J Anesth (2012) 26:900–904
DOI 10.1007/s00540-012-1431-5

CLINICAL REPORT

Heart rate variability during abdominal surgical manipulation under general and epidural anesthesia

Naoyuki Hirata · Ryo Miyashita · Daisuke Maruyama · Ryoichi Kawaguchi · Hitoshi Shimizu · Michiaki Yamakage

Received: 6 April 2012 / Accepted: 30 May 2012 / Published online: 21 June 2012

Abstract Anesthesiologists occasionally encounter bradycardia during abdominal surgery and recognize the phenomenon as a vagal reflex. The presence of bradycardia implies efferent vagal dominance in the autonomic nervous system during this vagal reflex. In this study, we investigated the effect of abdominal surgical manipulation on autonomic nervous activity, using heart rate variability analysis. Abdominal surgical manipulation decreased the heart rate and enhanced not only the high-frequency power (0.15–0.4 Hz) but also the low-frequency power (0.04–0.15 Hz) calculated from the power spectral density of heart rate variability. Our results suggest that both vagal tone and sympathetic tone could be activated during the vagal reflex caused by abdominal surgical manipulation.

N. Hirata · H. Shimizu
Department of Anesthesiology, Obihiro Kyokai Hospital, Obihiro, Japan
e-mail: jirokujr@gmail.com

N. Hirata (✉) · R. Miyashita · D. Maruyama · R. Kawaguchi · M. Yamakage
Department of Anesthesiology, Sapporo Medical University School of Medicine, South 1, West 16, Chuo-ku, Sapporo, Hokkaido 060-8543, Japan
e-mail: naohirata@mac.com

R. Miyashita
e-mail: evolution5_2002@yahoo.co.jp

D. Maruyama
e-mail: d.maruyama@sapmed.ac.jp

R. Kawaguchi
e-mail: r.kawaguchi@sapmed.ac.jp

M. Yamakage
e-mail: yamakage@sapmed.ac.jp

Keywords Heart rate variability · Vagal reflex · Autonomic nervous activity

Introduction

Abdominal surgery, laparoscopy, laryngoscopy, and ophthalmic surgery under general anesthesia can trigger bradycardia and/or hypotension [1]. It is thought that a vagal reflex induced by these procedures leads to hemodynamic suppression. However, there is no study analyzing the detailed autonomic nervous activity, including sympathetic activity, which could compensate for increased parasympathetic tone during such a vagal reflex.

Heart rate variability (HRV) analysis has been widely used for assessing autonomic nervous system activity [2], and this analysis can not only detect autonomic dysfunction but can also predict long-term postoperative mortality [2–4]. The aim of the present study was to determine autonomic nervous activity, by using HRV analysis, during abdominal surgical manipulation in patients under general and epidural anesthesia.

Patients and methods

The institutional Ethics Committee at Obihiro Kyokai Hospital (Obihiro, Japan) approved this study, and all patients granted their written informed consent. Twenty-two adult patients (American Society of Anesthesiologists physical status class I or II) scheduled for elective abdominal general surgery were studied. Patients with congestive heart failure or arrhythmias (not in sinus rhythm) were excluded. On the patient's arrival at the operating room, noninvasive blood pressure (BP), heart

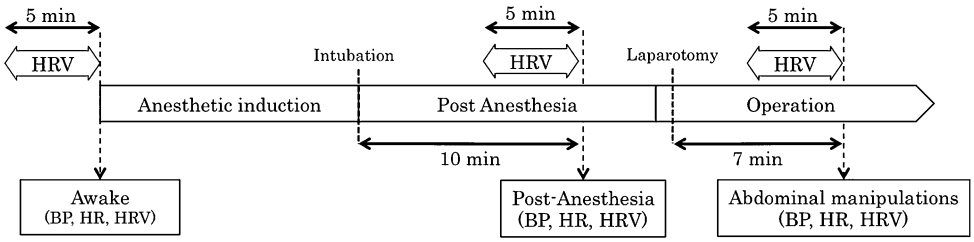

Fig. 1 Study protocol. *HRV*, Heart rate variability, *BP* blood pressure, *HR* heart rate

rate (HR), arterial oxygen saturation, bispectral index, and HRV were recorded with the patient in the supine position, and this period was classified as 'Awake'. Respiratory rates were maintained at 12–15 bpm in all patients, using a metronome, because respiratory rates could modulate HRV. HRV was determined with an HRV analyzer (SA-300P; Tokyo Iken, Tokyo, Japan), which used a 5-min epoch of HR to analyze HRV. A finger-clip sensor, which can detect R–R intervals from a plethysmogram, was placed on the index finger. To determine autonomic nervous activity, the total power of R–R interval data (TP; 0–0.4 Hz), and the low-frequency (LF; 0.04–0.15 Hz) and high-frequency (HF; 0.15–0.4 Hz) power were calculated from power spectral density analysis and recorded in absolute units (ms^2). TP reflects overall autonomic activity where sympathetic activity is a primary contributor. HF reflects parasympathetic activity and LF reflects both sympathetic and parasympathetic activity [2, 5]; in general, LF and the LF/HF ratio are indicators of sympathetic activity [6]. Figure 1 shows the study protocol. All patients received thoracic epidural anesthesia at the Th8-12 level to exclude the effects of pain stimulation on HRV. After epidural catheterization, 1.5 % lidocaine (5–8 mL) was administered via a catheter and local anesthesia onset was confirmed by the cold test at the site of skin incision 5 min after the administration of lidocaine. General anesthesia was induced with propofol (1.5 mg/kg), fentanyl (1 μg/kg), and vecuronium (0.1 mg/kg) after oxygenation. Anesthesia was maintained with sevoflurane (1.0–1.5 %) with oxygen (35–45 %) and vecuronium at a bispectral index of around 50. Tracheal intubation was performed 3 min after the administration of vecuronium. During anesthesia induction, respiratory rates and end-tidal pCO_2 were maintained at 12–15 bpm and 35–45 mmHg, respectively. HR, BP, and HRV were recorded for 10 min after tracheal intubation and this time period was classified as 'Post-anesthesia'. An additional recording was performed for 7 min from the beginning of laparotomy until abdominal surgical manipulation was completed, and this time period was classified as 'Abdominal manipulation'. Data were expressed as the means ± SD for each of the three time period measurements. BP and HR were compared among the three time periods using repeated-measures analysis of variance (ANOVA) followed by the Scheffè post-hoc test, and HRV parameters were compared using the Friedman test. *P* values of <0.05 were considered statistically significant.

Results

Table 1 shows the demographic data. Two patients were excluded from the analysis because they had received atropine (0.5 mg)–which could have affected HRV–for bradycardia (HR <45 bpm) during anesthesia induction. Therefore, data for 20 patients were analyzed in this study. Figures 2 and 3a–d respectively, show the hemodynamic and HRV data during the study. The anesthetic procedures decreased systolic BP (SBP) and HR. Abdominal surgical manipulation significantly decreased HR, but not SBP, compared with post-anesthesia values (Fig. 2). In the HRV analysis, anesthesia strongly suppressed TP, LF, HF, and the LF/HF ratio compared with the awake values. On the other hand, abdominal surgical manipulation enhanced all the parameters of HRV compared with the post-anesthesia

Table 1 Demographic data

Demographic data	Patients ($n = 20$)
Age (years)	65.3 ± 9.3
Male/female	11/9
BMI (kg/m^2)	23.0 ± 3.5
ASA PS (I/II)	11/9
Preoperative complications	***n***
Hypertension	5
Diabetes mellitus	4
Ischemic heart disease	1
Surgical procedures	***n***
Hemicolectomy	8
Low anterior resection	4
Gastrectomy	4
Rectectomy	2
Cholecystomy	2

BMI body mass index, *ASA* American Society of Anesthesiologists, *PS* physical status

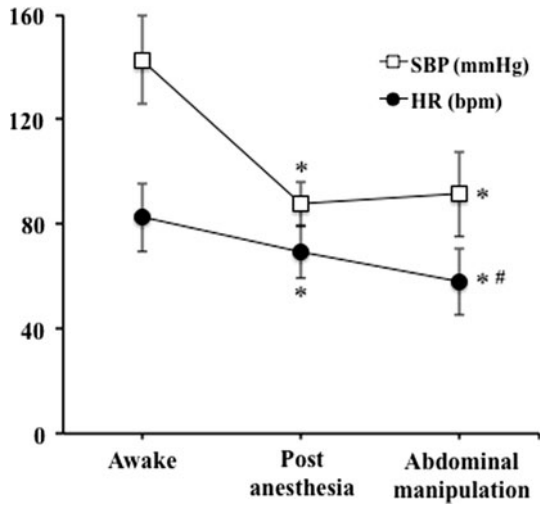

Fig. 2 Changes in hemodynamics during the study. Data are expressed as means ± SD. $^{*}P < 0.05$ versus awake and $^{\#}P < 0.05$ versus post-anesthesia. *SBP* Systolic blood pressure

values (Fig. 3a–d). LF/HF during abdominal surgical manipulation was significantly higher than that in the awake period, while HF during abdominal surgical manipulation was lower than that in the awake period.

Discussion

Our results showed that autonomic nervous activity dramatically varied according to anesthetic procedures and abdominal surgical manipulation. Importantly, our HRV data suggested that abdominal surgical manipulation could stimulate not only vagal activity but also sympathetic activity. Of interest, when the sympathetic and vagal nervous systems are simultaneously stimulated, the clinical outcome may present vagal dominance, shown as bradycardia.

It is well accepted that surgical manipulations occasionally result in a vagal reflex leading to bradycardia [1], and anesthesiologists believe that the sympathetic nervous system should be stimulated for compensation during vagal reflex. Nevertheless, no study has previously analyzed autonomic nervous activity in this type of vagal reflex by using HRV analysis, which is noninvasive and is widely used to measure autonomic nervous system activity [1–3]. Several studies using HRV analysis have shown that, in the perioperative period, autonomic nervous activity under general anesthesia depends on the agents used and/or the depth of anesthesia [3, 6–9]. In the present study, the anesthetic procedures significantly reduced both the LF and HF components, and this confirms our previous results [7]. HF has been well established as an indicator of vagal nervous activity triggered by respiration, and LF and/or the LF/HF ratio mainly reflect sympathetic activity [6]. Therefore, the present results imply that anesthetic procedures could decrease both the sympathetic tone and the vagal tone. Interestingly, we found that abdominal surgical manipulation under general and epidural anesthesia enhanced LF, HF, and the LF/HF ratio, although our finding of a decrease in HR under these conditions suggests only the occurrence of vagal stimulation. These results for HRV suggest that neurons of both the sympathetic and vagal nervous systems could discharge simultaneously during vagal bradycardia. Previously, Schubert et al. [10] demonstrated that manual abdominal exploration enhanced LF, HF, and the LF/HF ratio, similar to our results in abdominal surgery. However, in their study [10], BP and HR were increased by manual abdominal exploration and vagal bradycardia did not occur. Also in their study, no neuroaxial block was performed, and the dose of fentanyl (214 ± 93 μg) seemed to be insufficient for exerting anti-nociceptive effects in abdominal surgery. Therefore, their HRV data might simply represent sympathetic activation caused by surgical pain. We note that Schubert et al. [10] did not take account of the effects of surgical pain and vagal reflex on HRV during the abdominal exploration; however, we focused on HRV in the vagal reflex during abdominal surgery under general and epidural anesthesia. To determine HRV in the vagal reflex during abdominal manipulation, it is crucial to exclude surgical pain, which could strongly increase sympathetic tone. We believe that our epidural anesthesia had sufficient anti-nociceptive effects against surgical stimuli and we also believe that the concentration of sevoflurane was appropriate, because BP, HR, and the bispectral index did not increase during laparotomy and abdominal surgical manipulation.

We found that HF during abdominal manipulation was significantly lower than that in the awake period, while LF during abdominal manipulation was the same as that during the awake period. Consequently, the LF/HF ratio during abdominal manipulation was significantly higher than that in the awake period. Unoki et al. [11] found that, under deep sedation, the HF component was more likely to be suppressed than the LF component, although the detailed mechanisms underlying this difference are still unknown. In our study, general anesthesia, which could be regarded as deep sedation, might have been responsible for the differential modulation of the HF and LF components.

The present results should be interpreted within the constraints of several limitations. We believe that LF and LF/HF can represent sympathetic activity, but we note that the interpretation of LF is still controversial [12–16]. A future study, e.g., one including an analysis of catecholamine levels during the vagal reflex, is required to interpret LF and sympathetic activity precisely. In addition, we note that the epidural anesthesia in the present study could have

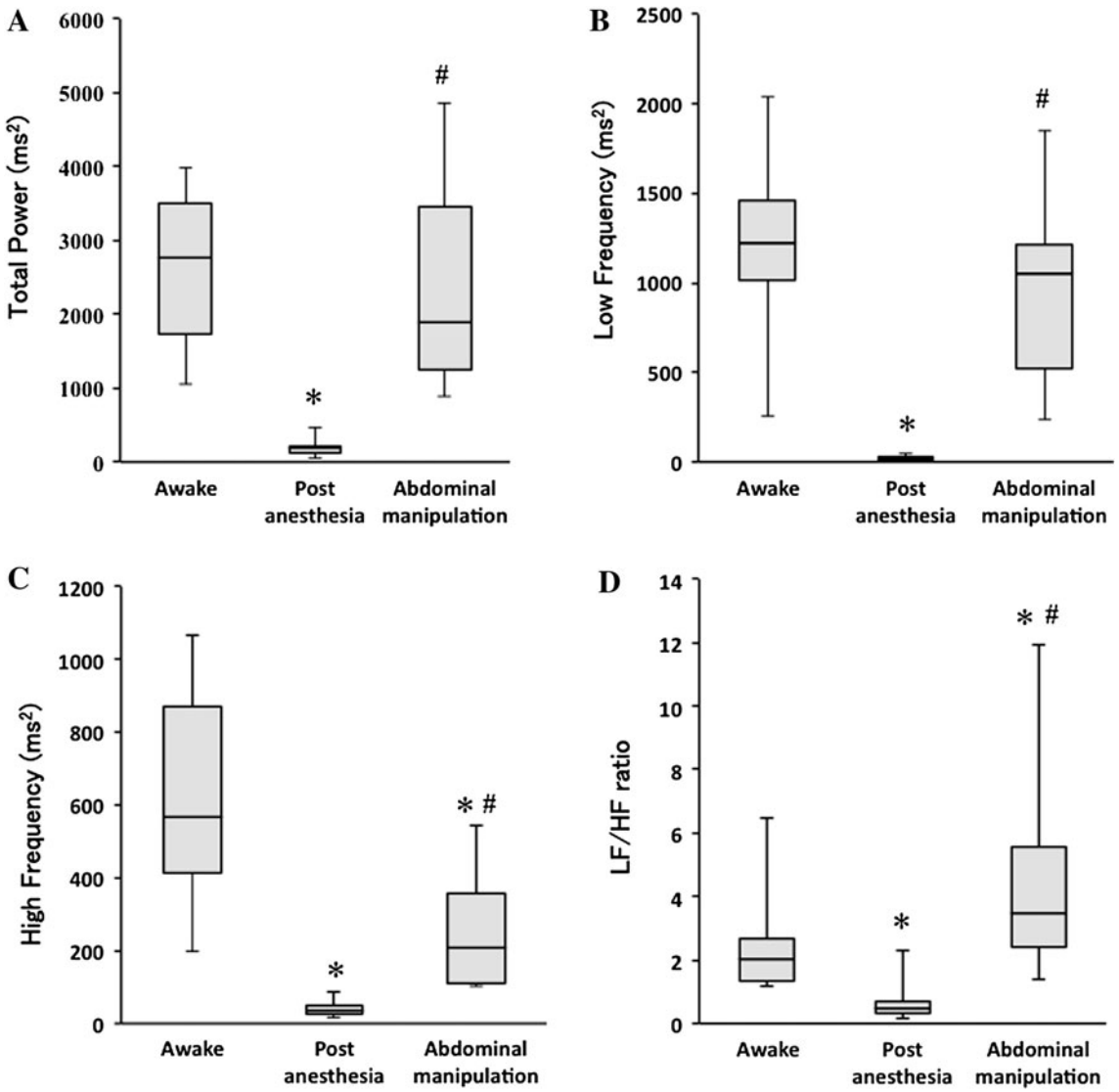

Fig. 3 Changes in HRV during the study. Medians and interquartile ranges of **a** total power (ms^2), **b** low frequency (LF; ms^2), **c** high frequency (HF; ms^2), and **d** the LF/HF ratio. $^{*}P < 0.05$ versus awake and $^{\#}P < 0.05$ versus post-anesthesia

led to modulation of the autonomic nervous system; namely, our epidural analgesia might have blocked cardiac sympathetic fibers and affected the results of the HRV analysis. Tanaka et al. [17] have demonstrated that a high level of epidural blocks (median upper/lower sensory block: C3/T8) produced a significant decrease not only in LF but also in HF. Thus, the effects of epidural block on cardiac sympathetic fibers and subsequent modulation of HRV are still controversial. The precise mechanisms regarding the effects of epidural anesthesia on HRV remain to be determined.

In conclusion, our study using HRV analysis suggested that abdominal surgical manipulation under general and epidural anesthesia could stimulate not only vagal nervous activity but also sympathetic activity. When the vagal and sympathetic nervous systems are activated simultaneously, the hemodynamics may indicate vagal dominance.

Acknowledgments This research was supported solely by institutional and/or departmental sources. None of the authors have any financial interests in products related to this study.

References

1. Doyle DJ, Mark PW. Reflex bradycardia during surgery. Can J Anaesth. 1990;37:219–22.
2. Task Force of European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Circulation 1996;93:1043–65.

3. Mazzeo AT, La Monaca E, Di Leo R, Vita G, Santamaria LB. Heart rate variability: a diagnostic and prognostic tool in anesthesia and intensive care. Acta Anaesthesiol Scand. 2011;55:797–811.
4. Laitio T, Jalonen J, Kuusela T, Sheinin H. The role of heart rate variability in risk stratification for adverse postoperative cardiac events. Anesth Analg. 2007;105:1548–60.
5. Lahiri MK, Kannankeril PJ, Goldberger JJ. Assessment of autonomic function in cardiovascular disease: physiological basis and prognostic implications. J Am Coll Cardiol. 2008;51:1725–33.
6. Akselrod S, Gordon D, Ubel FA, Shanon DC, Berger AC, Cohen RJ. Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat-to beat cardiovascular control. Science. 1981;213:220–2.
7. Kanaya N, Hirata N, Kurosawa S, Nakayama M, Namiki A. Differential effects of propofol and sevoflurane on heart rate variability. Anesthesiology. 2003;98:34–40.
8. Tsuchiya S, Kanaya N, Hirata N, Kurosawa S, Kamada N, Edanaga M, Nakayama M, Omote K, Namiki A. Effects of thiopental on bispectral index and heart rate variability. Eur J Anaesthesiol. 2006;23:454–9.
9. Riznyk L, Fijalkowska M, Przesmycki K. Effects of thiopental and propofol on heart rate variability during fentanyl-based induction of general anesthesia. Pharmacol Rep. 2005;57:128–34.
10. Schubert A, Palazzolo JA, Brum JM, Ribeiro MP, Tan M. Heart rate, heart rate variability, and blood pressure during perioperative stressor events in abdominal surgery. J Clin Anesth. 1997; 9:52–60.
11. Unoki T, Grap MJ, Sessler CN, Best AM, Wetzel P, Hamilton A, Mellott KG, Munro CL. Autonomic nervous system function and depth of sedation in adults receiving mechanical ventilation. Am J Crit Care. 2009;18:42–50.
12. Lombardi F, Stein PK. Origin of heart rate variability and turbulence: an appraisal of autonomic modulation of cardiovascular function. Front Physiol. 2011;95:1–7.
13. Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, Pizzinelli P, Sandrone G, Malfatto G. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympathovagal interaction in man and conscious dog. Circ Res. 1986; 59:178–93.
14. Pagani M, Somers V, Furlan R. Changes in autonomic regulation induced by physical training in mild hypertension. Hypertension. 1988;12:600–10.
15. Houle MS, Billman GE. Low-frequency component of heart rate variability spectrum: a poor marker of sympathetic activity. Am J Physiol. 1999;276:H215–23.
16. Hopf HB, Skyschally A, Heusch G, Peters J. Low-frequency spectral power of heart rate variability is not a specific marker of cardiac sympathetic modulation. Anesthesiology. 1995;82:609–19.
17. Tanaka M, Goyagi T, Kimura T, Nishikawa T. The effects of cervical and lumbar epidural anesthesia on heart rate variability and spontaneous sequence baroreflex sensitivity. Anesth Analg. 2004;99:924–9.