Effect of Increasing Heart Rate and Tidal Volume on Stroke Volume Variability in Vascular Surgery Patients

Nathan A. Roeth, MD,* Timothy R. Ball, MD,* William C. Culp Jr, MD,* W. Todd Bohannon, MD,† Marvin D. Atkins, MD,† and William E. Johnston, MD*

Objective: **Because heart rate affects ventricular filling, the aim of the present study was to assess the effects of increasing heart rate and tidal volume on stroke volume variability to determine whether this dynamic index is heart-rate dependent.**

Design: **Prospective, randomized study.**

Setting: **Single university hospital.**

Participants: **Eighteen vascular surgery patients having general anesthesia and endotracheal intubation with an arterial catheter connected to the Vigileo FloTrac system (Edwards Lifesciences, Irvine, CA) and a transesophageal atrial pacemaker (CardioComman Inc, Tampa, FL).**

Intervention: **A 2×2 factorial study of changes in heart rate (80 bpm and 110 bpm) and tidal volume (6 mL/kg and 10 mL/kg).**

Measurements and Main Results: **With tidal volume at 6 mL/kg, increasing heart rate from 80 mL/kg to 110 bpm caused stroke volume variability to increase from 12.2% ± 5.7% to 13.2% ± 5.3% ($p < 0.05$), and with tidal volume at 10 mL/kg, increasing heart rate from 80 mL/kg to 110 bpm caused stroke volume variability to increase from 19.7% ± 7.9% to 22.0% ± 8.6% ($p < 0.05$). In comparison, increasing tidal volume from 6 mL/kg to 10 mL/kg produced a significantly greater effect on stroke volume variability than increasing heart rate.**

Conclusions: **Stroke volume variability is sensitive to increases in heart rate in addition to tidal volume. Increasing heart rate caused stroke volume variability to increase significantly, although not to the same magnitude as increasing tidal volume. When using dynamic volume indices, clinicians should be aware of increases in heart rate, although its clinical impact may be relatively minor compared with changes in tidal volume.**

KEY WORDS: heart rate, tidal volume, stroke volume variability, surgical patients, general anesthesia, positive-pressure ventilation

A PRIMARY GOAL of hemodynamic management in critically ill patients is adequate volume replacement to achieve optimal cardiac performance. Dynamic volume indices such as stroke volume variation (SVV) supplement the use of static indices of filling pressures for predicting patient responsiveness to fluid resuscitation.[1,2] These dynamic indices relate to the cyclical changes in arterial blood pressure resulting from acute changes in right ventricular (RV) and left ventricular (LV) loading conditions involving both preload and afterload caused by positive-pressure ventilation. Any reduction in RV filling, such as occurring with hemorrhage, hypovolemia, an increase in tidal volume (V_t), or a reduction in chest wall compliance can further decrease RV stroke volume (SV) and linearly increase the variability in dynamic indices.[3–5]

Another potentially relevant factor that can alter venous return and RV preload is heart rate (HR). It long has been known that increasing HR by atrial pacing decreases LV filling pressures and SV[6] so that cardiac output remains unchanged.[7,8] More recent studies have shown that tachycardia by atrial pacing causes linear reductions in right-sided[8] and, consequently, left-sided[9] preload, thereby reducing SV by the Frank-Starling relationship. In the clinical management of hypovolemic patients, the overall reduction in cardiac filling may be compounded by a reflex tachycardia.

The effect of increasing HR on a dynamic index such as SVV has not been assessed. The authors hypothesized that increasing HR would increase the amount of SVV in anesthetized patients receiving positive-pressure ventilation. If so, the clinical use of these dynamic criteria would need to consider potential confounding effects of HR changes independent of the patient's volume status. Because V_t already has been shown to be a significant factor affecting SVV,[4] the effect of increasing HR by atrial pacing was compared with that of increasing V_t in perioperative vascular surgical patients to assess their relative magnitudes of effect.

MATERIALS AND METHODS

This research study was approved by the institutional review board, and written informed consent was obtained from each patient before study enrollment. Patients undergoing elective vascular surgery were enrolled and consented for study. Exclusion criteria included rhythm other than sinus, ejection fraction less than 50%, significant valvular heart disease, or patient refusal. Patients had nothing by mouth for at least 8 hours before surgery and were hydrated with 500 mL to 750 mL of 0.9% normal saline in the preoperative holding area. Twenty-two nonemergent American Society of Anesthesiology 3 and 4 patients in normal sinus rhythm were studied after induction of general anesthesia before initiation of various vascular surgeries. Each enrolled subject received a 20-gauge radial arterial catheter connected to a FloTrac/Vigileo algorithm software system (version 3.02, Edwards LifeSciences, Irvine, CA). Upon arrival in the operating room, standard monitors were applied to the patient, along with a bispectral index monitor (BIS Brain Monitor, Covidien, Mansfield, MA). General anesthesia was induced with intravenous propofol, 0.5 mg/kg to 1.0 mg/kg, sufentanil, 10 μg to 25 μg, and rocuronium, 0.6 mg/kg to 0.8 mg/kg, to facilitate endotracheal intubation. After intubation, a transesophageal atrial pacer (CardioComman, Inc., Tampa, FL) was inserted in the esophagus and atrial capture was confirmed with corresponding heart rate changes on the electrocardiogram and arterial waveform tracings. Patients with

*From the Departments of *Anesthesiology and †Surgery (Division of Vascular Surgery), Texas A&M University System Health Science Center College of Medicine, Temple, TX.*

This study was funded in part by a resident research grant from S&W Healthcare. Presented as an abstract at the 42nd Annual Congress of the Society of Critical Care Medicine, January 19-23, 2013, San Juan, Puerto Rico.

Address reprint requests to William E. Johnston, MD, Department of Anesthesiology, Scott & White Healthcare, 2401 South 31st Street, Temple, TX 76508. E-mail: wjohnston@sw.org

1053-0770/2601-0001$36.00/0

http://dx.doi.org/10.1053/j.jvca.2014.05.014

arrhythmias during pacing or without pacemaker capture were eliminated from the study protocol. General anesthesia was maintained with 0.5 MAC of sevoflurane and 100% oxygen. Bispectral index monitor readings were measured repeatedly to ensure an adequate depth of anesthesia throughout data collection. The respiratory rate initially was adjusted to maintain end-tidal carbon dioxide tension between 32 mmHg and 35 mmHg, with a baseline V_t of 6 mL/kg body weight and remained constant throughout the experimental protocol.

Each subject was assigned randomly to a 2 × 2 factorial design of changes in HR (80 bpm or 110 bpm) and V_t (6 mL/kg body weight or 10 mL/kg body weight). After each change in HR and V_t, a period of 3 minutes elapsed to allow for physiologic stabilization. At the end of each period, various hemodynamic parameters were recorded and the next randomized physiologic change was initiated. After the fourth and final physiologic change, V_t and HR were adjusted at the attending anesthesiologist's discretion, and the surgical case proceeded. If during any of the 4 study periods hemodynamic instability (defined as a decrease in mean arterial blood pressure below 50 mmHg) ensued, the attending anesthesiologist stopped the study and treated the patient as clinically indicated and that subject was eliminated from the data analysis.

At each measurement period, the various hemodynamic parameters were acquired. From the Vigileo FloTrac system, measurements included cardiac output (CO), cardiac index (CI), SV, stroke volume index (SVI), and SVV. The invasive blood pressures also were recorded at each interval, including systolic arterial pressure, diastolic arterial pressure, and mean arterial pressure. Recorded pulmonary parameters from each period included peak airway pressure as well as V_t.

On the basis of data from a previous study that examined the effects of increasing HR on RV filling and SV,[8] it was decided to study 16 patients. Accordingly, to account for potential dropout during the study, 25% more patients were enrolled for a final sample size of 22 patients. The research study was a 2 × 2 randomized factorial design, with the 2 factors being HR and V_t. Data, presented as mean ± standard deviation, were analyzed by ANOVA, which included a combined group effect if there was no interaction between groups. Statistical significance was considered at $p < 0.05$.

RESULTS

Twenty-two subjects were screened and enrolled in the study. Three subjects were excluded from the study secondary to hypotension during the study protocol and intervention by the attending anesthesiologist; one other patient was excluded due to proper capture failure by the transesophageal atrial pacer. Eighteen patients successfully completed the study protocol, and their surgeries consisted of femoral-popliteal artery bypass, carotid endarterectomy, and femoral-femoral artery bypass. Table 1 displays the demographics for the patients included in the data analysis.

Table 1. Patient Demographic Characteristics (n = 18 patients)

Variable	
Gender	
Male	11 (61%)
Female	7 (39%)
Age (years)	67 ± 15
Weight (kg)	76 ± 14
Height (cm)	172 ± 10
Body mass index	26 ± 4

NOTE. Data are expressed as mean ± standard deviation or frequency (%).

Table 2. Hemodynamic Measurements at the 4 Different Measurement Intervals

	Heart Rate 80 bpm		Heart Rate 110 bpm	
Measurement	V_t 6 mL/kg	V_t 10 mL/kg	V_t 6 mL/kg	V_t 10 mL/kg
Systolic arterial pressure (mmHg)	111 ± 23	103 ± 21†	113 ± 24	101 ± 22†
Diastolic arterial pressure (mmHg)	54 ± 8	52 ± 8†	61 ± 8*	55 ± 8*†
Mean arterial pressure (mmHg)	75 ± 14	68 ± 11†	77 ± 13	71 ± 12†
Cardiac output (L/min)	5.7 ± 1.3	5.1 ± 1.4†	6.3 ± 1.4*	5.6 ± 1.5*†
Stroke volume (mL/beat)	72 ± 15	63 ± 17†	57 ± 12*	51 ± 14*†
Tidal volume (mL)	451 ± 85	757 ± 143†	451 ± 85	757 ± 143†
Peak airway pressure (mmHg)	12 ± 4	18 ± 5	12 ± 4*	17 ± 6*

NOTE. Data expressed as mean ± standard deviation.
*$p < 0.05$ compared with corresponding heart rate 80 bpm value.
†$p < 0.05$ compared with corresponding V_t 6 mL/kg value.

Hemodynamic variables for the 4 measurement intervals during the experimental protocol are shown in Table 2. Increasing V_t from 6 mL/kg to 10 mL/kg produced significant and expected decreases in SV, cardiac output, and arterial blood pressures. Increasing HR from 80 bpm to 110 bpm caused a significant 20% reduction in SV at each V_t setting, although cardiac output increased overall. Diastolic arterial blood pressure increased significantly with an increase in HR without changes in the systolic arterial blood pressure.

There were significant and independent HR and V_t effects on SVV. Fig 1 summarizes the data from the 4 measurement periods, with combined group effects because no interactions between HR and V_t were found. Combining data from both V_t 6 mL/kg and 10 mL/kg groups in the 2 × 2 model, atrial pacing at 110 bpm HR produced greater SVV (17.6% ± 8.3%) than 80 bpm HR (15.9% ± 7.8%; $p = 0.027$). Similarly, when data from both HR 80 bpm and 110 bpm groups were combined, increasing V_t to 10 mL/kg produced significantly greater SVV (20.8 ± 8.2%) than ventilating with V_t 6 mL/kg (12.7% ± 5.5%; $p < 0.0001$).

Although the present data show a statistically significant effect from increasing HR (Fig 1), the overall clinical effect may be less significant. An SVV cutoff value of 12% as suggested by other authors[1,10,11] may be applied to our data to predict responders and non-responders to a fluid challenge (Fig 2). At V_t 6 mL/kg, increasing HR from 80 to 110 bpm would cause 2 nonresponders to become responders. In contrast, at V_t 10 mL/kg, nearly all patients become responders without any group effect from increasing HR. The pronounced effect from increasing V_t from 6 mL/kg to 10 mL/kg for the same HR on the number of responders is shown in in Fig 2.

DISCUSSION

Increasing HR from 80 bpm to 110 bpm caused a reduction in SV and a slight but statistically significant increase in SVV in normovolemic, anesthetized patients receiving positive-pressure ventilation. These findings indicated that HR should be considered when using dynamic indices to guide fluid