Masimo® Plethysmograph Variability Index as a Tool for Assessment of Fluid Responsiveness in Elective Major Abdominal Surgeries

Ahmed M. Essam¹, Mohamed Z. Ali¹*, Mohamed A. Maher¹, Ali M. Mokhtar², Sohila H. Omar¹, Hossam H. El-Sabae¹ and Mohamed H. Hafez²

¹Department of Anesthesiology, Surgical Intensive Care and Pain Management, Theodor Bilharz Research Institute, Ministry of High Education and Scientific Research, Giza, Egypt.
²Department of Anesthesiology, Surgical Intensive Care and Pain Management, Faculty of Medicine, Cairo University, Cairo, Egypt.

Authors’ contributions

This work was carried out in collaboration between all authors. Author AME contributed to the study design, anesthetized and followed up patients, helped in collecting data and revised the medical charts. Author MZA contributed to the study design, supervised anaesthesia and follow-up of patients, helped perform the statistical analysis and drafted the manuscript. Authors MAM and AMM contributed to the study design and revised the manuscript. Author SHO helped collect data, revise the medical charts, perform the statistical analysis and draft the manuscript. Author MHH helped collecting data, revised the medical charts and drafted the manuscript. Author HHES helped collecting data and revised the medical charts. All authors read and approved the final manuscript.

ABSTRACT

Background: Maximizing the stroke volume (SV) as measured by Trans-oesophageal Doppler (TED) optimizes preload, & is a goal-directed fluid therapy technique that has been used in a variety of clinical settings. Masimo® Plethysmograph variability Index (PVI) is a reliable, safe &
This study was designed to determine whether PVI, measured using finger co-oximetry, is an efficient predictor of fluid responsiveness in low-risk patients undergoing elective major abdominal surgery.

**Subjects and Methods:** 60 ASA I-II patients of either sex, 25-60 years old, undergoing major abdominal surgery were enrolled in this study. A Masimo® Radical-7 Pulse Co-Oximeter probe & a Cardio Q TED probe were applied to each patient. In all patients, a fluid bolus of 500 ml of 130/0.4 tetrastarch colloid solution was administered rapidly via pressurized IV infusion. Maintenance & deficits were calculated routinely. If the SV decreased by 10%, a 250-mL bolus of colloid was given via fast infusion. Patients' demography, TED-derived measurements: (SV & Flow Time corrected (FTc)), Masimo®-derived measurements: (PVI & PI), HR and MAP were all collected and statistically analyzed. Measurements were done at five minutes post-induction T1, Ten minutes after volume expansion (500 ml colloid) T2, If the SV decreased by 10%, (guided by TED) T3, Then 250 ml colloid is given. Ten minutes after a 250-ml colloid bolus T4.

**Results:** A significant difference was found in FTc, SV, PI & PVI in T1 vs. T2 & T3 vs. T4 (P=0.001). There was a significant difference in PI & PVI between responders & non-responders for the 1st bolus (P<0.05) and in SV & PVI in subsequent boluses (P<0.01). There was no significant difference between percent changes of SV and PVI at T3 & T4.

**Conclusions:** Plethysmograph Variability Index (PVI) measured by Masimo® Co-Oximeter is an efficient predictor of fluid responsiveness as SV measured by TED in low risk patients undergoing elective major surgery.

**Keywords:** MASIMO®; MASIMO® plethysmograph variability index; trans-oesophageal doppler.

**ABBREVIATIONS**

<table>
<thead>
<tr>
<th>ABP</th>
<th>arterial blood pressure</th>
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<tbody>
<tr>
<td>CI</td>
<td>cardiac index</td>
</tr>
<tr>
<td>CO</td>
<td>cardiac output</td>
</tr>
<tr>
<td>CVP</td>
<td>central venous pressure</td>
</tr>
<tr>
<td>FTc</td>
<td>systolic flow time corrected for heart rate</td>
</tr>
<tr>
<td>HR</td>
<td>heart rate</td>
</tr>
<tr>
<td>MAP</td>
<td>mean arterial pressure</td>
</tr>
<tr>
<td>PAC</td>
<td>pulmonary artery catheters</td>
</tr>
<tr>
<td>PI</td>
<td>perfusion index</td>
</tr>
<tr>
<td>PICCO</td>
<td>Pulse-induced continuous cardiac output</td>
</tr>
<tr>
<td>PPV</td>
<td>pulse pressure variation</td>
</tr>
<tr>
<td>PVI</td>
<td>plethysmograph variability Index</td>
</tr>
<tr>
<td>SV</td>
<td>stroke volume variation</td>
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<tr>
<td>SVV</td>
<td>stroke volume variation</td>
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<tr>
<td>TED</td>
<td>trans-oesophageal Doppler monitoring</td>
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<tr>
<td>VTIAo</td>
<td>aortic velocity-time integral</td>
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**1. INTRODUCTION**

Adequate assessment of the intravascular volume is primarily important to maintain cardiac output thus avoiding hypovolemia and tissue hypoxemia [1]. However, unnecessary fluid administration is associated with higher rates of morbidity and mortality [2]. Perioperatively, it is mandatory to optimize the haemodynamics in order to improve patient’s outcome and reduce mortality [1]. During major abdominal surgery, this optimization guarantees faster recovery of gut function [3] reduces length of hospital stay [4], rate of complications [3] and incidence of postoperative critical care unit admission [5].

Haemodynamic optimization has been enabled with a variety of devices such as pulmonary artery catheter, arterial line, or the oesophageal Doppler. Maximizing the stroke volume (SV), as measured by oesophageal Doppler, optimizes preload, and is a goal-directed fluid therapy technique that has been used in a variety of clinical settings in anaesthesia, critical care, and emergency medicine [2].

Dynamic variables (indices evaluating the response to a cyclic preload variation) provide a better forecast of fluid responsiveness [6]. During mechanical ventilation, arterial pressure waveform variation has been shown to be a dependable indicator of fluid responsiveness in patients with a stable heart rhythm [7]. Additionally, increases in stroke volume variation (SVV), [8] systolic pressure variation [9] and pulse pressure variation [10] have all been shown to be indicators of responsiveness to fluid administration.

Trans-oesophageal Doppler (TED) ultrasonography of the descending aorta could be a useful monitoring device that allows a continuous estimation of CO and facilitates the
assessment of preload, afterload, and myocardial contractility by calculating advanced haemodynamic variables. Although TED can be considered as a safe technology, yet, some complications were reported including buccal cavity minor traumata [11], transient vagal stimulation during probe insertion [12], inadvertent gastric tube removal during probe removal [13], epistaxis [14], and tracheal or bronchial probe misplacement [15, 16].

Masimo® Plethysmograph variability Index (PVI) was recently introduced as a reliable, safe, and noninvasive tool to guide fluid management. The pulse oximeter plethysmographic waveform differs from the arterial pressure waveform by measuring volume rather than pressure changes in both arteries and veins. The ‘pleth variability index’ (PVI) is an automated measure of the dynamic change in the perfusion index (PI) that occurs during a respiratory cycle. The perfusion index (PI) is the ratio between non-pulsatile and pulsatile blood flow through the peripheral capillary bed.

PVI has been shown to help clinicians to predict fluid responsiveness in mechanically ventilated patients under general anaesthesia during surgery and in the ICU [17, 18]. It has also been shown to help clinicians to improve fluid management and decrease lactate levels in contrast to standard care [19]. PVI demonstrates high accuracy in discriminating fluid responders from non-responders, it provides a unique chance to better fluid volume management through optimization of cardiac performance and organ perfusion.

We designed this novel adaptive clinical trial to determine whether PVI, measured using finger co-oximetry probe, is an efficient predictor of fluid responsiveness in low-risk patients undergoing elective major abdominal surgery during steady-state conditions before surgery, and during dynamic intra-operative conditions. The primary outcome will be Masimo® Pleth indices based on TED derived measurements. Secondary outcome parameters will be the evaluation of heart rate and mean arterial blood pressure.

2. MATERIALS AND METHODS

From September 2013 through June 2015, this prospective, adaptive clinical study was conducted in the Anaesthesia and Surgical Intensive Care Department, at Theodor Bilharz Research Institute after approval by local research ethics’ committee and the study was registered in Pan African Clinical Trial Registry (www.pactr.org) with identification number for the registry PACTR201512001373777. Written informed consents from sixty low-risk adult patients of either sex (ASA physical status I–II), aged 25–60 years, undergoing major abdominal or pelvic surgery with expected duration more than 120 minutes and blood loss more than 1000 ml (e.g. radical cystectomy, radical prostatectomy, gastrectomy, pancreatectomy, spleenectomy). Patients suffering from oesophageal pathology, uncontrolled diabetes mellitus or hypertension, irregular heart rhythm, advanced cardiac condition, severe metabolic, neurological, endocrinial, hepatic or renal impairment, metastatic malignancies, significant coagulopathy (INR > 1.5 or PTT >1.5 times normal) BMI<18.5 or >24.9 Kg/m², pregnant women, laparoscopic surgical plan and patients’ refusal to participate were excluded.

Premedication was given in the form of midazolam (0.05 mg/kg) intravenously half an hour before operation. Anaesthesia was then induced with intravenous propofol (2 mg/kg), fentanyl (3 µg/kg), and atracurium (0.5 mg/kg). General anaesthesia was then maintained by isoflurane 1-1.5 MAC in 100% oxygen in addition to supplemental doses of atracurium according to the nerve stimulator and fentanyl (1 µg/kg/hour). Mechanical ventilation was performed keeping a tidal volume of 6-8 ml/kg with the respiratory rate adjusted to maintain P_{ET}CO_2 between 35 and 40 mm Hg. All patients were monitored for electrocardiogram, noninvasive arterial blood pressure, peripheral oxygen saturation, end-tidal carbon dioxide tension, core temperature, peripheral nerve stimulator, hourly urinary output and Bispectral index BIS (for monitoring depth of anaesthesia).

After induction of anaesthesia, a Masimo® Radical-7 Pulse CO-Oximeter probe (Masimo® Corporation, Irvine, CA) was applied to the index finger of each patient. A Cardio Q Oesophageal Doppler (TED) probe (Deltex Medical, Chichester, UK) was inserted orally into the oesophagus and the position was adjusted to attain the maximal SV (approximately 35–40 cm from the teeth). All test fluids were administered via a fluid warmer, and a forced-air warming blanket were used to maintain a constant oesophageal temperature.
In all patients, intravenous fluid management was as follows; a fluid bolus of 500 ml of 130/0.4 tetrastarch colloid solution (Voluven™; Fresenius Kabi, Runcorn, Cheshire, UK) was administered rapidly into a peripheral vein via a pressurized IV infusion pump, maintenance was calculated for the first 10 kg; 4 ml/kg/h, second 10 kg; 2 ml/kg/h then next Kgs 1 ml/kg/h as lactated Ringer's solution, deficit was calculated as maintenance multiplied by fasting hours as lactated Ringer's solution, half of it in the first hour of the operation and the rest in the following 2 hours divided equally. Third space loss was calculated as 6-10 ml/kg/h as lactated Ringer's solution. If the SV decreased by 10% as measured by TED, a 250-mL bolus of 130/0.4 tetrastarch colloid solution was given via fast infusion with a 50-mL syringe. Blood transfusion was given based on hemoglobin level (less than 7 g/dL).

Patient's demography, TED-derived measurements: (Stroke Volume (SV) & Flow Time corrected (FT\textsubscript{c})), Masimo\textsuperscript{®} Radical-7 Pulse Co-Oximeter derived measurements: (Plethysmographic Variability Index (PVI) & Perfusion Index (PI)), Heart rate (HR) and Mean arterial blood pressure (MAP) were all collected and statistically analyzed.

Measurements were taken as follows; T1 at five minutes post-induction (baseline parameters). T2, ten minutes after volume expansion by 500 ml 130/0.4 tetrastarch colloid solution rapid infusion. T1 & T2 were considered as a steady-state measurements before start of surgery and the patients were classified into responders and non-responders according to whether they had an increase in TED-measured SV of >10% or not at T2. T3, if TED-measured SV decreased > 10%, T3 measurement will be recorded then 250 ml colloid is infused. T4, ten minutes after giving 250 ml colloid. T3 & T4 were considered as intra-operative measurement and the patients were classified into responders and non-responders according to whether they had an increase in TED-measured SV of > 10% or not at T4.

2.1 Statistical Analysis

Based on previous study (20), a sample size was calculated on change in PVI (40%) occurred immediately before and 10 minutes after volume expansion. Assuming a two-sided type I error of 0.05 and a power of 0.80, a sample size of 60 patients would be required. G Power 3.1 program was used in sample size calculation. Results are expressed as mean±standard deviation or number (%). Comparison between variables measured at baseline (T1) and at T2 or T3 and T4 in the same group was performed using either paired t test or Wilcoxon Signed Ranks test whenever it was appropriate. Comparison between variables in responder and non-responder groups was performed using either unpaired t test or Mann Whitney test whenever it was appropriate. Percent change is calculated from the equation: \( \frac{\text{T4-T3}}{\text{T3}} \times 100 \). The data were considered significant if \( P \) value was \( \leq 0.05 \) and highly significant if \( P \) value was < 0.01. Statistical analysis was performed with the aid of the SPSS computer program (version 16 windows).

3. RESULTS

Demographic data of the patients is shown in Table 1 as regards age, gender, weight, BMI, ASA physical status class.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Patients (n= 60)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>53.17±7.21</td>
</tr>
<tr>
<td>Gender (F-M)</td>
<td>4 (6.7%)-56 (93.3%)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>82.67±4.39</td>
</tr>
<tr>
<td>BMI (kg/m\textsuperscript{2})</td>
<td>23.45±1.14</td>
</tr>
<tr>
<td>ASA (I-II)</td>
<td>50 (83.3%)-10 (16.7%)</td>
</tr>
</tbody>
</table>

Data are expressed as mean±SD or number (%)

Haemodynamic data (Heart rate, Mean arterial blood pressure), Trans-oesophageal Doppler data (Flow Time corrected FT\textsubscript{c}, Stroke Volume SV), Masimo\textsuperscript{®} data (Plethysmograph Variability Index PVI, Perfusion Index PI) were compared during the procedure before (T1) and after (T2) 1\textsuperscript{st} bolus (500 ml 130/0.4 tetrastarch colloid solution) within 10 minutes before skin incision as shown in Table 2.

As regards the Stroke volume in T1 and T2, the patients are classified into Responders and Non-Responders according to an increase in Stroke volume > 10% in (T2). The study shows that there are 40 Responders and 20 Non-Responders. The comparison between mean values of haemodynamic variables (FT\textsubscript{c}, SV, PVI, PI, MAP and HR) in responder and non-responder patients in T1 is shown in Table 3.

The table shows that there is no significant difference in the baseline FT\textsubscript{c}, SV, HR, MAP between responders and non-responders with
**P-value > 0.05** while there is a significant difference between responders and non-responders in PI and PVI with **P-value < 0.05**.

Then, haemodynamic data (Heart rate, Mean arterial blood pressure), Trans-oesophageal Doppler data (FT$_c$, Stroke Volume SV), Masimo® data (PVI, PI) were compared if SV decreased > 10% from the baseline value (T3) and after administration 250 ml 130/0.4 tetraastarch colloid solution (subsequent boluses) (T4). Six patients were excluded due to haemodynamic stability and no need for further colloid bolus while 12 patients needed more than 250 ml 130/0.4 tetraastarch colloid solution (2 boluses 250 ml Voluven™) and blood. So, total number of occasions that required subsequent boluses was 66. The comparison between T3 and T4 is shown in Table 4.

There is high significant difference between T3 and T4 in FT$_c$, SV, PVI, PI, MAP with **P-value=0.001**.while HR shows no significant difference between T3 and T4 as **P-value > 0.05**.

As regards to the Stroke volume in T3 and T4, the patients were classified into responders and non-responders according to an increase in Stroke volume > 10% (at T4). The study shows that there are 56 responders and 10 non-responders. The Comparison between mean values of haemodynamic variables in responder and non-responder patients in T3 is shown in Table 5.

**Table 2. Haemodynamic variables before and after a 500-ml 130/0.4 tetraastarch colloid solution bolus given during steady state before the start of surgery (skin incision) in the studied patients**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>T1 (n= 60)</th>
<th>T2 (n= 60)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT$_c$ (mSec.)</td>
<td>362.83±23.71</td>
<td>391.07±23.27</td>
<td>0.001**</td>
</tr>
<tr>
<td>SV (ml/beat)</td>
<td>69.37±19.96</td>
<td>81.33±25.70</td>
<td>0.001**</td>
</tr>
<tr>
<td>PI</td>
<td>1.84±1.06</td>
<td>2.43±1.14</td>
<td>0.001**</td>
</tr>
<tr>
<td>PVI</td>
<td>16.97±3.47</td>
<td>10.20±1.72</td>
<td>0.001**</td>
</tr>
<tr>
<td>HR (beat/min.)</td>
<td>80.30±12.40</td>
<td>79.80±11.38</td>
<td>0.779</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>93.83±13.70</td>
<td>97.97±14.02</td>
<td>0.028*</td>
</tr>
</tbody>
</table>

Data are expressed as mean±SD; NS= P> 0.05= not significant; *P< 0.05= significant; **P< 0.01= highly significant

**Table 3. Comparison between mean values of haemodynamic variables in responder and non-responder patients for first bolus at T2**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Non-responders (n= 20) 33.3%</th>
<th>Responders (n= 40) 66.7%</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT$_c$ (mSec.)</td>
<td>364.2±29.11</td>
<td>362.15±20.89</td>
<td>0.755</td>
</tr>
<tr>
<td>SV (ml/beat)</td>
<td>67.6±21.69</td>
<td>70.25±19.27</td>
<td>0.632</td>
</tr>
<tr>
<td>PI</td>
<td>2.29±1.16</td>
<td>1.62±0.94</td>
<td>0.019*</td>
</tr>
<tr>
<td>PVI</td>
<td>15.8±1.77</td>
<td>17.55±3.96</td>
<td>0.021*</td>
</tr>
<tr>
<td>HR (beat/min.)</td>
<td>77.8±16.03</td>
<td>81.55±10.14</td>
<td>0.273</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>96.1±10.88</td>
<td>92.7±14.92</td>
<td>0.369</td>
</tr>
</tbody>
</table>

Data are expressed as mean±SD; NS= P> 0.05= not significant; *P< 0.05= significant

**Table 4. Haemodynamic variables T3 vs. T4 in the studied patients**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>T3 (n= 66)</th>
<th>T4 (n= 66)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT$_c$ (mSec.)</td>
<td>325.64±31.36</td>
<td>386.67±23.18</td>
<td>0.001**</td>
</tr>
<tr>
<td>SV (ml/beat)</td>
<td>58.94±19.56</td>
<td>79.76±20.33</td>
<td>0.001**</td>
</tr>
<tr>
<td>PI</td>
<td>1.29±0.88</td>
<td>1.69±0.80</td>
<td>0.001**</td>
</tr>
<tr>
<td>PVI</td>
<td>17.12±2.42</td>
<td>11.91±4.79</td>
<td>0.001**</td>
</tr>
<tr>
<td>HR (beat/min.)</td>
<td>84.91±15.94</td>
<td>83.33±11.37</td>
<td>0.213</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>78.88±13.54</td>
<td>93.36±12.84</td>
<td>0.001**</td>
</tr>
</tbody>
</table>

Data are expressed as mean±SD; NS= P> 0.05= not significant; **P< 0.01= highly significant

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There is a high significant difference between responder and non-responder patients in both SV and PVI with P-value < 0.01 while FTc, PI, HR, MAP show no significant difference between responders and non-responders with P-value > 0.05.

There was no significant difference between percent changes of SV and PVI at T3 and T4 as regard cases who received the subsequent boluses as the percent change of SV is 40.04±24.70 while that of PVI is 35.28±13.57 with P-value =0.196. This non-significant difference is also shown in Fig. 1.

ROC analysis demonstrated insignificant predictive ability of an increase in SV for PVI at the steady state. Area under the curve was 0.623 (95% confidence interval [CI], 0.530–0.710; P-value 0.155). A baseline PVI cutoff value of 11 had 96.67% sensitivity and 33.33% specificity for predicting >10% SV increase; positive predictive Value (PPV) was 59.2% while negative predictive value (NPV) was 90.9% (Fig. 2).

ROC analysis demonstrated significant predictive ability of an increase in SV for PVI at the intraoperative state. Area under the curve was 0.877 (95% confidence interval [CI], 0.809 – 0.928; P-value 0.0001). A baseline PVI cutoff value of 11 had 91.04% sensitivity and 81.82% specificity for predicting >10% SV increase; positive predictive Value (PPV) was 83.6% while negative predictive value (NPV) was 90.3% (Fig. 3).

4. DISCUSSION

Assessment of the adequacy of the intravascular volume is of the prime importance to maintain cardiac output and thus, avoid hypovolemia and tissue hypoxia [1]. Fluid optimization is reflected by faster recovery of gut function [3], reduced incidence of intensive care unit (ICU) admission [5], shortened length of hospital stay [4] and reduced rate of complication [3].

The semi-invasive TED is a useful device in optimizing intravascular volume. However it has reported complications e.g. buccal trauma, transient vagal stimulation and probe misplacement. So we investigated the ability of PVI measured by Masimo® as a non-invasive, safe and reliable tool to predict fluid responsiveness and to optimize intravascular volume.

**Table 5. Comparison between mean values of haemodynamic variables in responder and non-responder patients for subsequent boluses**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Non-responders (n= 10)15%</th>
<th>Responders (n= 56) 85%</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FTc (mSec.)</td>
<td>310.80±24.09</td>
<td>328.29±31.94</td>
<td>0.079</td>
</tr>
<tr>
<td>SV (ml/beat)</td>
<td>78.40±19.63</td>
<td>55.46±17.55</td>
<td>0.002**</td>
</tr>
<tr>
<td>PI</td>
<td>1.75±1.25</td>
<td>1.20±0.78</td>
<td>0.197</td>
</tr>
<tr>
<td>PVI</td>
<td>15.2±1.55</td>
<td>17.18±2.52</td>
<td>0.004**</td>
</tr>
<tr>
<td>HR (beat/min.)</td>
<td>85.00±14.89</td>
<td>84.89±16.24</td>
<td>0.802</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>81.00±9.71</td>
<td>78.50±14.15</td>
<td>0.351</td>
</tr>
</tbody>
</table>

*Data are expressed as mean±SD. NS= P > 0.05= not significant; **P< 0.01= highly significant*

![Fig. 1. Percent change in the two techniques](image-url)
To reach this finding, sixty ASA I-II patients scheduled for elective major surgeries were enrolled in this study. Prior to surgery, patients were classified according to their response to steady state bolus (1st bolus= 500 cc 6% hydroxyethyl starch 130/0.4; Voluven™), into 20 non-responders and 40 responders in the form of SV increase more than 10% measured by TED. During surgery, patients were administered a bolus of 250 cc Voluven™ whenever SV decreases more than 10% as measured by TED. Patients were then classified into 56 responders and 10 non-responders after intra-operative boluses according to an increase in SV more than 10% relative to the prior TED reading.

We found that the baseline value of PVI was higher in responders compared with non-responders after steady state bolus and during surgery after intraoperative boluses. This finding illustrates that Plethysmograph Variability Index (PVI) measured using finger co-oximeter probe is an efficient predictor of fluid responsiveness as stroke volume (SV) measured by semi-invasive oesophageal Doppler (TED) in low risk patients undergoing elective major surgery.

We also observed that steady state bolus (1st bolus) and intra-operative bolus (2nd bolus and subsequent boluses) produced a significant increase in flow time corrected (FTc), SV, Perfusion index (PI), mean arterial pressure (MAP) and significant decrease in Plethysmograph Variability Index (PVI) while heart rate (HR) showed no change. This observation offers PVI as an efficient index not only to predict fluid responsiveness but also to guide intraoperative fluid optimization.

Cannesson et al. [21] met our results, they showed that PVI can predict fluid responsiveness in cardiac patients undergoing coronary artery bypass grafting (CABG). They induced volume expansion and fluid responsiveness was defined as an increase in CI > or =15%. They also observed a significant increase in MAP, central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP) and significant decrease in PVI. Unlike our findings, PI showed no change with volume expansion. This may be due to different recording times, which was three minutes after volume expansion while our study readings were recorded ten minutes after volume expansion. Also they studied different type of patients i.e. ischemic and surgery i.e. CABG.

Similarly, Fu et al. [22] showed the ability of PVI measured by Masimo® Radical 7 monitor and SVV measured by Vigileo™ system in predicting fluid responsiveness during resection of primary retroperitoneal tumors in Chinese patients. They also reported that these two parameters are more efficient than cardiac index (CI), CVP and MAP as surgical stress factors did not affect SVV and PVI as a predictor of fluid responsiveness. Their study detected a significant positive linear correlation between PVI at baseline and percent changes in Stroke volume index (SVI) induced by intravascular volume expansion similar to our study, which emphasized the obvious percent change in SV and PVI after volume expansion.

Our results agree with Bahlmann et al. [23] who showed that there was no significant difference between SV measured by TED and PVI measured by Masimo® Radical 7 monitor in fluid optimization during open abdominal surgery.
Also, Forget et al. [24] designed a comparative study between goal directed PVI group with control group guided by MAP and CVP. They found that patients with PVI guided fluid management showed less crystalloid administration intra-operatively than the control group and there was a decrease in lactate level during and after major abdominal surgery.

Hood et al. [25] suggested that PVI measured at the finger may be an accurate predictor of fluid responsiveness during preoperative conditions and to lesser but still significant extent in dynamic intra-operative conditions. They claimed that factors affecting afterload could affect intraoperative PVI readings. These factors include their use of epidural anaesthesia, which induced sympathetic block, degree of muscle relaxation, long duration of application of the oximeter probe on the same finger and change in contractility due to changes in circulating catecholamine level. Due to our balanced anesthetic technique and continuous monitoring of muscle relaxation most of these factors do not apply on our study. Their study also showed that conventional haemodynamic variables as HR, arterial blood pressure (ABP), CVP are not reliable predictors of fluid responsiveness. Yu et al. [26] compared PVI guided group with MAP guided control group undergoing major abdominal surgeries. They found that the total amount of intraoperative crystalloid infusion was significantly lower in PVI group than control group. While they did not find significant difference between the two groups in the volume of colloid infusion, incidence of hypotension, lactate level in the 2nd and 3rd hours of surgery may be because the type of surgeries were mostly laparoscopic (18 patients out of 30).

Zimmermann et al. [27] compared Stroke Volume Variation (SVV) and PVI with CVP to predict the response of stroke volume index (SVI) in mechanically ventilated patients undergoing major abdominal surgeries. They showed that the baseline of SVV and PVI were correlated significantly with changes in SVI while CVP was not. Also, this study concluded that there is no significant difference between SVV and PVI values.

Also, Haas et al. [28] stated that PVI measured by Masimo® is as accurate as SVV Measured by PICCO in predicting fluid responsiveness in patients after CABAG. However cardiac rhythm changes after CPB and left or right ventricular impairment may decrease the predictive value of both SVV and PVI.

Loupec et al. [18] compared PVI derived from Masimo® and Cardiac output (CO) estimated by Echocardiography with variation in blood pressure before and after fluid challenge to detect the accuracy of PVI as a predictor of fluid responsiveness in critically ill patients. They did a fluid challenge with 500 ml HES and patients were classified into responders and non-responders according to increase in cardiac output ≥ 15%. Similar to our results, they found that the baseline values of PVI were higher in responder group in comparison with non-responder group.

Siswojo et al. [29] investigated the ability of PVI as non invasive tool versus SVI measured by TED in predicting intraoperative fluid responsiveness in mechanically ventilated patients undergoing non-cardiac surgery by giving volume expansion after induction of general anaesthesia. Patients were classified into 17 responders and 19 non-responders according to increase in SVI > 10%. Their results were similar to ours confirming that the baseline value of PVI is significantly different between responders and non-responders. They suggested that the non-responders may lay on the flat portion of Frank-Starling curve so any increase in the preload is not associated with increase in SVI or there was a proportion of patients who had an increase in SVI but less than 10%.

Feissel et al. [30] suggested that PVI is a feasible method to predict fluid responsiveness in early phase septic shock patients in the emergency department by comparing PVI with aortic velocity-time integral (VTIao) using transthoracic echocardiography. The patients were classified into 16 responders and 15 non-responders according to increase in VTIao > 15%. This study similarly shows that the baseline PVI before volume expansion is higher in responders than non-responders.

On the contrary to our and the previous findings that prove the efficiency of PVI as a predictor to the fluid responsiveness and a valuable guidance for intravascular volume optimization, some studies done on cardiac patients who underwent cardiac surgeries failed to show such value. This difference may be attributed to the fact that patients after cardiac surgeries frequently suffer some complications including cardiac dysrrhythmias and ventricular function impairment, both of which are considered limitations to the predictive value of PVI.
Broch et al. [31] suggested that PVI is not a reliable indicator of the fluid responsiveness with sufficient accuracy in stable cardiac patients undergoing CABAG. They claimed that accuracy of PVI to predict fluid responsiveness was improved in patients with higher PI values.

Also Maughan et al. [32] performed a study on postoperative cardiac surgery patients. They observed the inability of PVI to predict fluid responsiveness either in intubated patients or in spontaneously breathing patients when it was compared with cardiac index (CI) measured by pulmonary artery catheter (PAC). Also they included spontaneously breathing patients in which tidal volume is changing affecting the predictive value of PVI.

In addition, Haiseth et al. [33] showed the limited ability of pressure based dynamic variables as PVI & SVV to guide fluid therapy in patients with aortic stenosis preoperatively but SVV can be a moderate predictor of fluid responsiveness postoperatively while PVI is a poor predictor. This discrepancy may be due to the decrease in cardiac output in aortic stenosis patients, which may be reflected on tissue perfusion decreasing the ability of PVI to predict fluid responsiveness.

Also, Keller et al. [34] found that PVI is a weak predictor of fluid responsiveness in spontaneously breathing patients as PVI is not able to distinguish between changes in PI induced by respiration and changes induced by other causes. PVI is considered one of the dynamic predictors for fluid responsiveness so the patient should be mechanically ventilated.

Other limitations of PVI as a predictor of fluid responsiveness are unstable cardiac rhythm, right or left ventricular impairment and hypothermia which lead to poor tissue perfusion and spontaneously breathing patients.

5. CONCLUSION

From this adaptive study we can conclude that Plethysmographic Variability Index (PVI) measured by Masimo® Radical 7 monitor using finger Co-Oximetry probe is an efficient predictor of fluid responsiveness as efficient as stroke volume measured by semi-invasive oesophageal Doppler in low risk patients undergoing elective major abdominal surgery.

CONSENTS

All authors declare that written informed consents were obtained from all patients before participation & publication of this study. The study was primarily approved by local research ethics’ committee. A copy of the written consent is available for review by the Editorial office/Chief Editor/Editorial Board members of this journal.

ETHICAL APPROVAL

All authors hereby declare that this study was conducted in the Anaesthesia and Surgical Intensive Care Department, at Theodor Bilharz Research Institute after approval by local research ethics’ committee and the study was registered in Pan African Clinical Trial Registry (www.pactr.org) with identification number for the registry PACTR201512001373777.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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