Mean systemic filling pressure indicates fluid responsiveness and anesthesia-induced "unstressed" blood volume

Robert G. Hahn (robert.hahn@ki.se)
Södertälje Hospital, Karolinska Institutet at Danderyds Hospital (KIDS)
Rui He
Shaoxing People's Hospital
Yuhong Li
Shulan International Hospital

Research Article

Keywords: Mean systemic filling pressure, fluid therapy, fluid responsiveness, general anesthesia, stroke volume

Posted Date: March 15th, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1442276/v1

Abstract

Background. The mean systemic filling pressure (P_{ms}) play a central role for the understanding of the circulation. We studied whether the P_{ms} indicates fluid responsiveness before and after induction of general anesthesia and whether the P_{ms} response to bolus infusions of fluid indicates an anesthesia-induced increase of the "unstressed" blood volume.

Methods. An analog to P_{ms} based on cardiac output, the mean arterial pressure, and central venous pressure (P_{msa}) was calculated in 86 patients before induction of general anesthesia and before each of 3 successive bolus infusions of 3 ml/kg of colloid fluid. An increase in stroke volume of ≥ 10% from a bolus infusion indicated fluid responsiveness. Changes in blood volume were estimated from anthropometric data and the hemodilution.

Results. P_{msa} was lower in fluid responders than in non-responders before induction (13.2 ± 2.2 vs. 14.7 ± 2.7 mmHg; mean ± SD, $P < 0.01$) and after induction of general anesthesia (11.4 ± 2.1 vs. 12.8 ± 2.1 mmHg; $P < 0.006$). Changes in P_{msa} resulting from the infusions did not differ depending on the stroke volume response. Receiver operator characteristic curves showed an average area under the curve of 0.70. The decrease in P_{msa} due to the anesthesia-induced increase of the vascular compliance was fully compensated by the colloid fluid. A linear correlation between P_{msa} and the volume changes suggested that the anesthesia increased the "unstressed" blood volume by 1.2 L.

Conclusions. P_{msa} was lower in fluid responders than in non-responders. General anesthesia increased the need for blood volume by 1.2 L.

Introduction

The mean systemic filling pressure (P_{ms}) is the pressure that develops in the systemic circulation if the heart suddenly stops [1]. The importance of P_{ms} for the vascular status was first studied by the British physiologist Arthur Guyton. His view was that the heart fills passively. Therefore, cardiac output (CO) is determined by the venous return (VR), which is, in turn, driven by the difference between P_{ms} and the central venous pressure (CVP) divided by the resistance to venous return (RVR). The theories surrounding the role of P_{ms} as a key determinant of the circulation are sometimes called “Guyton´s hemodynamics” and offer complementary views on how to interpret hemodynamic data [2, 3].

A key problem is that P_{ms} is difficult to measure, which necessitated the development of predictive algorithms. The best-known analog, P_{msa}, is based on CVP, mean arterial pressure (MAP), and CO [4, 5]. This analog is implemented in a commercially available monitor, Navigator (Applied Physiology, Pty Ltd., Sydney, Australia). Cecconi *et al.* connected a Navigator module to a pulse contour hemodynamic monitor and recorded Guyton´s variables in postsurgical patients. Their reported hemodynamic changes

agreed with Guyton´s views [6]. Later evaluations showed that P_{msa} correlates well with more invasive laboratory methods of measuring P_{ms} [7–11].

The purpose of the present report was to evaluate if the P_{msa} predicts whether a patient is fluid responsive. The assessment of fluid responsiveness is the key methodology used for clinical evaluation of the need for fluid administration during surgery and intensive care. A secondary purpose was to estimate the increase in "unstressed blood volume" that occurs when general anesthesia is induced. To our knowledge, this is a novel use of P_{msa}.

The hypotheses were that P_{msa} predicts fluid responsiveness and that data on P_{msa} can provide information about the "unstressed" blood volume.

Materials And Methods

Patients

This is a retrospective analysis of a prospective study that included patients with suspected or established gastric, colonic or rectal cancer who were recruited to participate in an open-labeled clinical trial [12, 13]. They underwent laparoscopic or open gastrointestinal surgery under combined intravenous and inhalational general anesthesia. Exclusion criteria were liver or renal dysfunction (liver enzymes > 50% or serum creatinine > 50% of normal), coagulation disturbances, obstructive pulmonary disease, atrial fibrillation, and mental disorders.

The protocol was approved by the Ethics Committee of the First Affiliated Hospital, College of Medicine, Zhejiang University (Hangzhou, PR of China; No. 2011150, Official in charge: Zhangfei Shou) and the registered at the Chinese Clinical Trial Registry (http://www.chictr.org/en; No. ChiCTR-TNRC-14004479). Written informed consent was obtained from each study subject. Reporting adhered to the CONSORT checklist.

Anesthesia

The patients fasted overnight and no premedication was given. Anesthesia was induced and tracheal intubation performed by using propofol, fentanyl, and cisatracurium. Mechanical ventilation was used with a tidal volume set to 8 ml/kg, 12 breaths/min, and a PEEP of 3 cm H_2O. The anesthesia was maintained by 1–2% of sevoflurane and infusion and remifentanil. No adrenergic drugs were administered.

Fluid program

No fluid was induced during the induction of general anesthesia. Beginning 10 min after the tracheal intubation, three bolus infusions of 6% hydroxyethyl starch 130/0.4 (Voluven®; Fresenius Kabi Deutschland GmbH, Bad Homburg, Germany) were given in the volume at 3 ml/kg over 7 min via an infusion pump (IEC 601–1; Abbott Laboratories, Chicago, IL). The hemodynamic response was recorded

5 min after the end of each bolus infusion. The flat recumbent body position was maintained, and surgery was not initiated until all three optimizations had been completed [12].

Measurements

When the patient entered the operating theater, catheterization of the left radial artery and right interval jugular vein was performed under local anesthesia and sedation by midazolam. The arterial line was connected to a FloTrac™ sensor, from which data were sent for analysis to a Vigileo monitor (Software version 3.6; Edwards Lifesciences, Irvine, CA). The arterial waveform pulse contour was used to calculate the stroke volume (SV). Monitoring also included central venous pressure (CVP), pulse oximetry, electrocardiography, and heart rate (Datex-Ohmeda, Hoevelaken, the Netherlands).

The CVP was calibrated prior to induction of anesthesia. The zero point corresponded to the level of 4th rib in the anterior axillary line. The effect of a few extreme outliers was reduced by setting changes in CVP > 4 mmHg in response to a single bolus infusion at 4 mmHg.

Data on central hemodynamics were collected before and after induction of anesthesia, just before the first bolus infusion was initiated, and again 5 min after each of the bolus infusions ended.

Fluid responsiveness

The target in flow-guided optimization with fluid loading is to reach the top of the Frank-Starling curve. Therefore, the patient is a responder if a bolus infusion raises SV by ≥ 10% and non-responder if the increase is < 10% [14, 15]. As flow-guided optimization implies a titration process, a bolus is indicated if given after an infusion in which the patient was fluid responsive, but the subsequent bolus is warranted only if the SV increased by ≥ 10%.

Guyton´s hemodynamic variables

An analog to the mean circulatory filling pressure (P_{msa}) has been derived from measurements of CVP, MAP, and CO, assuming a constant veno-arterial compliance of 24:1 [4–6]:

$P_{msa} = a\,CVP + b\,MAP + c\,CO$

where $a = 0.96$ and $b = 0.04$ ($a + b = 1$) while c is a resistance derived from anthropometric data [6];

$c = 0.0038\,(904.17 + 0.193\,age) / [4.5\,(0.99^{\,age-15}\,0.007184\,(height^{0.725})\,weight^{0.425}]$

Pressure gradient for venous return (dVR) is obtained as: $dVR = P_{msa} - CVP$

The global pumping efficiency (Eh) is calculated as: $Eh = (P_{msa} - CVP) / P_{msa}$

The resistance to venous return (RVR) was obtained as: $RVR = dVR / CO$

Blood volume

The blood volume changes in response to the bolus infusions were calculated by multiplying the change in the blood hemoglobin concentration with the baseline blood volume, which was estimated based on the height and weight of each volunteer [16].

Statistics

The data are presented as mean (SD) and differences between groups evaluated by one-way analysis of variance (ANOVA). $P < 0.05$ was considered statistically significant.

Receiver operator characteristic (ROC) curves were created with IBM SPSS Statistics Version 22. The ROC curves are probability curves in which sensitivity (true positive fraction) is plotted *versus* 1 – specificity (false positive fraction). The calculated area under the curve (AUC) for this relationship reflects how well the ranges of fluid intake can be separated. The given prediction is statistically significant if the 95% confidence interval does not include 0.5.

Results

The cohort consisted of 86 patients (65% male). Data were missing from 7 patients, so the final analysis consisted of 79 subjects. They subjects were 56 ± 13 years old, had a height of 184 ± 8 cm, and body weight of 60 ± 8 kg. All patients received three bolus infusions after general anesthesia had been induced. The hemodynamic data are summarized in **Table 1.**

Table 1
Basic hemodynamic data for all patients. Mean ± SD.

	Before anesthesia	**Before 1st bolus**	**Before 2nd bolus**	**Before 3rd bolus**	**After 3rd bolus**
Stroke volume (ml)	82 ± 25	53 ± 16	60 ± 15	65 ± 16	67 ± 16)
MAP (mmHg)	104 ± 13	76 ± 10	75 ± 10	74 ± 11	75 ± 10
CVP (mmHg)	5.0 ± 3.0	6.1 ± 3.3	6.7 ± 3.2	7.4 ± 3.1	8.3 ± 3.2
P_{msa} (mmHg)	13.8 ± 2.5	11.9 ± 2.2	12.4 ± 2.2	13.1 ± 2.3	14.0 ± 2.4
dVR (mmHg)	9.0 ± 1.1	4.8 ± 1.8	5.3 ± 1.2	5.2 ± 1.3	5.1 ± 1.2
Eh (no unit)	0.67 ± 0.11	0.42 ± 0.18	0.44 ± 0.12	0.41 ± 0.13	0.37 ± 0.11
RVR (mmHg min/L)	1.5 ± 0.3	1.3 ± 0.4	1.4 ± 0.4	1.4 ± 0.4	1.3 ± 0.3
VR (L/min)	6.3 ± 1.7	3.8 ± 1.1	3.9 ± 1.0	4.0 ± 1.2	3.9 ± 1.1
”Warranted” bolus		63%	44%	22%	

Fluid responsiveness

Fluid responsiveness was evident in 63%, 44%, and 22% of the patients before each bolus infusion. The ROC curves showed that P_{msa} could separate responders from non-responders with an AUC of approximately 65–70% before administration of any of the bolus infusions. Fluid responsiveness could be indicated even before anesthesia induction (**Fig. 1**).

P_{msa} measured before the induction of anesthesia indicated how many of the bolus infusions that would later become "warranted" ($P < 0.03$; **Fig. 2**).

P_{msa} differed significantly between subjects who would be non-responders and those who would be responders during the subsequent bolus infusion. This was a consistent finding (**Fig. 3A**). Induction of anesthesia was followed by a marked decrease in both dVR and Eh, but further changes and differences between non-responders and responders were negligible (**Fig. 3B, C**). RVR tended to be higher in the responders, but differences were small (**Fig. 3D**). Stroke volume showed the same pattern as P_{msa}, but the differences between responders and non-responders were smaller for stroke volume (**Fig. 3E**).

To understand **Fig. 3**, note that the patients were continuously redefined as non-responders and responders and that each patient could switch between these groups at different points in time.

"Unstressed volume"

Figure 4 illustrates how the "unstressed blood volume" was increased by general anesthesia. The onset of anesthesia decreased P_{msa} by 3.3 mmHg, as indicated by an arrow. The blood volume at baseline amounted to 4.3 ± 0.8 L, and the volume expansion from the bolus infusions is plotted versus P_{msa} to obtain a vascular compliance curve. The increase in the "unstressed blood volume" amounted to 1.2 L, which is indicated by the horizontal shift from the baseline P_{msa} (13.8 mmHg) to this curve.

Discussion

Key results

P_{msa} predicted fluid responsiveness before a fluid bolus was infused during general anesthesia. P_{msa} also indicated the fluid responsiveness prior to anesthesia induction and even how many fluid boluses would be needed until SV no longer increased by ≥ 10%. However, the overall discriminating capacity of P_{msa} to predict fluid responsiveness was not impressive. The ROC curves yielded confidence intervals that were statistically significant but only with modest margins.

We used the fluid-induced responses in P_{msa} to estimate how much the anesthesia-induced reduction in the vascular compliance increased the "unstressed" blood volume, which is the fraction of the intravascular volume that does not generate pressure [3]. Fig. 4 shows that 1.2 L of blood would be needed to restore P_{msa} to its pre-anesthesia level.

Guyton´s parameters

The research works by Arthur Guyton from the 1950s link circulatory volume with pressure and flow. The central concept is the mean circulatory filling pressure (P_{mcf}), which is the pressure that in develops in vascular system if the blood flow is quickly stopped. A closely related variable is the mean systemic filling pressure (P_{ms}) which denotes the pressure when equilibrated throughout the systemic circulation [2]. The P_{ms} and P_{mcf} values are usually similar and are often used interchangeably.

The driving force for venous return (dVR) is the difference between P_{ms} and the right atrial pressure, which is measured clinically as the CVP. Thus, a high CVP operates as a resistor to the venous return, which governs CO. The flow gradient is stronger if P_{ms} is high, which is expressed by the parameter denoted Eh, because the resistance to flow by the CVP then becomes less important. One may say that Eh is a measure of how effectively a volume change increases the CO.

Hemodynamic findings

The likelihood of fluid responsiveness was higher when P_{msa} was low. This is logical, as P_{msa} reflects the "stressed" blood volume and the vascular compliance. The fluid-induced increases in P_{msa} did not differ significantly between responders and non-responders, and this was also expected because the same fluid volume was given to all patients.

The dVR and Eh decreased by 30–40% after the induction of general anesthesia, which was reflected in a drop in stroke volume by 35%. By contrast, P_{msa} only decreased by 25%, as shown in Fig. 4. This difference can be explained by the increase in CVP, which is mostly likely due to the positive-pressure that was initiated as soon as patients were anesthetized. This suggests that 2/3 of the reduction of the SV could be accounted for by an anesthesia-induced increase in the vascular compliance. The resistance to venous return (RVR) is not expected to change during hyper- or hypovolemia [8], and only small changes were found in the present study.

"Unstressed" blood volume

The "unstressed" blood volume is usually obtained from the intercept of the y-axis (volume) at zero pressure in a vascular compliance plot [3]. Fig. 4 shows this type of plot, but it is based on only the changes within the narrow interval of the present measurements. Here, the horizontal shift between the

baseline P_{msa} and the compliance curve indicated the *increase* in the "unstressed" blood volume due to general anesthesia. This is the volume that the anesthetist aims to compensate using intravenous fluid.

The particularly pronounced blood volume response to the first bolus infusion is probably due to the capillary refill that always occurs in response to the decrease in arterial pressure accompanying anesthesia induction, even in the absence of intravenous fluid administration [17]. Capillary refill is also the reason why the first post-induction P_{msa} could not be used in Fig. 4, as the blood volume change was not zero and no matching hemoglobin value was available.

Overall, the bolus infusions expanded the blood volume by more than the infused amount. This is reasonable, as the colloid osmotic pressure of the fluid is 33% higher than normal blood plasma [18]. However, the intravenous retention of the infused fluid, being higher for colloids than for crystalloids, is unlikely to matter much for the present calculations.

Crystalloid fluid might even offer an alternative way to estimate the anesthesia-induced increase of the "unstressed" volume. Kinetic analysis of hemodilution curves in women scheduled for abdominal hysterectomy showed that capillary leakage of fluid was arrested when 1.24 L (16.6 ml/kg) of Ringer´s had been infused, which is similar to the value found here [19]. This finding suggests that a low P_{msa} counteracts the capillary leakage of fluid when the blood volume is expanded by crystalloid fluid.

Literature

The central idea of Guyton´s hemodynamics is that CO is determined by the venous return, whereas the heart plays a permissive role [3]. Despite critical views, this concept has received widespread attention among physiologists, anesthetists, and intensivists alike [20].

Basic studies have been performed in pigs, where P_{ms} has been derived by ventilatory maneuvers [21, 22] and, recently, with extracorporeal membrane oxygenation [11].

Attempts to use P_{ms} in the clinic have been made over the past decade. Three methods are used. One is to calculate P_{ms} when VR is suppressed by stepwise deep inspirations. The second is to arrest the circulatory flow in one arm by inflating a blood pressure cuff and then to obtain P_{ms} when the arterial and venous pressures have become equal. The third method is to calculate the P_{ms} analog called P_{msa}, which was the approach used in the present work.

Comparisons between these methods in cardiac surgery have shown, in one study, acceptable agreement between P_{ms} values, but good agreement between changes in effective blood volume [7]. Meijs *et al.* compared inspiratory holds with P_{msa} in cardiac surgery and found the methods to be interchangeable [10].

Cecconi *et al.* measured Guyton´s hemodynamic parameters in 39 postoperative patients who received different vasoactive therapies and respiratory support [6]. The P_{msa} showed great variability and did not increase in response to a fluid bolus consisting of either crystalloid or colloid fluids.

A review by Cooke *et al.* supports our finding that P_{msa} is lower in fluid responders than in non-responders [9]. However, fluid challenges and passive leg raising increased P_{msa} more in the responders, and by a greater incremental change than we found. These differences may be due the anesthetized state of our patients.

Limitations

CO was measured by the arterial waveform pulse contour analysis implemented in the FloTrac/Vigileo system. This hemodynamic monitor can be used in conscious patients, but it is uncalibrated and may then have a higher coefficient of variation than is observed with calibrated monitors [23].

The data were collected in the clinical setting, and the occasionally high SV apparent at baseline may be due to preoperative stress.

The strengths of the study include the uniform anesthetization of the patients and their freedom from acute disease. All patients also received the same fluid treatment. Sampling was carefully timed by a single set of investigators. No adrenergic drugs were used, as they may affect vascular tone and P_{msa} [24].

The original patient series included 25 additional patients who received volume loading with Ringer´s solution in addition to the 86 who received colloid. Those who received Ringer were not included, as 3 ml/kg of crystalloid could not adequately challenge fluid responsiveness [12]. Only 20% of these patients showed fluid responsiveness during the first bolus infusion, which is 1/3 of the fraction of patients who received the colloid bolus.

Conclusion

A mean systemic filling pressure analog (P_{msa}) indicated fluid responsiveness in patients who were given general anesthesia followed by three successive bolus infusions of colloid fluid. A comparison between the changes in P_{msa} and the estimated blood volume changes suggested that general anesthesia increased the "unstressed blood volume" by as much as 1.2 L.

Abbreviations

AUC: area under the curve; BIS; bispectral index; CO: cardiac output; CVP: central venous pressure; dVR: pressure gradient for venous return; Eh: global pumping efficiency (Eh); PEEP: positive end-expiratory pressure; P_{ms}: mean systemic filling pressure; P_{msa}: mean systemic filling pressure analog; P_{mcf} : mean circulatory filling pressure; ROC: Receiver operator characteristic; RVR: resistance to venous return; SV: stroke volume; VR: venous return.

Declarations

Acknowledgements. Dr. Xiaojiang Ying recruited the patients. Operation theater nurse Guofang Meng assisted during the experiments.

Author's contributions. YL and RGH planned the study. YL rote the applications and arranged the funding. RH and YL collected the data. RGH made the calculations and authored the manuscript.

Funding: This project was funded by Qianjiang Talents Project of the Technology Office in Zhejiang province (No. 2012R10033), PR of China, and by a grant from the Östergötland City Council (No. LiO-297751), Sweden.

Availability of data and materials. The data used for the statistics is appended as Additional File 1.xls. All original is given in Additional File 2.xls.

Ethics approval. The protocol was approved by the Ethics Committee of the First Affiliated Hospital, College of Medicine, Zhejiang University (Hangzhou, PR of China; No. 2011150, Official in charge: Zhangfei Shou) and the registered at the Chinese Clinical Trial Registry (http://www.chictr.org/en; No. ChiCTR-TNRC-14004479). The study was performed in accordance with the Declaration of Helsinki. Written informed consent was obtained from each study subject.

Consent for publication. Not applicable.

Competing interests. RGH has received a research grant from Grifols for studies of 20% albumin and is Member of Baxter´s IV Fluid Therapy management Advisory Board.

YL and RGH have no conflicts of interest to report.

ORCID ID Robert G. Hahn 0000-0002-1528-3803

References

1. Rothe CF. Mean circulatory filling pressure: its meaning and measurement. J Appl Physiol 1993; 74: 499-509.

2. Henderson WR, Griesdale DEG, Walley KR, Sheel AW. Clinical reviw: Guyton - the role of mean circulatory filling pressure and right atrial pressure in controlling cardiac output. Crit Care 2010; 14: 243.
3. Gelman S. Venous function and central venus pressure. Anesthesiology 2008; 108: 735-748.
4. Parkin WG, Leaning MS. Therapeutic control of the circulation. J Clin Monit Comput 2008; 22: 391-400.
5. Sondergaard S, Parkin G, Aneman A. Central venous pressure: we need to bring clinical use into physiological context. Acta Anaesthesiol Scand 2015; 59: 552-560.
6. Cecconi M, Aya HD, Geisen M, Ebm C, Fletcher N, Grounds RM, Rhodes A. Changes in the mean systemic filling pressure during a fluid challenge in postsurgical intensive care patients. Intensive Care Med 2013; 39:1299-1305.
7. Maas JJ, Pinsky MR, Geerts BF, de Wilde RB, Jansen JR. Estimation of mean systemic filling pressure in postoperative cardiac surgery patients with three methods. Intensive Care Med 2012; 38: 1452-1460.
8. Wijnberge M, Sindhunata DP, Pinsky MR, Vlaar AP, Ouweneel E, Jansen JR, Veelo DP, Geerts BF. Estimating mean circulatory filling pressure in clinical practice: a systematic review comparing three bedside methods in the critically ill. Ann Intensive Care 2018; 8:73.
9. Cooke K, Sharvill R, Sondergaard S, Aneman A. Volume responsiveness assessed by passive leg raising and a fluid challenge: a critical review focused on mean systemic filling pressure. Anaesthesia 2018; 73: 313-322.
10. Meijs LPB, van Houte J, Conjaerts BCM, Bindels AJGH, Bouwman A, Houterman S, Bakker J. Clinical validation of a computerized algorithm to determine mean systemic filling pressure. J Clin Monit Comput 2021; doi: 10.1007/s10877-020-00636-2.
11. Werner-Moller P, Heinisch PP, Hana A, Bachmann KF, Sondergaard S, Jakob SM, Takala J, Berger DC. Experimental validation of a mean systemic pressure analog against zero-flow measurements in porcine VA-ECMO. J Appl Physiol (1985). 2022 Jan 27. doi: 10.1152/japplphysiol.00804.2021.
12. Li Y, He R, Ying X, Hahn RG. Dehydration, hemodynamics and fluid volume optimization after induction of general anesthesia. Clinics 2014; 69: 809-816.
13. Hahn RG, He R, Li Y. Central venous pressure as an adjunct to flow-guided volume optimisation after induction of general anaesthesia. Anaesthesiol Intensive Ther 2016; 48: 110-115.
14. Davies SJ, Minhas S, Wilson RJT, Yates D, Howell SJ. Comparison of stroke volume and fluid responsiveness measurements in commonly used technologies for goal-directed fluid therapy. J Clin Anesth 2013; 25: 466-474.
15. Bahlmann H, Halldestam I, Nilsson, L. Goal-directed therapy during transthoracic oesophageal resection does not improve outcome. Eur J Anaesthesiol 2019; 36: 153-161.
16. Nadler SB, Hidalgo JH, Bloch T. Prediction of blood volume in normal human adults. Surgery 1962; 51:1224-1232

17. Dull RO, Hahn RG. Transcapillary refill: the physiology underlying fluid reabsorption. J Trauma Acute Care Surg 2021; 90: e31-e39.
18. Zdolsek J, Bergek C, Lindahl TL, Hahn RG. Colloid osmotic pressure and extravasation of plasma proteins following infusion of Ringer´s acetate and hydroxethyl starch 130/0.4. Acta Anaesthesiol Scand 2015; 59: 1303-1310.
19. Hahn RG, Nemme J. Volume kinetic analysis of fluid retention after induction of general anaesthesia. BMC Anesthesiol 2020; 20: 95.
20. Beard DA, Feigl EO. CrossTalk opposing views: Guyon´s venous return curves should not be taught. J Physiol 2013; 23: 5795-5797.
21. Versprille A, Jansen JRC. Mean systemic filling pressure as a characteristic pressure for venous return. Pflügers Arch 1985; 405: 226-233.
22. Ogilvie RI, Zborowska-Sluis D, Tenaschuk B. Measurement of mean circulatory filling pressure and vascular compliance in domestic pigs. Am J Physiol 1990; 258: H1925-932.
23. Samoto M, Orii R, Otsuji M, Bougaki M, Imai Y, Yamada Y. Reliability of cardiac output measurements using LiDCOrapid™ and FloTrac/Vigileo™ across broad ranges of cardiac output values. J Clin Monit Comput 2017; 31: 709-716.
24. Tabrizchi R, Pang CCY. Effects of drugs on body venous tone, as reflected by mean circulatory filling pressure. Cardiovasc Res 1992; 26: 443-448.

Figures

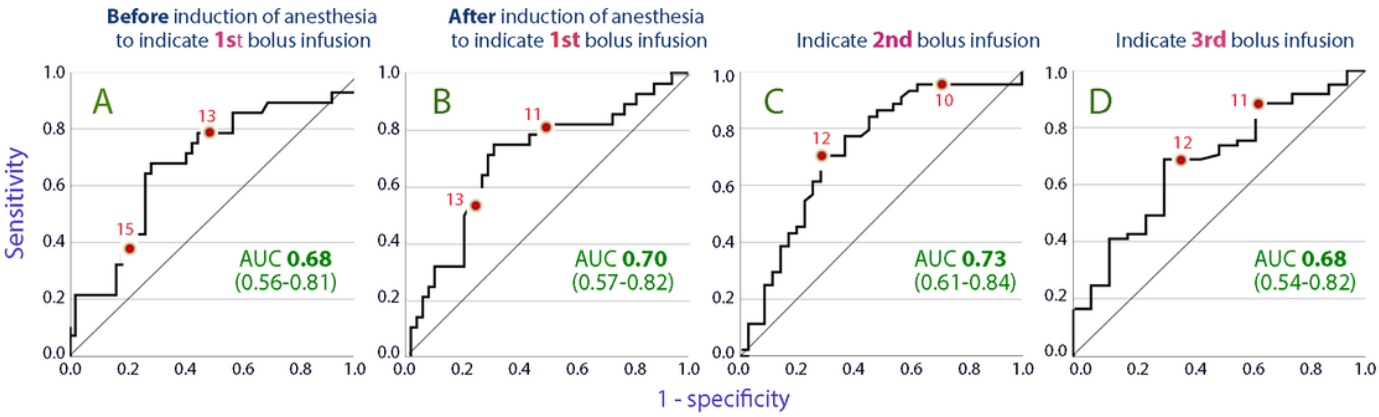

Figure 1

Receiver operating characteristic (ROC) curves was used to express the ability of P_{msa} to predict whether a patient was fluid responsive.

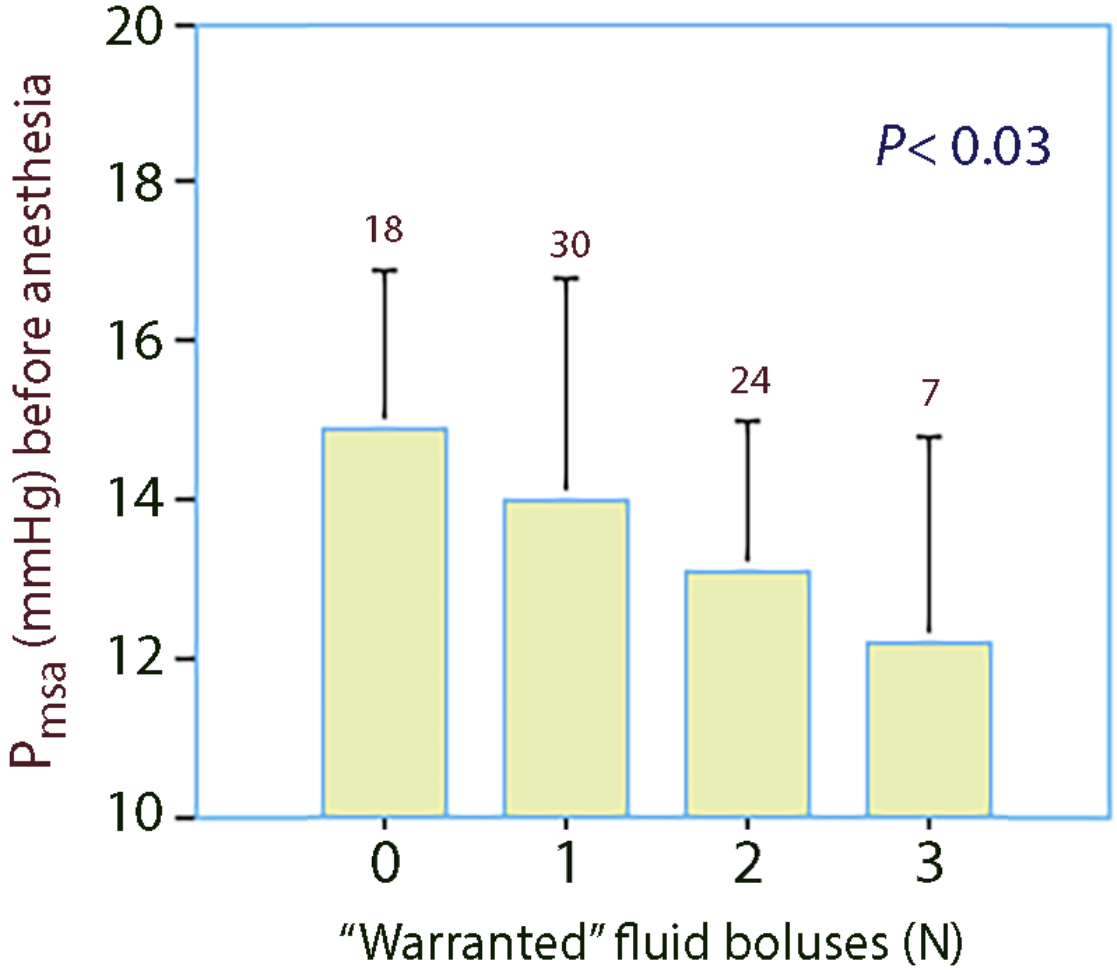

Figure 2

The mean systemic filling pressure (P_{msa}) measured prior to induction of general anesthesia indicated how many of the three subsequent fluid boluses that would be warranted (showed fluid responsiveness). Digits on top of each bar show the number of patients in each group.

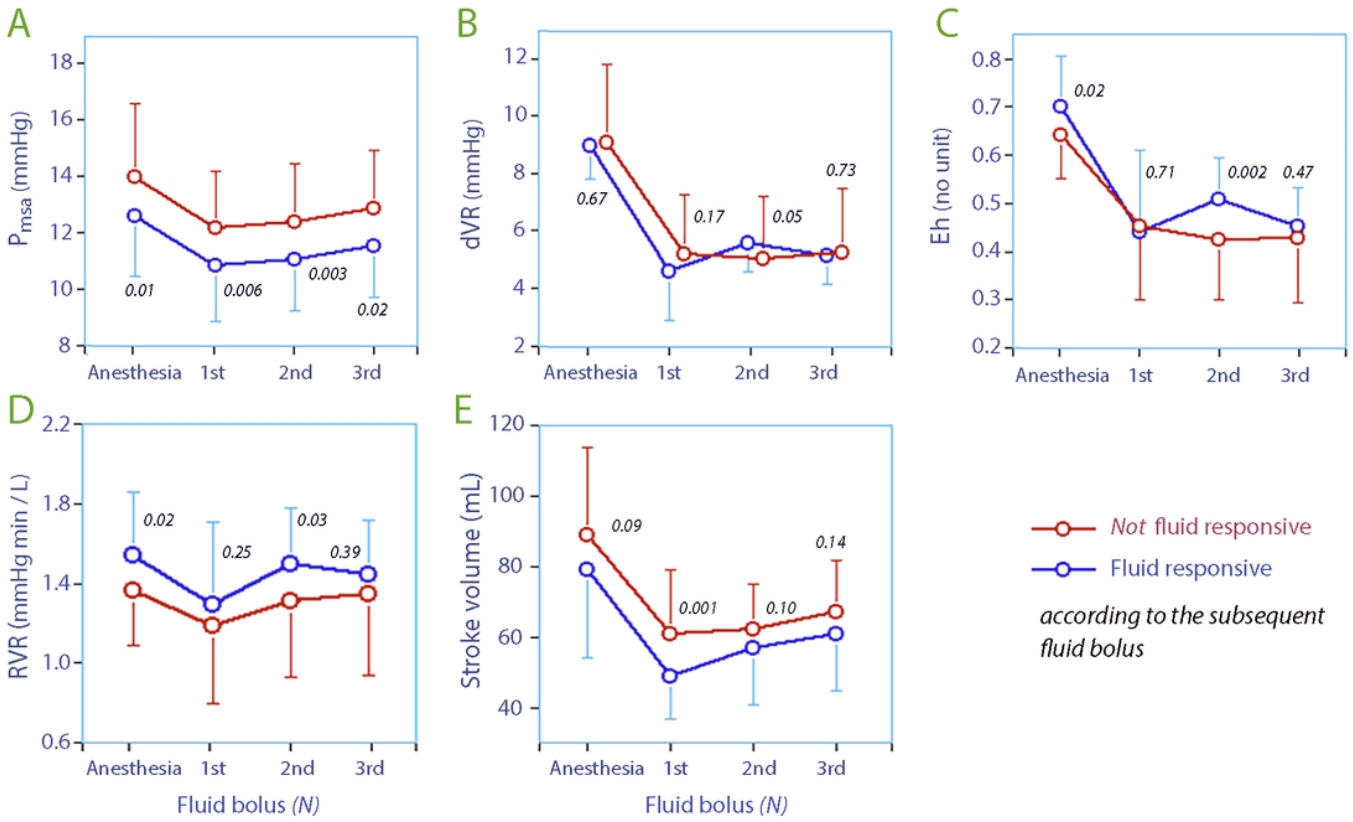

Figure 3

Guyton´s hemodynamic parameters (A-D) and the stroke volume (E) depending on whether patients were non-responders and responders before anesthesia was induced and just prior to administration of the three subsequent fluid bolus infusions. Statistical comparison was made at each level. Note that the number of included patients changed for each comparison.

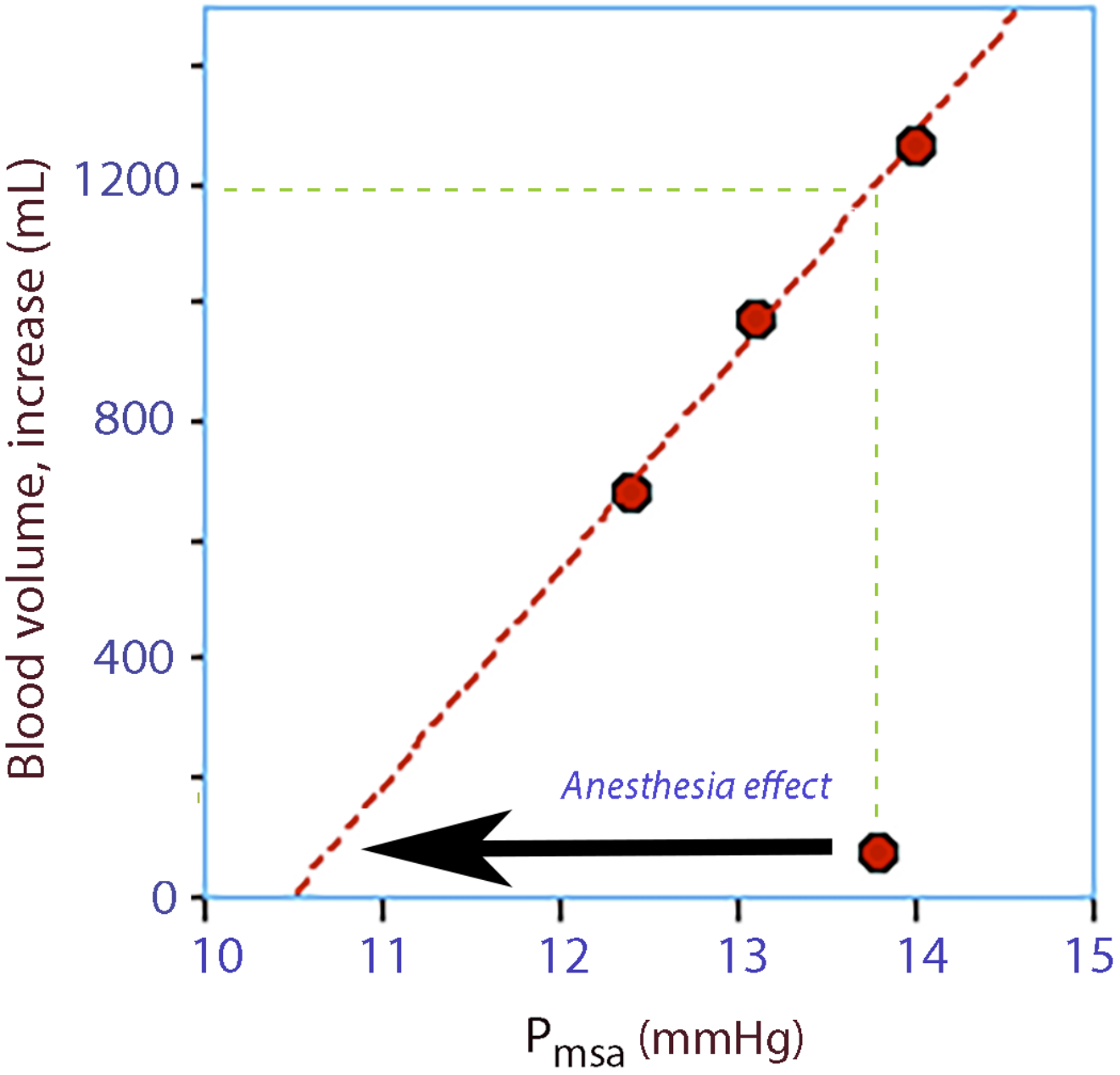

Figure 4

Increases in the "non-stressed blood volume" by general anesthesia. The plot shows the P_{msa} for three successive bolus infusions *versus* the gradual increase in blood volume. The arrow indicates the reduction of the P_{msa} by the induction general anesthesia. The distance between P_{msa} at baseline (before anesthesia) to the vascular compliance curve indicates the anesthesia-induced increase of the "unstressed" blood volume. Mean values for all patients were used.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- AdditionalFile1.xls
- AdditionalFile2.xls