Comparison of arterial pressure and plethysmographic waveform-based dynamic preload variables in assessing fluid responsiveness and dynamic arterial tone in patients undergoing major hepatic resection


Department of Anesthesiology, University of Groningen, University Medical Center Groningen, Hanzeplein 1, PO Box 30 001, Groningen 9700 RB, The Netherlands
* Corresponding author. E-mail: j.j.vos@umcg.nl

Editor’s key points

- Changes in blood pressure and central venous pressure are unreliable indicators of optimal intravascular volume status.
- Both (semi-invasive) arterial pressure and plethysmographic waveform analyses can be used to estimate fluid responsiveness.
- The ratio of pressure-to-stroke volume variation reflects arterial elastance (vascular tone).
- Knowledge of arterial elastance may be helpful to guide vasopressor or fluid therapy.

Background. Dynamic preload variables to predict fluid responsiveness are based either on the arterial pressure waveform (APW) or on the plethysmographic waveform (PW). We compared the ability of APW-based variations in stroke volume (SVV) and pulse pressure (PPV) and of PW-based plethysmographic variability index (PVI) to predict fluid responsiveness and to track fluid changes in patients undergoing major hepatic resection. Furthermore, we assessed whether the PPV/SVV ratio, as a measure of dynamic arterial elastance (Eadyn), could predict a reduction in norepinephrine requirement after fluid administration.

Methods. Thirty patients received i.v. fluid (15 ml kg\(^{-1}\) in 30 min) after hepatic resection and were considered responders when stroke volume index (SVI) increased ≥20% after fluid administration. SVV and SVI were measured by the FloTrac-Vigileo\textsuperscript{©} device, and PVI was measured by the Masimo Radical 7 pulse co-oximeter\textsuperscript{©}.

Results. The areas under a receiver operating characteristic curve for SVV, PPV, and PVI were 0.81, 0.77, and 0.78, respectively. In responders, all dynamic variables, except PVI, decreased after fluid administration. Eadyn predicted a reduced norepinephrine requirement (AUC = 0.81).

Conclusions. In patients undergoing major hepatic resection, both APW- and PW-based dynamic preload variables predict fluid responsiveness (preload) to a similar extent. Most variables (except PVI) also tracked fluid changes. Eadyn, as a measure of arterial elastance (afterload), might be helpful to distinguish the origin of hypotension.

Clinical trial registration. ClinicalTrials.gov, NCT01060683.

Keywords: arterial pressure; circulation; fluid therapy; measurement techniques; monitoring; cardiopulmonary

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Adequate assessment of volume status in patients undergoing major surgery is imperative to prevent both inadvertent hypovolaemia and fluid overload, both of which are associated with increased morbidity and mortality.\textsuperscript{1} Static indicators of cardiac preload, such as central venous pressure (CVP) and pulmonary artery occlusion pressure, have repeatedly been shown to be inaccurate measures of volume status and are unable to predict fluid responsiveness reliably.\textsuperscript{2–4} Instead, current research has focused on the use of dynamic preload variables for the prediction of fluid responsiveness to predict whether fluid administration will increase cardiac output (CO).\textsuperscript{5–7} These dynamic preload variables are based on the heart–lung interaction and are derived from circulatory fluctuations secondary to changes in intrathoracic pressure during volume-controlled mechanical ventilation.

Dynamic preload variables are either pressure-based (e.g. pulse pressure variation; PPV), flow-based (e.g. stroke volume variation; SVV), or volume-based (plethysmographic variability index; PVI) and are obtained either from the arterial pressure waveform (APW; semi-invasive) or from the plethysmographic waveform (PW; non-invasive).
A comparable ability to predict fluid responsiveness
between APW- and PW-derived PPV has recently been
demonstrated in critically ill patients. A recent report showed the reduced accuracy of dynamic preload variables to predict fluid responsiveness under ‘normal’ clinical conditions in the intra-operative setting and another report illustrated that in <30% of intra-operative cases, all conditions are met for the use of dynamic preload variables to predict fluid responsiveness.

In addition, even when preload dependency is assessed correctly, an assessment of cardiac afterload (i.e. arterial tone) might be additionally useful for guidance of appropriate vasopressor or i.v. fluid therapy to provide adequate organ perfusion. Recently, the ratio between the pressure-based PPV and the flow-based SVV has been proposed to reflect dynamic arterial elastance (Eadyn; i.e. a surrogate of cardiac afterload). By this means it becomes possible to differentiate arterial vasodilatation from hypovolaemia as a cause of hypotension.

The aim of this study was to assess an estimate of both cardiac preload and dynamic arterial elastance in a clinical setting of patients undergoing major hepatic resection. Therefore, we provide a comparison of the ability of the most commonly used APW- and PW-based dynamic preload variables to predict fluid responsiveness and to track its changes after fluid administration. We hypothesized that PVI was as good as the arterial pressure-based dynamic preload variables in predicting and tracking changes induced by fluid administration. In addition, we assessed whether Eadyn was able to predict changes in arterial tone in response to changes in norepinephrine requirement after fluid administration.

Methods

We evaluated 30 patients involved in a previous study investigating the accuracy of continuous non-invasive measurement of haemoglobin concentration using the Masimo Radical 7 device (Masimo, Inc., Irvine, CA, USA), which additionally records the PVI from the same waveform. In these patients, the other CO-based data were obtained as part of routine clinical monitoring. Each patient served as his/her own control. The original study was approved by the local ethics committee (Ref: 2009/174, University Medical Centre Groningen, The Netherlands) and was registered at clinicaltrials.gov (NCT01060683).

All eligible ASA I–III patients undergoing major hepatic resection were included into this study after written informed consent had been obtained. Patients with intra-operatively diagnosed incurable disease, cardiac dysrhythmia, or patients who required additional i.v. fluids to maintain haemodynamic stability before the fluid bolus was administered (the latter was required for the aim of the original study) were excluded. All patients received a standardized general anaesthesia after placement of a thoracic epidural catheter, as described previously.

A radial artery was cannulated (20 G) for continuous monitoring of arterial blood pressure and for blood gas analysis and the right internal jugular vein was cannulated (7F triple lumen) for monitoring CVP and drug infusion. Both pressure transducers were, after zeroing, adjusted to the height of the right atrium.

Norepinephrine was administered when mean arterial pressure (MAP) decreased to <60 mm Hg and adjusted to keep MAP between 60 and 80 mm Hg. Patients were mechanically ventilated with a mixture of O2 (FiO2 = 0.30–0.35), air, and isoflurane in a volume-controlled mode with tidal volumes of 8 ml kg⁻¹ lean body mass, with PEEP 5 cm H2O. The respiratory rate was adjusted to maintain end-tidal CO2 pressure between 4.5 and 5.5 kPa. Respiratory settings were not changed throughout the measurements.

Immediately after completion of hepatic resection, all patients received a fluid bolus of 15 ml kg⁻¹ within 30 min. As per original protocol, patients were allocated to receive a fluid bolus of either colloids (n=15) or crystalloids (n=15). Patients were considered fluid responsive when stroke volume index (SVI, stroke volume divided by body surface area for normalization) increased by at least 20% after fluid administration compared with the value before administration of fluid. SVI was measured continuously using the FloTrac-Vigileo device (Edwards Lifesciences, Irvine, CA, USA).

Dynamic preload variables

The FloTrac-Vigileo® device (software V03.02) analyses continuously the APW for estimation of CO and cardiac index (CI). The device also calculates SVV over a 20-s time frame using the formula: SVV = \( SV_{\text{max}} - SV_{\text{mean}} \times SV_{\text{mean}} \).

The FloTrac sensor was attached to the arterial catheter and the Vigileo® was connected to the vital signs monitor (Philips MP70; Philips, Eindhoven, Netherlands) for continuous data registration.

The Masimo Radical 7 SET (V7.6.0.1, sensor version R2–25, Rev E) uses transcutaneous multi-wavelength analysis for non-invasive measurement of arterial oxygen saturation and total haemoglobin concentration with the use of a finger clip. This device also calculates PVI, which is based on the perfusion index (PI). The PI represents the ratio between pulsatile and non-pulsatile blood and is a measure of local blood flow. PVI is subsequently calculated as \( \frac{[PI_{\text{max}} - PI_{\text{min}}]/PI_{\text{max}}] \times 100 \) over a period of time sufficient to include multiple respiratory cycles. The Masimo sensor was attached to the index finger contralateral to the arterial catheter according to the manufacturer’s instructions.

The APW-based pulse pressure variation (PPV) was recorded online by the clinically used vital signs monitor and calculated off-line afterwards using dedicated software developed by the authors. For the interested reader, also systolic pressure variation (SPV; APW-based), the PW-based variation in peak amplitude (PWpeak), and pulse amplitude (PWpulse) were calculated. The results of these three dynamic preload variables are presented in supplementary material. All dynamic preload variables were, after synchronization, calculated over a time frame of 20 s. Obvious artifacts were eliminated by visual inspection of waveforms. Eadyn was calculated as the SVV/PPV ratio, as described previously.

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Data recording

Data were recorded by a Windows XP-based medical grade personal computer running RugLoop II data collection software (Demed Engineering, Temse, Belgium). RugLoop II output files were exported to Microsoft Excel 2010 (Microsoft, Redwood, MS, USA) and PASW Statistics 18 (IBM, Inc., Chicago, IL, USA) for further analysis. All discrete variables (SpO2, tidal volume, heart rate, MAP, CVP, SVI, CO, CI, SVV, and PVI) were recorded with a sampling rate of 1 Hz, whereas the original APW and PW were stored with a sampling rate of 100 Hz and later used for calculation of dynamic preload variables.

Statistical analysis

All continuous variables were tested for normality using the Kolmogorov–Smirnoff test. Student’s t-test was used for normally distributed continuous variables and the Mann–Whitney test for non-normally distributed variables. For categorical variables, Fisher’s exact test was applied. The Wilcoxon signed-rank test was used to compare haemodynamic variables before and after fluid administration.

We assessed the ability of dynamic preload variables to predict fluid responsiveness using receiver operating characteristic (ROC) analysis and subsequently calculated the areas under the ROC curve (AUROCs). AUROCs were compared using the DeLong methodology. Optimal cut-off values maximizing appropriate classification were calculated using the Youden index (calculated as: sensitivity + specificity – 1), along with its sensitivity and specificity. In addition, the ability of Eadyn to predict a decrease in norepinephrine requirement at the end of fluid administration was assessed using calculation of its AUROC.

Results

A total of 30 patients were included in this study. Patient characteristics, except for blood loss and surgery duration, were normally distributed as previously described.12

Changes in static variables during fluid administration

MAP remained stable [74 (11) vs 74 (9) mm Hg]. CVP increased from 6 (3) to 9 (4) mm Hg (P<0.05). The mean SVI increased from 36 (12) ml m⁻² at the start of fluid administration to 45 (13) ml m⁻² at the end of fluid administration (P<0.05). The mean percentage increase of SVI was 9% with a range from –14 to 33%. In total, 17 out of 30 patients showed an increase in SVI of at least 20% after fluid administration and were considered fluid responsive.

Haemodynamic effects of fluid administration in fluid responders and non-responders

All dynamic preload variables and heart rate were higher in responders than in non-responders at the start of the fluid administration, while SVI was lower in these patients (Table 1). MAP and CVP were not different between groups. In fluid responders, all dynamic preload variables except PVI decreased at the end of fluid administration (P<0.05); CVP increased slightly (P<0.05). Of note, the decrease of PPV was more pronounced than the decrease of SVV (–14 vs –10%, respectively). In non-responders, both SVV and PPV also decreased (P<0.05) but again being more pronounced for PPV. CVP increased slightly (P<0.05). PVI and static preload variables did not change.

Prediction of fluid responsiveness

Figure 1 depicts the ROC curve for the prediction of fluid responsiveness of SVV, PPV, and PVI. All individual AUROCs
were different from zero \( (P<0.05) \); the associated confidence intervals are also shown in Figure 1. Comparison between all the AUROCs of the dynamic preload variables revealed no significant differences using Hanley and McNeil analysis.

In Table 2, optimal cut-off values, as determined by the Youden index, are shown for dynamic preload variables along with the associated sensitivity and specificity. Additionally, the AUROC of CVP was 0.46 (CI: 0.24–0.68; \( P=0.71 \)) and was significantly different from AUROCs of all dynamic preload variables.

### Influencing factors on haemodynamic variables

Median (range) norepinephrine dosages at the start of fluid administration were similar between responders [0.15 (0.02–0.55) \( \mu \text{g kg}^{-1} \text{ min}^{-1} \)] and non-responders [0.15 (0.01–0.74) \( \mu \text{g kg}^{-1} \text{ min}^{-1} \); \( P=0.3 \)], as was the reduction of norepinephrine dosage at the end of fluid administration [0.13 (0.0–0.46) vs 0.12 (0.01–0.51) \( \mu \text{g kg}^{-1} \text{ min}^{-1} \); \( P=0.7 \)] to keep MAP between 60 and 80 mm Hg. In addition, the mean tidal volume was 10.5 (1.8) ml kg\(^{-1} \) lean body mass at the start and end of the fluid administration, while the mean heart rate/respiratory rate was 7.9 (1.7) at the start and 7.8 (1.3) at the end of the fluid administration.

### Agreement between dynamic preload variables

All dynamic preload variables were significantly correlated with each other at the start of the fluid administration (Table 3, orange boxes). After fluid administration (Table 3, green boxes), PVI did not correlate significantly with SVV.

All other investigated dynamic preload variables were correlated significantly.

### Association \( E_{\text{adyn}} \) with norepinephrine requirement and MAP

\( E_{\text{adyn}} \) was neither associated with MAP at the start of fluid administration (\( r=0.28 \), n.s.) nor with the absolute difference in MAP between the end and start of fluid administration (\( r=-0.1 \), n.s.). In patients in whom norepinephrine could be decreased at the end of fluid administration (\( n=15 \)), mean (so) \( E_{\text{adyn}} \) was 1.6 (0.4), while \( E_{\text{adyn}} \) was 1.2 (0.4) in patients in whom norepinephrine requirement was at least equal to the required dose at the start of fluid administration (\( P<0.05 \)). Furthermore, \( E_{\text{adyn}} \) was significantly correlated with the norepinephrine requirement (\( r=0.47 \)) and was also inversely correlated with the difference in norepinephrine requirement before and after fluid administration (\( r=-0.55 \)).

The AUROC of \( E_{\text{adyn}} \) to predict a decrease in norepinephrine requirement (\( n=15 \)) was 0.81 (CI: 0.65–0.97; \( P<0.05 \)). The optimal cut-off \( E_{\text{adyn}} \) value was found at 1.25 with a sensitivity of 80% and a specificity of 73%.

### Discussion

We investigated the ability to predict fluid responsiveness of dynamic preload variables that were either APW based or PW based. We found all of these dynamic preload variables to have a similar ability to predict fluid responsiveness in patients undergoing major hepatic resection, although optimal cut-off values seem to be somewhat different. Furthermore, all the APW-based dynamic preload variables, but not PVI, decreased after fluid administration, that is, were able to track the haemodynamic changes associated with this intervention. Additionally we found the dynamic arterial elastance (\( E_{\text{adyn}} \)) capable of predicting a reduction in norepinephrine requirement at the end of fluid administration, suggesting that \( E_{\text{adyn}} \) is able to estimate the influence of norepinephrine administration on intrinsic arterial tone after volume expansion.

In recent years, many studies have shown the superior ability of dynamic preload variables over static preload variables (e.g. CVP) for the prediction of fluid responsiveness.\(^3\)\(^5\)\(^6\) Data from the present study are consistent with these studies, with the AUROC for the prediction of fluid responsiveness of CVP being poor whereas all dynamic preload variables performed better. Interestingly, CVP increased both in fluid responders and in fluid non-responders significantly, although the observed increase was small (from 6 to 9 and from 7 to 9 mm Hg, respectively).

PPV and SVV are the best-known dynamic preload variables. In our population, the optimal cut-off values for SVV and PPV to predict fluid responsiveness were 15% and 14%, respectively. A meta-analysis considering studies that investigated these APW-based dynamic preload variables found that optimal cut-off SVV and PPV values are generally between 11% and 13% with a pooled AUROC of 0.84 and 0.94.\(^1\)\(^4\) Most previous studies assessing the ability of dynamic

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\( \text{PPV} \) (mm Hg) | 0.70* | 0.71* | 0.72* | 0.80* | 0.81* |
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preload variables to predict fluid responsiveness are done under optimal circumstances either during surgery or in the intensive care unit; these often report high sensitivity and specificity (>90%).

For the correct interpretation of dynamic preload variables, patients must be in sinus rhythm and be mechanically ventilated in a volume-controlled mode with tidal volumes >8 ml kg\(^{-1}\) and a heart rate/respiratory rate ratio >3.6. Frequently, in routine clinical care, particularly in the intra-operative setting, these criteria are not met resulting in a decreased ability of dynamic preload variables to predict fluid responsiveness. Despite the fact that all our patients met these criteria during the period of fluid administration, we found lower AUROCs for both SVV and PPV compared with the literature. Our data were obtained in patients under general anaesthesia undergoing extensive hepatic surgery: during hepatic surgery, the surgeon can easily manipulate central veins (i.e. the inferior caval vein), which can subsequently cause alterations in cardiac venous return influencing preload and thus influencing dynamic preload variables. Although measurements were obtained only after hepatic resection was completed, we cannot rule out some influence of the surgery being performed at the time of measurement causing lower sensitivity and specificity of SVV and PPV. Therefore, our data probably represent ‘real life’ clinical circumstances.

A previous report investigated the ability of PPV to predict fluid responsiveness in patients undergoing liver surgery. Here, the AUROC of PPV either derived from the APW or from the non-invasive Finapres device (Ohmeda Monitoring Systems, Englewood, CO, USA) were 0.79 and 0.81, respectively (comparable with our data), but the AUROC of PW-based ‘PPV’ was only 0.68. In contrast, we demonstrated in our population that all PW-based dynamic preload variables, calculated either using proprietary algorithms (PVI) or using automated algorithms (PW\(_{\text{peak}}\), PW\(_{\text{pulse}}\), see supplementary material) predict fluid responsiveness to a similar extent. Importantly, we observed relatively wide 95% confidence intervals of AUROCs of the studied dynamic preload variables. This finding most probably reflects the inclusion of a limited number of patients (n=30) in the current study.

We found that all individual dynamic preload variables were significantly correlated to each other before fluid administration (Table 3) and, most importantly, could predict fluid responsiveness to a similar extent, irrespective of the fact whether these variables were derived from the APW or from the PW. This finding is compatible with some previous studies investigating simultaneously both APW- and PW-based variables, while it is noteworthy that we assessed almost all of the most common dynamic preload variables altogether in one population. One of these studies in critically ill patients assessed the ability to predict fluid responsiveness of APW-based PPV and ‘PPV’ calculated by the CNAP device.

One aspect not frequently taken into account in studies involving dynamic preload variables is the ability of these variables to track changes induced by fluid administration, despite its obvious clinical importance. In the present study, we observed the APW-based dynamic preload variables, but not PVI, to decrease after fluid administration in fluid responsive patients. In addition, the correlation of PVI with SVV was also lost (Table 3), suggesting that PVI is unable to track changes induced by fluid administration. As per our original protocol, patients were randomly assigned to receive a fluid bolus of either colloid (n=15) or crystalloid (n=15) solution and based on this, we previously reported that colloid solution might influence the accuracy of haemoglobin measurement by the Masimo Radical 7 device. This might also be true for calculation of PVI, given the inability of this dynamic preload variable to track changes induced by fluid administration. Nevertheless, because of the limited sample size per group, we cannot draw a firm conclusion on this issue.

The finding that the Masimo Radical 7 device is unable to track changes after fluid administration despite providing an adequate prediction of fluid responsiveness is however of major clinical importance: if an anaesthetist were using this device alone, one may conclude that no significant haemodynamic effect resulted from a fluid bolus and that the patient is either a non-responder or is in need for more fluid; both of which are of course incorrect if fluid did produce an adequate effect on SVI. From a physiological viewpoint, it can be speculated that the flow-derived dynamic variable (i.e. SVV) is more important than pressure-based (i.e. PPV) or volume-based variables (i.e. PVI), since blood flow is the main determinant of oxygen delivery. Of interest, while the volume-based PVI was not at all to track changes after volume expansion, the pressure-based PPV did track changes in volume responders, but also gave a high percentage of false-positive results in non-responders (specificity 62%, Table 2). The flow-based SVV both tracked changes after volume expansion adequately and also had the largest AUROC in predicting fluid responsiveness.

Another important aspect of intra-operative haemodynamic optimization is the assessment of cardiac afterload, for which we calculated the pressure/flow ratio (PPV/SVV; E\(_{\text{adyn}}\)). In a recent study in ICU patients with acute circulatory failure, E\(_{\text{adyn}}\) was shown to accurately predict an increase in MAP (≥15%) after fluid administration with an AUROC of 0.986. E\(_{\text{adyn}}\) was also related in this study to the increase in MAP after fluid administration and was different in MAP responders and non-responders. In our study, norepinephrine was titrated to obtain an MAP above 60 mm Hg and because norepinephrine was reduced during fluid administration meticulously in accordance with the MAP on clinical reasons, we found no clear relationship between E\(_{\text{adyn}}\) and MAP. We could however demonstrate that E\(_{\text{adyn}}\) was associated with the reduced norepinephrine requirement after fluid administration. Additionally, E\(_{\text{adyn}}\) proved to be able to predict a decrease in norepinephrine requirement at the end of fluid administration and E\(_{\text{adyn}}\) was significantly higher in those patients (n=15) in whom norepinephrine requirement decreased. We speculate that these patients requiring less vasopressor support after volume expansion had more intrinsic vascular tone.
thereafter, as reflected by the Ea_syn value, which is simply calculated as the PPV/SVV ratio. These findings suggest the Ea_syn value is able to assess vascular tone and might subsequently aid in the decision as to whether a patient requires afterload support using vasopressors (e.g., norepinephrine) after preload is assessed and optimized using assessment of fluid responsiveness by dynamic preload variables.

**Study limitations**

The SVI was calculated from the measured CI by the FloTrac-Vigileo™ device, which uses auto-calibrated pulse contour analysis of the APW. As a limitation, every device monitoring CI has an intrinsic variability, for which a generally allowed inter-device percentage error as high as up to 30% is considered reasonable. The accuracy of CI calculation by this device is generally believed to be within this accuracy limit. Although, irrespective of its accuracy, we used relative instead of absolute SVI values in this study. Therefore, we suppose our study results are not affected by the absolute value of CI and SVI. The use of norepinephrine may have affected the auto-calibration of the FloTrac-Vigileo™ device. The value of assessing dynamic arterial tone using Ea_syn with respect to the ability of the FloTrac-Vigileo™ device to track changes in CO after dosage changes of vasopressors should be subjected to further research. The fluid bolus administered in our study, required for clinical reasons, was larger than that usually administered in a general surgical population, so that less distinct results might be expected in other settings.

In conclusion, PVI can predict fluid responsiveness comparable with that of dynamic preload variables based on the APW. Yet, each dynamic preload variable has a different optimal cut-off value. In addition, the APW-based dynamic preload variables, but not PVI, were able to track changes induced by fluid administration. Further technical improvements of the Masimo Radical 7 software and its sensor are necessary to reduce variability in PVI measurements and to improve the ability to track changes induced by fluid administration. Furthermore, the pressure/flow relationship of PPV and SVV, expressed as the Ea_syn, was able to predict a reduction in norepinephrine requirement. Thus we speculate that Ea_syn may serve as a sensitive indicator of intrinsic arterial tone and the influence of norepinephrine thereon in mechanically ventilated patients undergoing major hepatic resection.

**Supplementary material**

Supplementary material is available at British Journal of Anaesthesia online.

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**Declaration of interest**

M.M.R.F.S. is an editor for the British Journal of Anaesthesia, but had no role in the handling of this manuscript. He also served three times as a panel member of the Masimo Advisory Board. T.W.L.S. received honoraria for lectures from Edwards Lifesciences.

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**References**

14. Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived variables and fluid responsiveness in


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