

The Effect of Changing Arterial Transducer Position on Stroke Volume Measurements Using FloTrac System Version 4.0: A Pilot Experimental Study

OBJECTIVES: We conducted a pilot study using an experimental study protocol to evaluate the measurement error of arterial pulse contour analysis-derived stroke volume due to improper transducer leveling during the passive leg raising test and the impact of such error on the determination of fluid responsiveness.

DESIGN: Prospective observational study.

SETTING: A medical-surgical ICU at a tertiary referral center in Kobe, Japan.

PATIENTS: Consecutive critically ill adult patients using the FloTrac system Version 4.0 (Edwards Lifesciences, Irvine, CA) for hemodynamic monitoring between September 1, 2018, and November 31, 2018.

INTERVENTIONS: None.

MEASUREMENTS AND MAIN RESULTS: Using 20 patients, we estimated the change in the zero-reference level of an arterial transducer during head-down tilting as the vertical distance between the zero-reference levels of the transducer in the 45° semi-recumbent and supine positions. Using the FloTrac system Version 4.0, we recorded the hemodynamic variables every 20 seconds for 180 seconds at each of the following three points: 1) baseline, 2) after the transducer was elevated by the predetermined distance, and 3) after the transducer had returned to baseline. With respect to the predetermined change in the transducer level, a mean value of 18 ± 3 cm resulted in an increase in stroke volume measurement (mean value, 11 mL/beat; 95% CI, 10–13). This value corresponded to 20% (95% CI, 18–23%) of the baseline value 20 seconds after changing the transducer level. A significant correlation was observed between the predetermined change in the transducer level and the increase in the measured stroke volume ($r^2 = 0.58$; $p < 0.001$).

CONCLUSIONS: When using the FloTrac system Version 4.0, a rapid increase in stroke volume was observed after elevating the arterial transducer. Clinicians and researchers are advised that proper leveling of the arterial transducer is necessary in order to accurately assess the change in arterial pulse contour analysis-derived stroke volume during the passive leg raising test.

KEY WORDS: arterial pressure; cardiac output; hemodynamic monitoring; pulse pressure; stroke volume

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To the Editor:

Optimizing fluid status in order to maintain adequate tissue blood flow and prevent fluid overload is a fundamental concept in critical care practice. The passive leg raising (PLR) test is a “virtual” preload challenge in that it relies on changes in the pressure gradient of endogenous fluids, rather than exogenous fluid administration. During this test, a volume of approximately 300 mL of blood pooled in the lower extremities is driven to the central compartment by downward tilting of the head and elevation of the legs. When combined with hemodynamic monitoring, this technique has been validated as a predictor of fluid responsiveness (1–3). Arterial pulse contour analysis (APCA) provides real-time stroke volume (SV) measurements while being both minimally invasive and straightforward (4). Nonetheless, a recent study reported that the level of the arterial transducer may affect the accuracy of APCA-derived SV measurements (5). Hence, there is some concern that APCA may be affected during head-down tilting in the PLR test, as this action changes the zero-reference level of the transducer. If the transducer is not set correctly, then both SV and fluid responsiveness may become subject to measurement errors. Therefore, we conducted a pilot study using an experimental study protocol to evaluate the measurement error of APCA-derived SV caused by improper transducer leveling during the PLR test and the impact of such error on the determination of fluid responsiveness.

MATERIALS AND METHODS

This prospective observational study was conducted in an eight-bed medical-surgical ICU at the Kobe City Medical Center General Hospital, a 760-bed tertiary referral center in Kobe, Japan. Our local institutional review board approved the present study and waived the need for written informed consent (approval number: 18129).

Patients

Eligibility was restricted to consecutive adult patients admitted to the ICU between September 1, 2018, and November 31, 2018, for hemodynamic monitoring using the FloTrac system Version 4.0 (Edwards Lifesciences, Irvine, CA). The exclusion criteria

comprised the presence of the following characteristics: unstable hemodynamic status, arrhythmia, severe atrial regurgitation, over- or under-damping of the arterial waveform, and an intra-aortic balloon pump (6).

FloTrac System

The FloTrac system consists of a FloTrac sensor and a Vigileo monitor (Edwards Lifesciences, Irvine, CA). The FloTrac system algorithm is based on the principle that pulse pressure is proportional to SV and inversely related to vascular tone. Pulse pressure is assessed using the SD of the arterial pressure (σ_{AP}) around the mean arterial pressure (MAP) value that, in turn, is calculated by analyzing the arterial pressure waveform for 20 seconds at 100 times per second and thereby capturing 2,000 data points. Vascular compliance is assessed by a multivariate polynomial equation known as Khi (χ). This equation updates the FloTrac system algorithm on a rolling 60 seconds average, applying data pertaining to pulse rate, MAP, SD of MAP, large vessel compliance as estimated by patient demographics (age, sex, weight, and height), and skewness and kurtosis of the arterial waveform. The value of χ is then multiplied by the SD of the arterial pressure to calculate the SV in milliliters per beat. SV is then multiplied by the pulse rate to determine the cardiac output (CO) in liters per minute (6, 7).

$$SV \text{ (mL/beat)} = \sigma_{AP} \text{ (mm Hg)} \times \chi \text{ (mL/mm Hg)}$$

$$CO \text{ (L/min)} = SV \text{ (mL/beat)} \times \text{pulse rate (beats/min)}$$

SV and CO are continuously measured by a FloTrac sensor and updated on a Vigileo monitor (Edwards Lifesciences, Irvine, CA) every 20 seconds.

Demographic Characteristics

Patient data, including those on age, sex, height, actual body weight, body mass index, reason for ICU admission, time since ICU admission, Acute Physiology and Chronic Health Evaluation II score (8), invasive mechanical ventilation, and catecholamine use, were collected on enrollment.

Study Protocol

All patients received standard care with no research-related interventions. In order to minimize invasion in patients and control variables other than the arterial transducer level, we designed the following study protocol.

The study protocol was performed on patients with a stable hemodynamic status, which was defined as no more than 5% variation in any of heart rate (HR), MAP, and SV for a period of 10 minutes. No changes were made in terms of patient positioning, ventilator settings, sedation, vasoactive drugs, or infusions throughout the study protocol.

Patients maintained a 45° semi-recumbent position while a transducer was moved up and down to simulate the changes in SV caused by the variations in the transducer level associated with head-down tilting. The zero-reference level of the transducer was defined as the phlebostatic axis, which signified the intersection of the fourth intercostal space with the midpoint of the anterior and posterior surfaces of the chest. The change in the zero-reference level of the transducer during head-down tilting was estimated as the vertical distance between the zero-reference levels of the transducer in the 45° semi-recumbent and supine positions

and measured using a tape measure prior to the following sessions (**Fig. 1A**). Hemodynamic variables, including HR, MAP, SV, and CO, were continuously recorded every 20 seconds for 180 seconds during the baseline, experimental, and post-experimental sessions. The first set of measurements was recorded in the 45° semi-recumbent position with the transducer level positioned at the phlebostatic axis (**Fig. 1B**). A second set of measurements was recorded after elevating the transducer from the phlebostatic axis by the predetermined distance in the manner described above (**Fig. 1C**). The transducer was then returned to the baseline level, and a third set of measurements was recorded (**Fig. 1D**).

Statistical Analysis

Continuous variables are presented as mean \pm SD or median (interquartile range) depending on the

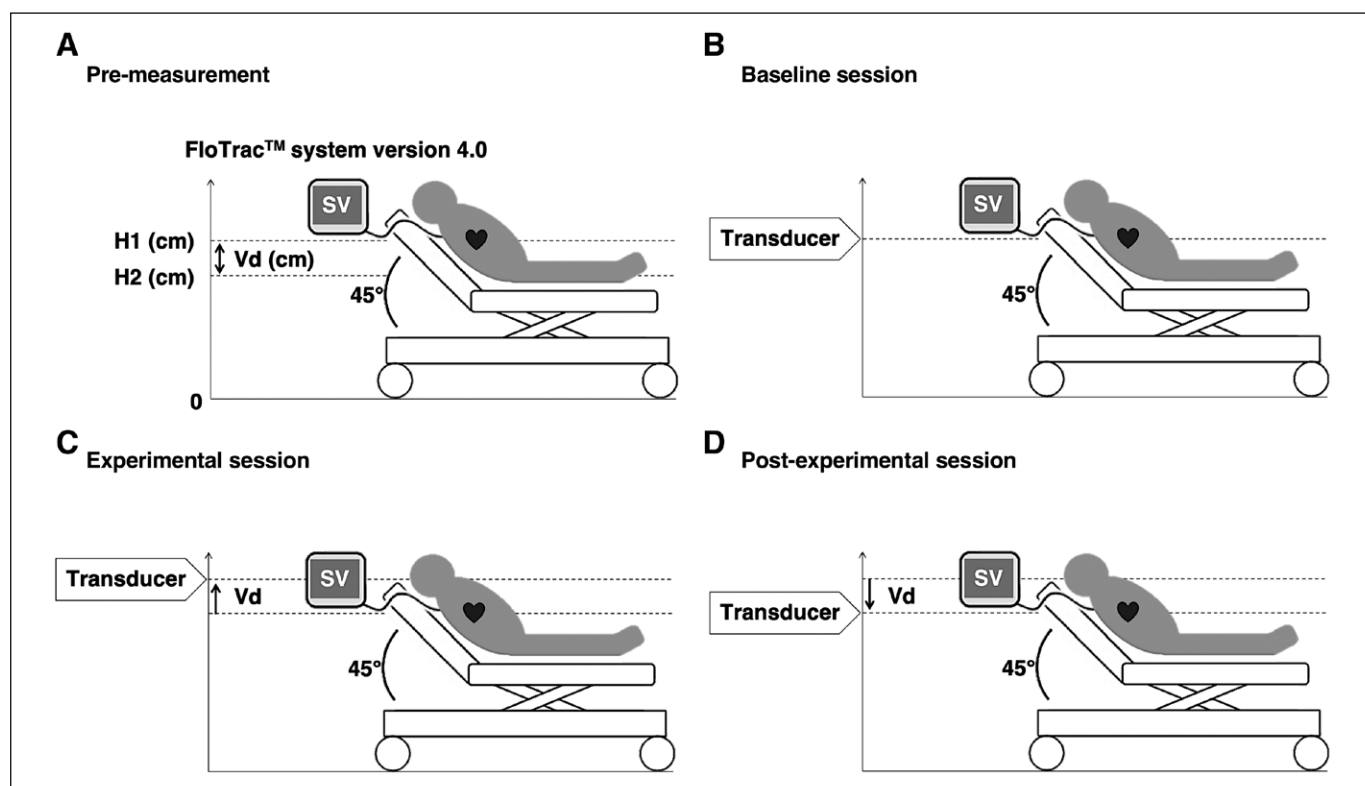


Figure 1. Study protocol. **A**, Pre-measurement. The zero-reference level of the transducer was defined as the phlebostatic axis, which was defined as the intersection of the fourth intercostal space and the midpoint between the anterior and posterior surfaces of the chest. The expected change in the transducer level during head-down tilting in the passive leg raising test was defined as the vertical distance (V_d) between the zero-reference levels of the transducer in the 45° semi-recumbent (H_1) and supine positions (H_2) and measured using a tape measure at the beginning of the study protocol. **B**, Baseline session. Baseline hemodynamic measurements, including heart rate, mean arterial pressure, stroke volume (SV), and cardiac output, were recorded in the 45° semi-recumbent position every 20 s for 180 s. **C**, Experimental session. Immediately after the transducer was elevated to the degree indicated by V_d (cm), the hemodynamic measurements were recorded every 20 s for 180 s. **D**, Post-experimental session. The transducer was returned to the baseline level, and the hemodynamic measurements were recorded again.

distribution. Categorical variables are presented as *n* (%). The hemodynamic variables measured during the three sessions were compared using repeated-measures analysis of variance (ANOVA). Post hoc pairwise comparisons using paired *t* tests were conducted only when repeated-measures ANOVA showed statistically significant results of *p* value of less than 0.05. The mean change and mean percentage change in the hemodynamic variables between baseline and the experimental sessions were calculated and expressed with corresponding 95% CIs. Both the correlations between the predetermined change in transducer level in relation to the phlebostatic axis and the mean changes in MAP and SV between the baseline and the experimental sessions were assessed using a scatter plot, and the strength of the linear correlations was determined using Pearson's coefficient. All data were analyzed using JMP 15 (SAS Institute, Cary, NC).

RESULTS

We enrolled 20 patients in the study (Fig. S1, Supplementary Digital Content, <http://links.lww.com/CCX/A689>). In the collected datasets, there were no missing data. At the time of enrollment, all patients had an arterial pressure catheter inserted into the radial artery and connected to the FloTrac system at the discretion of the physician. The mean patient age, height, actual body weight, and body mass index were 67 ± 13 years, 159 ± 8 cm, 57 ± 17 kg, and 22 ± 3 kg/m², respectively. The vertical distance between the zero-reference levels of the arterial transducer in the 45° semi-recumbent and supine positions was 18 ± 3 cm. The main reasons for ICU admission were post-cardiovascular surgery and sepsis. The study protocol was performed in a median of 22 hours (18–45 hr) after admission to the ICU. During the study protocol, nine and seven patients were treated with catecholamines and invasive mechanical ventilation, respectively (Table 1).

Figure 2 shows the sequential changes in MAP and SV across the three sessions. Compared with the mean values measured in the baseline session, a significant decrease in MAP was observed immediately following elevation of the transducer during the experimental session ($p < 0.001$). At this time, the mean change of MAP (Δ MAP) was -13 mm Hg (95% CI, -14 to -12 mm Hg) (Table 2). SV increased significantly after the first 20 seconds of the experimental session ($p < 0.001$);

TABLE 1.
Baseline Characteristics of Study Patients

| Baseline Characteristics | (<i>n</i> = 20) |
|---|------------------|
| Age, yr | 67 (13) |
| Male sex | 9 (45) |
| Height, cm | 159 (8) |
| Actual body weight, kg | 57 (11) |
| Body mass index, kg/m ² | 22 (3) |
| Body surface area, m ² | 1.58 (0.17) |
| Predetermined change in arterial transducer level ^a , cm | 18 (3) |
| Reason for ICU admission | |
| Post-cardiovascular surgery | 13 (65) |
| Sepsis | 4 (20) |
| Other | 3 (15) |
| Time since ICU admission, hr | 22 (18–45) |
| Acute Physiology and Chronic Health Evaluation II score | 15 (5) |
| Invasive mechanical ventilation | 7 (35) |
| Vasopressor use | 7 (35) |
| Inotrope use | 2 (10) |

^aDefined as the vertical distance between the zero-reference levels of the transducer in the 45° semi-recumbent position and supine position.

Data are presented as mean (SD), median (interquartile range), or *n* (%).

the mean change in SV (Δ SV) was 11 mm Hg (95% CI, 10–13 mm Hg), corresponding to a 20% increase (95% CI, 18–23%) from baseline. With respect to the predetermined change in the transducer level, significant correlations were observed with Δ MAP ($r^2 = 0.82$; $p < 0.001$) and Δ SV ($r^2 = 0.58$; $p < 0.001$), respectively (Fig. 3). The MAP and SV values returned to baseline levels after the transducer position was reset in the post-experimental session. HR did not change significantly throughout the study protocol.

DISCUSSION

In this study, we evaluated the sequential changes in hemodynamic variables after elevation of the arterial transducer. Our main findings were as follows: 1) the estimated change in the zero-reference level during head-down tilting was an average of 18 cm; 2) elevation of the transducer by the predetermined distance resulted in a significant decrease in MAP and an increase in SV; and 3) the significant increase in SV

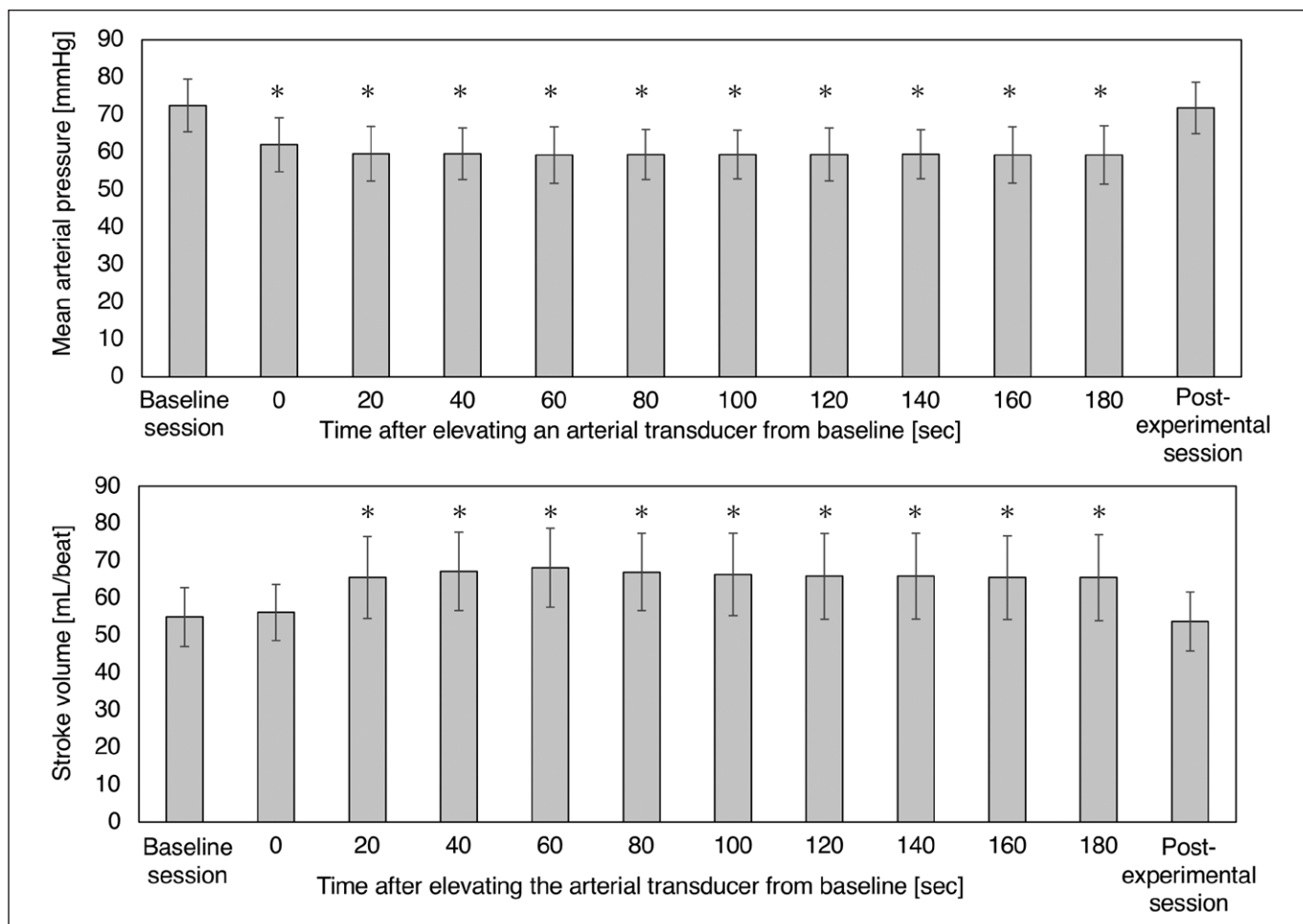


Figure 2. Sequential changes in mean arterial pressure and stroke volume during the experimental session. Data are expressed as the mean \pm SD. * $p < 0.001$ versus the mean value during the baseline session.

was observed as soon as 20 seconds after elevating the transducer and corresponded to 20% of the baseline value. The most important implication of our study is that improper transducer leveling during the PLR test can lead to false positive results in terms of fluid responsiveness, which, in turn, may lead to unnecessary fluid infusion during the course of clinical practice.

When performing the PLR test, the patient is typically placed in a 45° semi-recumbent position at baseline, rather than in a supine position. This is done to maximize the change in the preload induced by raising the legs (3). Importantly, the zero-reference level of the arterial transducer moves downward during head-down tilting. Assuming that the average distance from the hip to the chest is 20% of the height (9), the predicted change in the zero-reference level after head-down tilting would be approximately 14% of the height. In the present study, the mean value of the measured vertical distance between the zero-reference levels of

the transducer in the 45° semi-recumbent and supine positions corresponded to 11% of the mean height of the patients.

The relationship between improper transducer leveling and arterial pressure has been established in prior studies. Theoretically, a 10-cm increase in the level of an arterial transducer should decrease the measured arterial pressure by 7.4 mm Hg (10, 11). This relationship was confirmed by our findings. A previous study also investigated the effect of variable arterial transducer level on APCA-derived SV measurements in critically ill patients, albeit using the pulse contour cardiac output (PiCCO) system. In that study, vertical elevation of the transducer by 20 cm in relation to the zero-reference level brought about a statistically significant increase in SV index, defined as SV divided by the patient's body surface area, of approximately 10% compared with baseline (5). The difference in the degree of change in SV measurements between

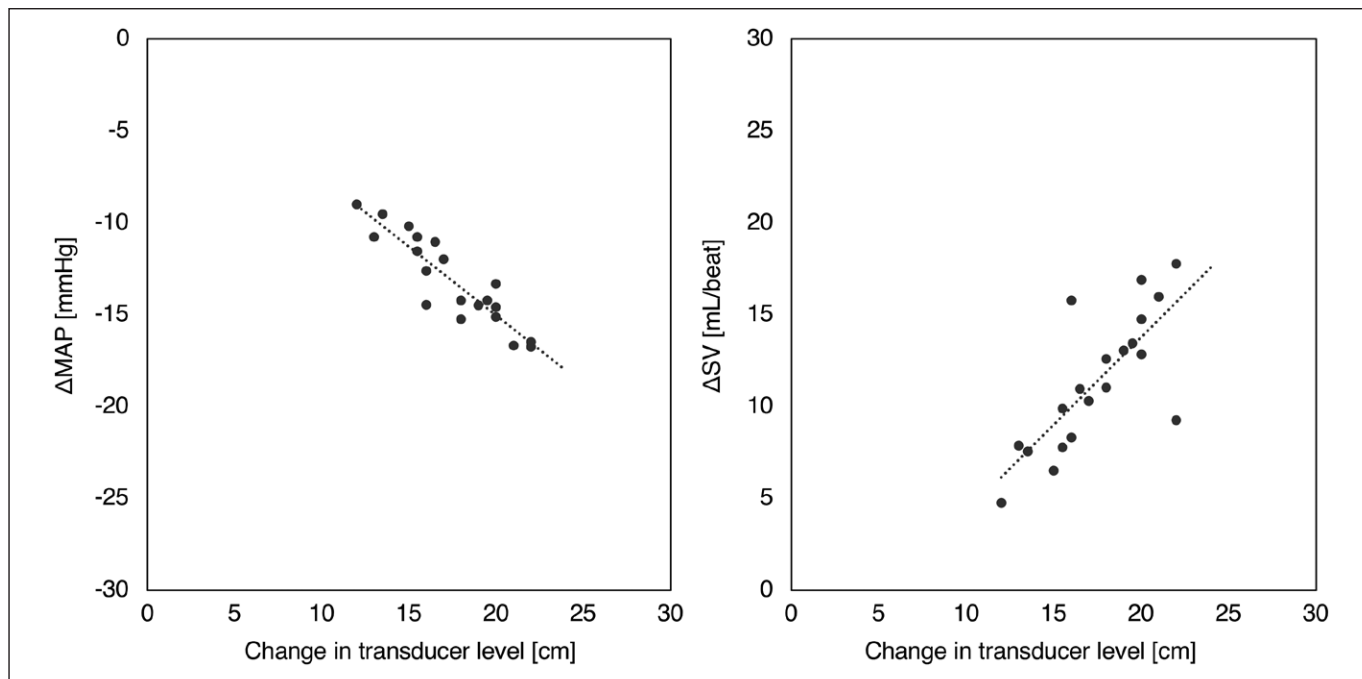


Figure 3. Correlations between changes in transducer level and hemodynamic variables. Δ MAP and Δ SV were defined as the mean changes in mean arterial pressure and stroke volume as measured after elevating the arterial transducer above baseline. The change in the transducer level was predetermined as the vertical distance between the zero-reference levels of the transducer in the 45° semi-recumbent and supine positions.

this study and ours might be due to the physical and hemodynamic status of the patients and differences in the APCA devices used in the studies. Although details regarding the algorithm of the FloTrac system have not been published by the manufacturer due to their proprietary nature, the relationship between false decreases in arterial pressure caused by improper transducer leveling and the resulting SV values can be explained by the fact that according to the APCA algorithm, pulse pressure is proportional to SV and inversely related to vascular tone (7). If the true hemodynamic variables were to be held constant, a false decrease in arterial pressure would not be related to the change in pulse pressure or waveform; instead,

they would lead to a false decrease in vascular tone and a consequent overestimation of SV. Furthermore, an upgraded feature of FloTrac System 4.0 may play an important role in the effect of improper transducer leveling on the measurement of SV. In Version 4.0 of the FloTrac system, a novel factor called “Khi-fast” was introduced. The purpose of Khi-fast is to regulate the rapid arterial pressure changes that occur during vasopressor administration. Khi-fast is assessed every 20 seconds and is inversely affected by arterial pressure (6). The addition of this factor to Version 4.0 of the FloTrac system explains the rapid false increase in SV in our study, where the change in SV was observed as early as 20 seconds after elevation of the arterial

TABLE 2.
Changes in Hemodynamic variables After Elevation of Arterial Transducer Level

| Hemodynamic Variables | Baseline Session | Experiment Session | Mean Change (95% CI) | Mean Percentage Change (95% CI) |
|-------------------------------|------------------|--------------------|----------------------|---------------------------------|
| Mean arterial pressure, mm Hg | 73 ± 7 | 60 ± 7 | -13 (-14 to -12) | -18 (-20 to -16) |
| Stroke volume, mL/beat | 55 ± 8 | 66 ± 11 | 11 (10-13) | 20 (18-23) |
| Cardiac output, L/min | 4.1 ± 0.7 | 4.9 ± 1.0 | 0.9 (0.7-1.0) | 21 (18-23) |
| Heart rate, beats/min | 78 ± 11 | 78 ± 12 | 0 (-1 to 2) | 0 (-1 to 2) |

Data are presented as mean ± sd unless otherwise noted.

transducer. Since the maximal change in SV during the PLR test is usually expected to occur within 30–120 seconds (12, 13), a rapid change in SV due to improper transducer leveling may affect the interpretation of the PLR test. In addition, the 20% increase in SV observed in our study was above the cutoff value for fluid responsiveness as defined by the existing literatures (1–4, 12, 13). To avoid measurement error due to the change in the zero-reference level after head-down tilting, we suggest that the arterial transducer should be attached on the patient's body (i.e., upper arm or lateral side of the chest) temporarily so that its position referring the phlebostatic axis is not changed during the PLR test (14).

This study had several limitations. First, we did not perform “actual” PLR test including head-down tilting. Although comparison of hemodynamic variables during the “actual” PLR test measured using FloTrac system with and without adjustment of the transducer level seems to be a reasonable and appropriate design for our clinical questions, this design also has the following limitations: changing the patient's position may introduce confounding factors related to physiologic changes derived from changes in the posture rather than those in the transducer level. In addition, this design requires two consecutive experiments in the same patient with and without adjustment of the transducer level; therefore, changes in hemodynamic variables over time may also be a confounding factor. Based on these considerations, we finally decided to adopt the experimental study design described in the methods rather than performing the “actual” PLR test. Second, this was a pilot study that enrolled only a small number of patients. Nonetheless, the significant and clinically important increases in measured SV brought about by changes in the transducer level support the clinical significance of our findings. Third, the relatively small body sizes of our patients may limit the generalizability of our findings to patients of different body sizes. Fourth, we enrolled hemodynamically stable patients in order to control for variables other than the arterial transducer level. Therefore, the impact of inappropriate transducer leveling on the reliability of PLR testing in situations where infusion is required was not addressed in this study. Finally, the changes in hemodynamic variables were assessed using FloTrac system Version 4.0. As such, those using other versions of the FloTrac or PiCCO systems may observe different

results. Nonetheless, proper transducer leveling is essential for accurate measurement of hemodynamic variables when using APCA devices.

CONCLUSIONS

A rapid increase in SV measurement using the FloTrac system Version 4.0 was observed following elevation of the arterial transducer. Clinicians and researchers are advised that proper leveling of the arterial transducer is necessary in order to accurately assess the change in APCA-derived SV during the PLR test.

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Drs. Ito and Kawakami mainly participated in study conception, design, and execution. Dr. Ito was responsible for data collection and cleaning. Drs. Ito and Morimoto performed the statistical analysis. Dr. Ito wrote the article and produced the tables and figures. All authors participated in data interpretation, revised the article for important intellectual content, and approved the final version of the report.

The authors have disclosed that they do not have any potential conflicts of interest.

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