez RS. Hemodynamic changes during a combined psoas compartment-sciatic nerve block for elective orthopedic surgery. Anesth Analg. 2011;112:719–24.
8. Wajima Z, Shiga T, Imanaga K, Inoue T. Do induced hypertension and hypotension affect stroke volume variation in man? J Clin Anesth. 2012;24:207–11.
9. Cannesson M, Musard H, Desebbe O, Boucau C, Simon R, Hénaine R, Lehot JJ. The ability of stroke volume variations obtained with Vigileo/FloTrac system to monitor fluid responsiveness in mechanically ventilated patients. Anesth Analg. 2009; 108:513–7.
10. Biais M, Nouette-Gaulain K, Cottenceau V, Revel P, Sztark F. Uncalibrated pulse contour-derived stroke volume variation predicts fluid responsiveness in mechanically ventilated patients undergoing liver transplantation. Br J Anaesth. 2008;101:761–8.
11. Cassoria L, Lee J-W. Patient positioning and anesthesia. In: Miller RD, editor. Miller's anesthesia, vol 1. Philadelphia, PA: Churchill Livingstone Elsevier; 2010. p. 1163.
12. Curatolo M, Scaramozzino P, Venuti FS, Orlando A, Zbinden AM. Factors associated with hypotension and bradycardia after epidural blockade. Anesth Analg. 1996;83:1033–40.
13. Lesser JB, Sanborn KV, Valskys R, Kuroda M. Severe bradycardia during spinal and epidural anesthesia recorded by an anesthesia information management system. Anesthesiology. 2003;99:859–66.
14. Urmey WF. Case studies of regional anesthesia. In: Finucane BT, editor. Complications of regional anesthesia. 2nd ed. New York: Springer Science + Business Media, LLC.; 2007. p. 417–20.
15. Scavone BM, Ratliff J, Wong CA. Physiologic effects of neuraxial anesthesia. In: Wong CA, editor. Spinal and epidural anesthesia. Columbus: McGraw–Hill; 2007. p. 115–6.
16. Tarkkila P. Complications associated with spinal anesthesia. In: Finucane BT, editor. Complications of regional anesthesia. 2nd ed. New York: Springer Science + Business Media, LLC.; 2007. p. 151.
17. Mackey DC, Carpenter RL, Thompson GE, Brown DL, Bodily MN. Bradycardia and asystole during spinal anesthesia: a report of three cases without morbidity. Anesthesiology. 1989;70: 866–8.
18. Campagna JA, Carter C. Clinical relevance of the Bezold–Jarisch reflex. Anesthesiology. 2003;98:1250–60.
19. Ponhold H, Vicenzi MN. Incidence of bradycardia during recovery from spinal anaesthesia: influence of patient position. Br J Anaesth. 1998;81:723–6.