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ORIGINAL RESEARCH

The effects of patient positioning on hemodynamic parameters

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Abstract

Elevation of legs increases stroke volume (SV) as the blood moves into the intrathoracic compartment. However, the degree of effects caused by the increase in blood remains controversial as it only has temporary effect. As for hemodynamically unstable patients, special positioning and hemodynamic monitoring are essential. We investigated the effective posture to increase SV and the relationships between the hemodynamic parameters and the changes in the SV by adjusting the upper and the lower body angles. This repeated-measures cross-sectional study included 42 normal subjects to study the relationship between SV and hemodynamic variables. Hemodynamic parameters, such as SV, end-tidal carbon dioxide ($ETCO_2$), and anterior-posterior diameter of the inferior vena cava (IVC) and internal jugular vein (IJV) were repeatedly measured with position changes. The changes in the variables were measured in the upper body elevations of 60° and 30°, the supine position, and the lower body elevations of 30° and 60°. The mean SV at the 30° lower body elevation showed the highest value among the SV values which was measured in two minutes after each position change. The SV and the SV value change showed the strongest positive correlation with the IJV's anterior-posterior diameter and the IJV value change (odds ratio = 5.617 and 15.277; $p = 0.004$ and <0.001, respectively) while the $ETCO_2$ and the IVC diameter showed no relation to the SV. The SV showed the highest value at the lower body elevation of 30°. The scale of IJV diameter and the change were predictable factors of the SV and its changes. These findings might have clinical implications in the positioning and the hemodynamic monitoring of SV.

Keywords

Stroke volume; Ultrasonography; Jugular veins; Supine position

1. Introduction

Leg elevation in the event of circulatory collapse is a rescue maneuver employed by first responders, which induces straight passive elevation above the cardiac level [1, 2]. The leg elevation can increase venous blood volume and cardiac preload by moving venous blood from the leg to the intrathoracic compartment [2, 3]. According to a previous study, about 150 mL of blood per leg is transfused into the central circulation by this maneuver [4]. These changes produce an autotransfusion effect that increases the venous return to the heart and enhances stroke volume (SV) or cardiac output (CO) [1, 2].

The position most effective to elicit the largest increase in SV has yet to be determined. The Trendelenburg position lead to a surge of SV instantaneously within one minute but resulted in a reduction of the SV over two minutes while the effect of leg elevation maintained over 2 minutes [2]. The authors proposed that the leg elevation could be better than the Trendelenburg position for hypovolemic patients. In previous studies of leg elevation reported that the angle of leg elevation showed various effects on SV increase. The leg elevations of 30° and 60° induced CO increase (0.3 L/min) over 5 minutes in several studies [5, 6] but the SV generated by the leg elevations of 20° and 60° was similar to the SV in the supine position in another study [7]. Additionally, some authors reported that after seven minutes, leg elevations of 60° did not affect autotransfusion [8] or that the hemodynamic effect on leg elevations of 45° was not sustained over one minute [1].

Also, hemodynamic monitoring is essential to measure these SV changes according to the legs elevation, especially in unstable patient. Handy substitutes for monitoring SV are required in the clinical field. Hemodynamic variables such as blood pressure, End-tidal CO_2 ($ETCO_2$), and the diameter changes in the inferior vena cava (IVC) and internal jugular vein (IJV) may reflect SV changes. Conventionally, blood pressure has been used to monitor hemodynamic stability. A decrease in CO was significantly associated with decreases in $ETCO_2$ [9]. The fact that the size of central veins, such as IVC and IJV influence SV was identified through the induction of dehydration [10]. These substitutes might help monitor hemodynamic change of SV that can be induced due to position change.

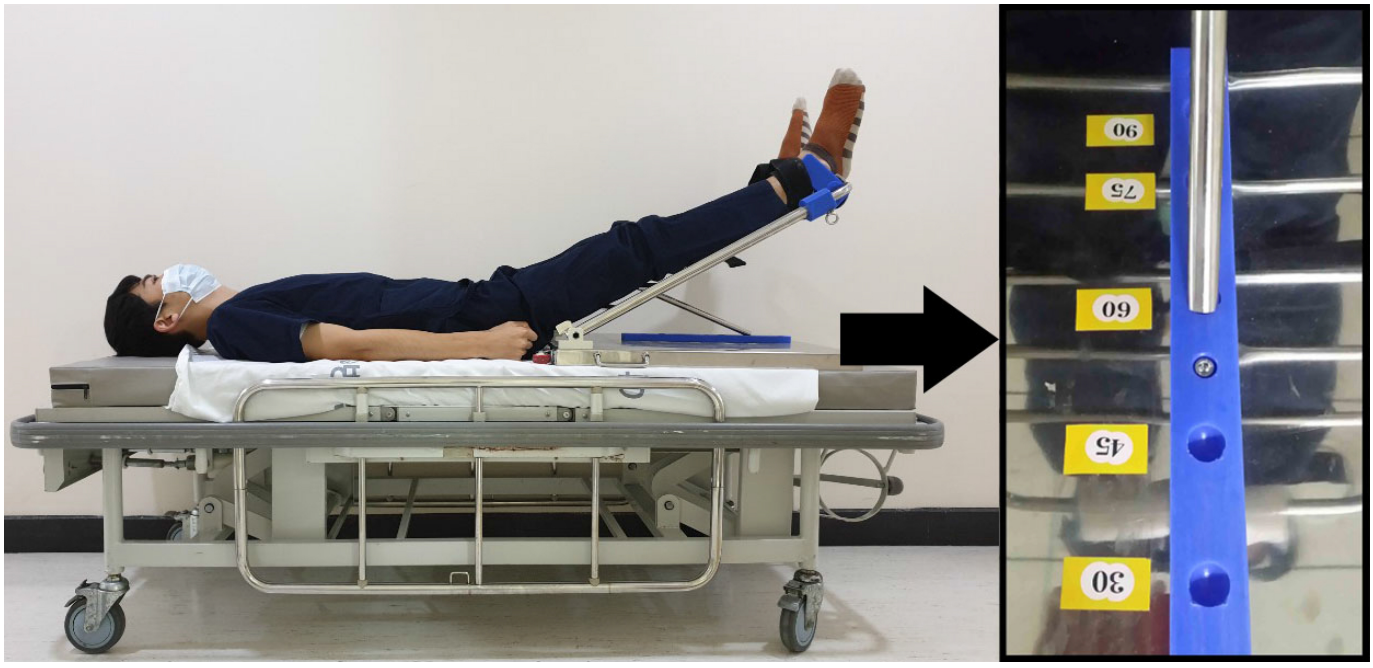

FIGURE 1. Lower body 30° elevation using a position changer. Jae Hoon Lee owns the copyright.

Therefore, we aimed to determine the most efficient posture for hemodynamic stability and to evaluate the correlation between hemodynamic parameters (blood pressure, sizes of the IVC and IJV, and $ETCO_2$) and the SV in each different position.

2. Subjects and methods

2.1 Study population

This repeated-measures cross-sectional study included 42 male volunteers, each of whom provided written informed consent to participate. The ethics committee of our tertiary care university teaching hospital approved the study protocol (DAUHIRB-16-161). Healthy subjects who were more than 18 years-old and well hydrated were included. The exclusion criteria were subjects with failure to identify a transthoracic echo window on ultrasound examination in a depressed or obese chest wall, and difficulty performing postural changes.

2.2 Repeated measurements using a position changer

The correlation between the SV and other hemodynamic variables were investigated in the following positions: at the upper body at 60° and 30°, in the supine position, at the lower body at 30° and 60°. We used a position changer that can set the proper angle as 30° or 60° made by the author who had patent rights for the device (Fig. 1). All hemodynamic variables, including systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), time velocity integral (TVI) of the left ventricular outflow tract (LVOT), dimensions of the IVC and IJV, and $ETCO_2$, were repeatedly measured in each position.

2.3 Noninvasive hemodynamic monitoring and measurement

SV was obtained as mean value of 3 beats around maximum value because the minimum SV value could dynamically change according to a subjects' thoracic movement or breath-holding time or the investigation's hand motion. The mean maximum SV (SVmax) was calculated from the LVOT diameter and LVOT TVI [11]. The LVOT diameter was measured on a long-axis parasternal view and LVOT TVI was measured with pulsed Doppler placing sampling volume as parallel as possible to the LVOT in a 5-chamber apical view using a 4.5-MHz phased-array transducer. The echocardiographic variables including SVmax, IJVmax, and IVCmax were examined within about 12 minutes per a posture. The SVmax during two respiratory cycles was measured in two minutes after each position change and maximum diameters of the right IJV (IJVmax) and IVC (IVCmax) as hemodynamic factors for comparing with SVmax were sequentially measured. The IJVmax was visualized at the level of the cricoid cartilage using a 7–12-MHz linear-array transducer. The IJVmax and IVCmax were recorded as anteroposterior diameters in the right IJV and around 2 cm from the orifice of the right heart chamber in the B-mode or the M-mode respectively. The values of SVmax, IVCmax, and IJVmax were extracted from the parameters measured by the ACUSON X300 Ultrasound System (Siemens Medical Solutions USA Inc., Mountain View, CA, USA). An identical sonographer measured the SVmax and other hemodynamic parameters by using transthoracic echocardiography to reduce bias and an assistant checked vital parameters and $ETCO_2$ while measurements of the SV and central veins were taken. Portable $ETCO_2$ monitoring via nasal cannula (Philips® Intellivue MP2, Suresnes, France) was performed after instructing the subjects not to breathe too deep or swallow. The SBP and DBP was obtained in each position by using an automated blood pressure cuff and mean arterial

pressure (MAP) was calculated. The $ETCO_2$, SBP, DBP, and HR values, as well as the ultrasound measurements, were repeatedly examined in each position.

2.4 Statistical analysis

The data were reported as means ± SD. A correlation between an independent variable and the SV according to position change was analyzed by using the generalized estimating equation method as a repeated measures analysis model because 5 consecutive measurements of parameters at each position were repeatedly conducted. Continuous variables that were in different units were replaced with the standardized values to minimize the effect of scale differences. Additionally, the differences of the values in the present posture from the values in prevenient posture (delta value) were again analyzed by the generalized estimating equation as multivariable analysis for observing the change in hemodynamic variables according to the SV change. Consecutively and repeatedly measured values according to position change were compensated by statistical working correlation (autoregression). The correlation between hemodynamic variables and the SV was expressed as a coefficient (β) and the precision was defined using P values and confidence intervals. All the calculations were made using a standard software (SPSS v23 for Windows, IBM Corp, Armonk, NY, USA) with a statistical significance set at $p < 0.05$.

3. Results

The baseline characteristics of the healthy subjects were shown in Table 1. Each variable was repeatedly evaluated in each of the 42 subjects for the angled positions (Table 2). The SVmax value measured in two minutes after each position change showed the highest values in the 30° lower body elevations (Fig. 1) among all positions. Compared to the SVmax and the IJVmax in 30° upper body elevations, the SVmax and the IJVmax in supine position revealed statistically significant differences, respectively ($p = 0.039$; $p < 0.001$, Fig. 2). SVmax, COmax, IJVmax, and $ETCO_2$ in the 60° upper body elevation showed the lowest value of values in the other positions but, MAP and IVCmax were not. MAP and IVCmax showed irrelevant pattern when compared with SVmax and were not related with SVmax ($p = 0.262$ and 0.7). However, importantly, the IJVmax had changed along with movement in the SVmax by position changes ($p = 0.01$, Table 2).

TABLE 1. Subjects' characteristics.

Age (mean ± SD, years)	24 ± 3.15
Sex (male/female, no. of subjects)	42/0
Weight (mean ± SD, kg)	72.1 ± 9.34
Height (mean ± SD, cm)	175.3 ± 4.82
BMI (mean ± SD, kg/m^2)	23.4 ± 2.69
BSA (mean ± SD, m^2)	1.9 ± 0.13

BMI, body mass index; BSA, body surface area.

Compared with MAP, $ETCO_2$, IVCmax, and positions, IJVmax was the factor that was most correlated with SVmax despite the spontaneous breathing (β coefficient, 1.726; OR = 5.617; $p = 0.004$; Table 3). $ETCO_2$ were not significantly correlated with the SVmax induced by the position change ($p = 0.075$; Table 3). Similar correlation was also observed in between change values of IJVmax and SVmax. The change value of IJVmax by position change was significantly correlated with the change value in SVmax compared to other parameters (β coefficient, 2.726; OR = 15.277; $p < 0.001$; Table 4). The significant relationship between SVmax and IJVmax although repeated measurements in the same subject were not considered, was presented in Fig. 3 ($p < 0.001$).

4. Discussions

The SVmax was the highest in the 30° leg elevation position and was increased with borderline significance compared to the supine position. In the positions elevated upper body, the more elevation of the upper body, the more decrease in SVmax was observed. In addition, the IJVmax and its change in value were similarly followed with the SVmax and its change in value. However, MAP, $ETCO_2$, and IVCmax were not associated with the SVmax. Therefore, the IJVmax was a more reliable predictor of the SVmax than the IVCmax or $ETCO_2$.

Many studies have compared the SV between leg elevation and the supine position but comparing the SV in various positions has not been frequently attempted. Although a study reported that all SVs were similar in leg elevations of 20° and 60° and the supine position [7], our results showed the slight SV differences at leg elevations of 30° and 60° and in the supine position but was not statistically significant. The differences between the previous study and our study may be attributed to the measurement method. In our study, transthoracic ultrasonography that measures the SV on the closest chest wall from the LVOT was used, whereas in the previous study, an ultrasonic CO monitor that measure the SV through the suprasternal approach on the farther point from the LVOT was used. Moreover, the measurement timing after positioning might have influenced the SVmax. The hemodynamic effect by the leg elevation was temporary [1, 2, 8] and it is not known how long the autotransfusion effect from the leg elevation could be maintained. A study reported that the SV increase by the leg elevation was only observed in the first 20 seconds after the leg elevation and disappeared after seven minutes [8]. Another study revealed that the hemodynamic effects of the leg elevation reached their maximum within one minute and diminished rapidly thereafter [1, 12]. In contrast, a review article concluded that the effect of the leg elevation seemed to sustain CO increase (6%) for 2 to 10 minutes [2]. Additionally, a recent study reported that the hemodynamic effect of 30° to 45° leg elevations was temporary and neutralized completely after 15 minutes but the elevations reduced hypotensive events and increased the mean arterial pressure and central venous pressure before and after the procedures [13].

The decrease in SBP or MAP have alerted physicians to the risk of various types of shock. However, increase in the SV were not associated with the change in SBP and the pulse

TABLE 2. Parameters measured repeatedly in each position and the relationship with stroke volume at the upper body at 60° and 30°, in the supine position, at the lower body at 30° and 60°.

	60° elevation in upper body	30° elevation in upper body	Supine	30° elevation in lower body	60° elevation in lower body	*p* value*
$ETCO_2$ (mmHg)	37.7 ± 3.05	38.6 ± 3.31	38.5 ± 2.31	38.6 ± 2.67	38.7 ± 2.89	0.065
SBP (mmHg)	118.6 ± 10.66	118.2 ± 10.03	122.8 ± 11.36	115.9 ± 12.21	116.5 ± 12.31	0.362
DBP (mmHg)	69.9 ± 10.11	66.5 ± 8.1	65 ± 7.78	63 ± 11.38	63.3 ± 9.95	0.335
MAP (mmHg)	86.1 ± 2.97	83.7 ± 7.72	84.3 ± 7.88	80.6 ± 10.92	81 ± 9.77	0.262
Heart rate (beats/min)	67.2 ± 8	66.3 ± 8.37	67.6 ± 8.47	64 ± 7.97	62.8 ± 9.06	0.250
LVOT TVI max (cm)	13.6 ± 2.95	14.2 ± 2.96	15.6 ± 2.88	17.1 ± 3.49	16.3 ± 3.1	<0.001
SV max (mL)	44.5 ± 10.12	46.5 ± 10.39	51.3 ± 10.36	56.3 ± 13.27	53.8 ± 12.08	
CO max (L/min)	3 ± 0.67	3.1 ± 0.68	3.5 ± 0.77	3.6 ± 0.89	3.4 ± 0.9	<0.001
IVC max (cm)	1.78 ± 0.31	1.8 ± 0.71	1.6 ± 0.32	1.8 ± 0.27	1.7 ± 0.29	0.700
IJV max (cm)	0.3 ± 0.14	0.5 ± 0.2	0.9 ± 0.22	0.8 ± 0.2	0.8 ± 0.23	0.010

$ETCO_2$, end tidal carbon dioxide; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; LVOT TVI, left ventricular outflow tract time velocity integrals; SV, stroke volume; IVC max, maximum diameter of inferior vena cava; IJV max, maximum diameter of internal jugular vein.

* *The relationship between an independent variable and SV according to position change was analyzed by using the generalized estimating equation method as a repeated measures analysis.*

TABLE 3. Correlation of standardized hemodynamic parameters with stroke volume followed by position change.

Parameters*	β coefficient	Standard error	t	Odds ratio	$p^{\dagger}$	95% confidence interval (CI)
MAP	–0.912	0.7528	1.467	0.402	0.226	0.092–1.757
$ETCO_2$	1.595	0.8974	3.16	4.929	0.075	0.849–28.617
IVC diameter	0.309	0.4506	0.471	1.362	0.493	0.563–3.295
IJV diameter	1.726	0.604	8.163	5.617	0.004	1.719–18.349

MAP, mean arterial pressure; $ETCO_2$, end tidal carbon dioxide; IVC, inferior vena cava; IJV, internal jugular vein.

* *All hemodynamic parameters were standardized.*

† *p value analyzed by supposing generalized estimating equation.*

TABLE 4. Correlation between change in hemodynamic parameters and change in stroke volume followed by inducing the differences of the values in the present posture from the values in prevenient posture.

Parameters*	β coefficient	Standard error	t	Odds ratio	$p^{\dagger}$	95% CI
Intercept	0.5	2.0482	0.06	1.65	0.807	0.03–91.365
Position	0.505	0.585	0.746	1.658	0.388	0.527–5.217
Δ MAP	–1.364	0.6464	4.454	0.256	0.035	0.072–0.907
Δ $ETCO_2$	1.155	0.8086	2.04	3.173	0.153	0.651–15.481
Δ IVC	0.7	0.6845	1.046	2.014	0.306	0.526–7.704
Δ IJV	2.726	0.7526	13.122	15.277	<0.001	3.495–66.787

MAP, mean arterial pressure; $ETCO_2$, end tidal carbon dioxide; IVC, inferior vena cava; IJV, internal jugular vein.

* *All hemodynamic parameters were standardized.*

† *p value analyzed by supposing generalized estimating equation.*

pressure [14]. Furthermore, a rapid or gradual decrease in the SV induced transient decrease in MAP but the MAP was shortly recovered in a previous study [15]. The SV and MAP including SBP and DBP were also not correlated in our result as well.

The relationship between the $ETCO_2$ and SV has been suspected in previous studies. The change in $ETCO_2$ during IVC clamping and unclamping predicted changes in the CO [16] and decreases in the $ETCO_2$ detected decreases in CO but not hypovolemia [9]. Nevertheless, the correlation between the absolute $ETCO_2$ and CO values was not significant because the absolute $ETCO_2$ value may be influenced by many other

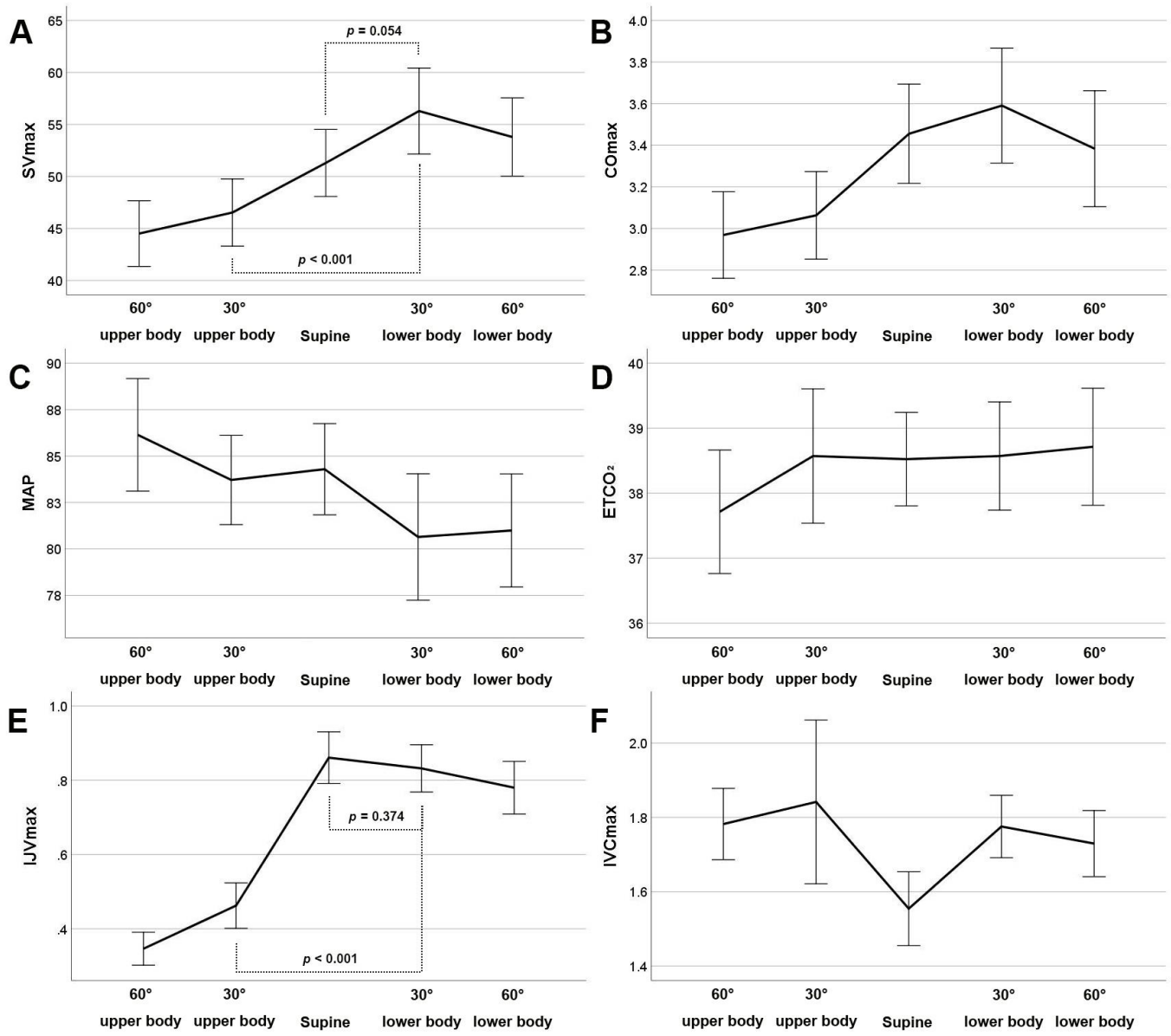

FIGURE 2. Hemodynamic variables at each position. (A) Mean maximum stroke volume (SVmax). (B) End-tidal carbon dioxide ($ETCO_2$). (C) Diastolic blood pressure (DBP). (D) Systolic blood pressure (SBP). (E) Maximum diameter of the internal jugular vein diameter (IJVmax). (F) Maximum diameter of the inferior vena cava (IVCmax).

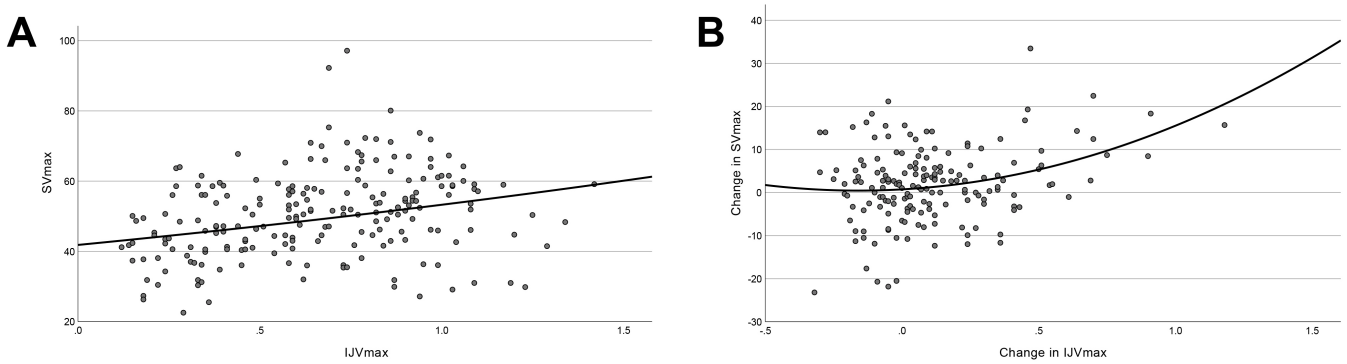

FIGURE 3. The correlation between SVmax and IJVmax. (A) The relationship between SVmax and IJVmax ($p < 0.001$). (B) The relationship between change values of SVmax and IJVmax from a posture to the next posture ($p < 0.001$).

factors [17]. The conditions causing prominent change in $ETCO_2$ to respond to SV change might be required for a clinical use of $ETCO_2$. First, hypovolemic state causes SV to decrease and generates greater change in SV by respiratory change [18]. The SV decrease or the change caused by hypovolemia may induce bigger change in $ETCO_2$. Second, $ETCO_2$ monitoring are available in lung parenchyma without lesions because acute lung injury can lead to decreased $ETCO_2$ that failed to reflect SV [19]. Third, deep respiration can make increases in $ETCO_2$. Also, abrupt increase and change in the $ETCO_2$ may result from large tidal volumes that increased the SV and the SV variation [20]. Lastly, atrial fibrillation, vasodilatation, and intraabdominal pressure must be identified because those state could decrease the SV [21] and $ETCO_2$. Changes in $ETCO_2$ were not significantly related with the changes in the SV according to our study. The indistinctive change in $ETCO_2$ regarding the change in SV may result from euvolemic status and shallow respiration of our subjects. Additionally an inaccurate measurement via nasal cannula could have been an issue.

Increases in the preload can enhance the SV according to the Frank-Starling curve but the SV increase caused by the preload increase in euvolemic or hypervolemic states are less [22]. Measuring the change of the preload is a method for monitoring the SV. Size changes in the IVC and IJV (collapsibility or distensibility) were significantly related to decreased preload [23–25]. However, in the spontaneously breathing subjects of our study, the SV value was more associated with the IJV diameter than the IVC diameter. The IVC diameter may be difficult to reflect preload or SV because it can be compressed by the descent of the diaphragm [26] and influenced by intra-abdominal hypertension [27]. The IVCmax can be significantly smaller or larger by the increased intra-thoracic or the intra-abdominal pressure but the IJVmax may not vary significantly. The dynamic change value of IJV diameter was also correlated with the change value of SV in our study. The IJV diameter and the change value may be a potential predictor of SV change although change in the SV may be relatively small in normal subjects who were not severely dehydrated.

This study had several limitations. First, because the subjects were healthy, it is necessary to prove the clinical utility of our results in a clinical setting. The present observations in the volunteers would be partially due to different hemodynamic response between the normal and abnormal hemodynamic condition. Second, there may be a gender specific bias although SV appeared to be slightly greater in females versus males and SV change according position change showed no specific gender differences in a previous study [28]. Third, a passive leg raising or fluid responsiveness test was not applied in the present study. Finally, the SV was not measured by the referred technique, which is thermodilution technique, and more precise measurement of the SV should be considered.

5. Conclusions

The leg elevation at 30° had the largest effect on the SV two minutes after each position change. Elevated upper body significantly lowered the SV and must be avoided in hemodynamically unstable patients to increase the SV. The IJV diameter and its change in value were significantly correlated with the SV and the SV changes generated by position change. The scale of IJV diameter and the change were predictable factors of the SV and its changes while the IVC diameter was not. The monitoring of IJV could be a convenient and practical tool for observing the changes in SV. Overall, these findings from the present study might have clinical implications in the positioning and the hemodynamic monitoring of SV with respect to hemodynamically unstable patients.

AUTHOR CONTRIBUTIONS

JaeHL conceived, drafted, planned, investigated and managed the manuscript concerning all aspects of the study. JuHL performed the statistical analysis and interpretation. JaeHL and JuHL analyzed the data, reviewed and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The Dong-A University Hospital served as the main institutional review board (IRB) approved the study with the following entry code (DAUHIRB-16-161). All subjects received written informed consent to participate.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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