Mean systemic filling pressure : from Guyton to the ICU

Maas, J.J.

Chapter 5

Estimation of mean systemic filling pressure in postoperative cardiac surgery patients with three methods

Jacinta J. Maas[1], Michael R. Pinsky[2], Bart F. Geerts[3], Rob B.P. de Wilde[1] and Jos R.C. Jansen[1]

[1]Department of Intensive Care Medicine, Leiden University Medical Center, The Netherlands,
[2]Department of Critical Care Medicine, University of Pittsburgh, PA, USA,
[3]Department of Anesthesiology, Leiden University Medical Center, The Netherlands

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Abstract

Effective circulating blood volume can be estimated by measuring mean systemic filling pressure. We assessed the level of agreement between different bedside estimates of mean systemic filling pressure (Pmsf), arm equilibrium pressure (Parm) and model analogue (Pmsa) in eleven mechanically ventilated postoperative cardiac surgery patients. Sequential measures were made in supine position, rotating the bed to 30° head-up tilt and after fluid loading (500 ml colloid). During each condition four inspiratory hold maneuvers were done to determine Pmsf, arm stop-flow was created by inflating a cuff around the upper arm for 30 seconds to measure Parm, and Pmsa was estimated from a Guytonian model of the systemic circulation. Mean Pmsf, Parm and Pmsa across all three states were 20.9 ± 5.6, 19.8 ± 5.7 and 15.9 ± 4.9 mmHg, respectively.

Bland-Altman analysis for the difference between Parm and Pmsf showed a non-significant bias of -1.0 ± 3.08 mmHg ($p = 0.062$), a coefficient of variation (COV) of 15% and limits of agreement (LOA) of -7.3 and 5.2 mmHg. For the difference between Pmsf and Pmsa we found a bias of -6.0 ± 3.1 mmHg ($p < 0.001$), COV 17% and LOA -12.4 and 0.3 mmHg. Changes in Pmsf and Parm and in Pmsf and Pmsa were directionally concordant in response to head-up tilt and volume loading. In conclusion, Parm and Pmsf are interchangeable. Changes in effective circulatory volume are tracked well by changes in Parm and Pmsa.

Introduction

Accurate assessment of cardiovascular state in the critically ill is difficult because easily measured parameters, such as blood pressure and cardiac output (CO), can co-exist with different levels of ventricular pump function and effective circulating blood volume. Thus, identifying the appropriate therapy and targeting specific measurable endpoints of therapy are problematic. Although assessing dynamic changes in arterial pulse pressure or left ventricular stroke volume during ventilation and passive leg-raising maneuvers improves identification of fluid responsiveness, they do not quantify effective circulating blood volume or the cause or lack thereof. Although fluid resuscitation therapy is important in the management of unstable patients, excessive fluid resuscitation can be harmful in acute lung injury[1], head injury[2] and postoperative patients.[3] Thus, a measure of effective volume status is useful to avoid volume overload since even volume-overloaded patients may remain volume responsive.

Mean systemic filling pressure (Pmsf) is a functional measure of effective intravascular volume status. It is the pressure anywhere in the circulation during circulatory arrest.[4] Importantly, central venous pressure (Pcv) to Pmsf pressure difference defines the driving pressure for venous return, and together with the resistance to venous return defines CO. We have shown that Pmsf can be measured in ventilator-dependent patients using inspiratory hold maneuvers defining Pcv-CO data pairs that when extrapolated to zero CO reports Pmsf.[5,6] This calculated Pmsf parameter accurately follows changes in intravascular volume.[5,7]

Unfortunately, this inspiratory hold technique requires a sedated and ventilated patient, not universally seen in critically ill patients. We thus studied two simpler bedside methods for determining Pmsf as previously suggested by Anderson[8] and Parkin.[9] Anderson hypothesized that the circulation of the arm behaves similar to total systemic circulation during steady-state conditions. Accordingly, we measured transient stop-flow forearm arterial and venous equilibrium pressure, referred to as arm equilibrium pressure (Parm). Parkin[9] proposed estimating the effective circulatory volume based on an electrical analog simplification of Guytonian circulatory physiology estimating mean circulatory pressure (Pmsa) from directly measured Pcv, mean arterial pressure and CO. The aim of our study was to compare the level of agreement between simultaneously measured Pmsf, Parm and Pmsa in three intravascular volume states in critically ill patients.

Materials and methods

The study was approved by the hospital ethics committee of Leiden University Medical Center (P01.111, 29 January 2002) and carried out in Leiden. Written informed consent was obtained from all patients prior to surgery. The institutional review board of University of Pittsburgh approved review and analysis of data. Eleven patients were enrolled and studied after cardiac surgery.

Patients

We limited our study to cardiac surgery patients requiring pulmonary artery and radial artery catheters for perioperative monitoring. Our study partially used hemodynamic data from the same patients reported in another study but examined different protocol-based measures.[7] All patients had coronary artery or valvular disease with preserved ventricular function (EFlv > 0.4). Patients with aortic aneurysm, severe peripheral vascular disease, postoperative arrhythmia, postoperative valvular insufficiency or needing artificial pacing or the use of a cardiac assist device were excluded. All subjects were studied during their initial postoperative period in the ICU, while sedated (propofol 3.0 $mg \cdot kg^{-1} \cdot h^{-1}$ and sufentanil 0.06-0.19 $\mu g \cdot kg^{-1} \cdot h^{-1}$) and mechanically ventilated with airway pressure release ventilation adjusted to achieve normocapnia, with 7-11 $ml \cdot kg^{-1}$ tidal volumes, 5 cmH_2O positive end-expiratory pressure, FiO_2 0.4 and f = 11-13 min^{-1} (Evita 4, Dräger AG, Lübeck, Germany). During the study interval all subjects were hemodynamically stable and no changes were made in their vasoactive drug therapy.

Measurements

All subjects also had a central venous catheter. Arterial pressure (Pa) and Pcv were recorded onto a computer for offline analysis. Pa and Pcv pressure transducers were referenced to the intersection of the anterior axillar line and the 5th intercostal space and re-referenced after a 30° head-up rotation. Airway pressure (Paw) was measured at the proximal end of the endotracheal tube. Beat-to-beat cardiac output (CO) was obtained by Modelflow pulse contour analysis as previously described by us.[10-12] We calibrated the pulse contour CO measurements with 3 therm odilution CO measurements equally spread over the ventilatory cycle.[11]

We have previously described the inspiratory hold method for estimating Pmsf.[5] Briefly, four 12-second inspiratory holds were applied at Paw of 5, 15, 25 and 35 cmH_2O respectively. The resulting Pcv and CO were measured during the plateau phase (between 7-12 seconds of each inspiratory hold maneuver), and the zero CO intercept of the Pcv and CO pairs estimated Pmsf.

Parm estimates of Pmsf[8] assumes Pa and Pv equilibrium following rapid vascular occlusion. We performed a pilot study in nine patients after either cardiac surgery or cardiopulmonary resuscitation to determine the stop-flow time. We measured arterial and venous pressures in the same hand and created upper extremity blood stop-flow using a rapid cuff inflator (Hokanson E20, Bellevue, Washington) to pressures 50 mmHg above systolic pressure and held occlusion for 35-60 seconds (figure 5.1). Measurements were performed three times to assess repeatability (table 5.1). Arterial and venous pressures equilibrated after 25-30 seconds of stop-flow, with a mean difference of -0.73 ± 1.07 mmHg at 30 seconds. Thus, we chose the 30-second value of the arterial pressure for Parm for the present study.

The Pmsa estimate[9] uses a mathematical model of the systemic circulation comprising compliant arterial and venous compartments and resistances to blood flow. The model

parameters are adjusted to match those of the patient's current measured variables, such that Pmsa = a•Pcv + b•Pa + c•CO, where a and b are dimensionless constants (a + b = 1, typically a = 0.96, b = 0.04) and c has the dimensions of resistance and is a function of patient's height, weight and age.

$$c=0.038\bullet(94.17+0.193\bullet age)/(4.5\bullet[0.99^{(age-15)}]\bullet 0.007184\bullet [height^{0.725}]\bullet[weight^{0.425}])$$

Protocol

Measurements were carried out within 2 hours of arrival in the ICU following initial hemodynamic stabilization. To induce changes in volume status, measurements were performed in supine position (baseline), in a 30° head-up tilt (HUT) and again in supine position after 500 ml hydroxyethylstarch (HES 130/0.4) rapid fluid administration (VOL). Measurements of Pa, Pv, Pcv, CO were done during baseline in supine position, 2 minutes after change to HUT and 2-5 minutes after fluid loading with Pmsf, Parm and Pmsa calculated for each step. Repeatability of Parm was determined by two measurements during baseline and after VOL. The study protocol lasted about 60 minutes. All patients completed all steps of the protocol and there were no adverse events.

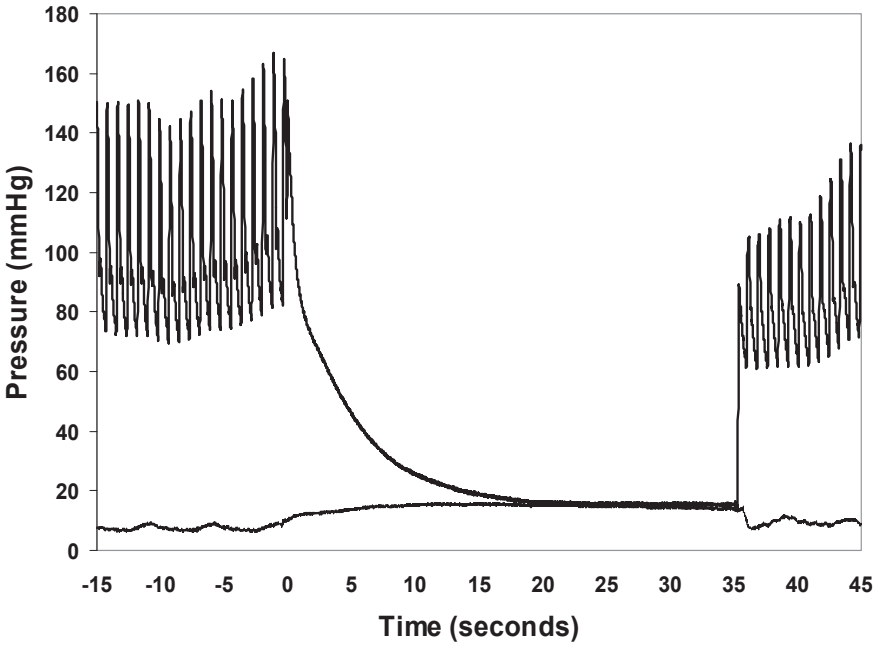

Figure 5.1 Example of an inspiratory hold maneuver

Representative registration of radial artery pressure and venous pressure before (–15 to 0 seconds), during (0 to 36 seconds) and after the occlusion of the upper arm of a patient. Arm vascular occlusion equilibrium pressure (Parm) is taken as the arterial pressure 30 seconds after stop-flow. Note the influence of mechanical ventilation on arterial and venous pressure before and after occlusion.

Statistical analysis

After confirming normal distribution of data with the Kolmogorov–Smirnov test, differences among Pmsf, Parm and Pmsa during baseline, HUT and VOL were analyzed

using paired t-tests. Calculations of bias, precision and limits of agreement (LOA) between Pmsf and both Parm and Pmsa were performed using Bland-Altman analysis with bias reflecting the mean difference between Pmsf and either Parm or Pmsa and precision as the standard deviation (SD) of these differences. After adjustment for the number of observations (n = 33) LOA are defined as bias ± 2.04 • SD. For repeatability of Parm (n = 40) LOA are bias ± 2.02 • SD. The coefficient of variation (COV) is calculated as 100% • SD/mean. Repeatability of Parm was calculated by Bland-Altman analysis using duplicate measurements at baseline and after VOL, which were pooled together. A p-value < 0.05 was considered statistically significant. Unless otherwise stated, data are presented as mean ± SD.

Table 5.1 Pilot study arm equilibrium pressure

Time	Pa			Pv			Pa-Pv		
	Mean	SD	Repeat	Mean	SD	Repeat	Mean	SD	Repeat
sec	mmHg	mmHg	%	mmHg	mmHg	%	mmHg	mmHg	%
15	23.32	2.41	5.45	21.96	2.05	9.20	1.35	2.69	4.89
20	22.11	1.88	6.11	22.12	2.02	9.58	-0.01	1.62	5.52
25	21.42	1.56	6.91	22.06	1.91	9.79	-0.63	1.02	5.18
30	21.08	1.38	6.55	21.81	2.05	9.58	-0.73	1.07	4.55

Effect of time on arterial pressure (Pa), venous pressure (Pv) and the difference between Pa and Pv during upper arm stop-flow. The results of a pilot study in 9 patients are indicated. Repeat, the averaged repeatability of three sequential measurements and SD, standard deviation.

Results

Patient characteristics are presented in table 5.2 and mean hemodynamic data for the protocol in table 5.3. Mean Pa decreased during HUT and was unchanged with VOL. Pcv, CO, Pmsf, Parm and Pmsa decreased during HUT and increased with VOL.
Pmsf, Parm and Pmsa decreased in all patients during HUT (3.4 ± 2.6, 3.0 ± 2.0 and 3.7 ± 2.3 mmHg, p < 0.001, p = 0.001 respectively). VOL was associated with an increase in Pmsf, Parm and Pmsa (8.7 ± 5.3, 8.7 ± 3.8 and 4.5 ± 2.1 mmHg, p < 0.001 all, respectively). Parm was not different from the Pmsf during baseline, HUT or VOL (p = 0.236, p = 0.423 and p = 0.173 respectively). However, Pmsf and Pmsa differed significantly for the three conditions (p < 0.001 all). Pmsf regressed significantly with Parm (figure 5.2A) (slope = 0.944, correlation coefficient (R) = 0.847) and Pmsa (figure 5.2B) (slope = 0.704, R = 0.822).

Baseline Pmsf and Parm did not correlate with Pcv, Pa and pulse pressure. Baseline Pmsa correlated with Pcv (Pearson correlation coefficient R = 0.846, p = 0.001) and with pulse pressure (R = 0.697, p = 0.017). Pmsa did not correlate with mean, systolic and diastolic arterial pressure (p > 0.28 for all).
For the changes in Pmsf, Parm and Pmsa induced by HUT only Pmsa correlated significantly with changes in Pcv (R = 0.931, p < 0.001). For the changes induced by

VOL both Pmsf and Pmsa correlated with changes in Pcv (R = 0.781, p = 0.005 and R = 0.911, p < 0.001). No significant correlation was found with changes in Pa or pulse pressure for changes in Pmsf, Parm and Pmsa.

Table 5.2 Patient Characteristics

		Mean	Range
Age (years)		64	50-80
Gender		9 male, 2 female	
Weight (kg)		86	73-112
Length (cm)		174	158-190
Surgery			
	CABG	9	
	AVR	2	
Respiratory rate (min^{-1})		12	11-13
Tidal volume/predicted ($ml \bullet kg^{-1}$)		9	7-11
PEEP (cm H_2O)		5	

		Number of patients	Range dose ($\mu g \bullet kg^{-1} \bullet min^{-1}$)
Vasoactive medication			
	Dobutamine	4	2-4
	Enoximone	1	2
	Norepinephrine	5	0.01-0.09
	Sodium nitroprusside	1	0.25

CABG, coronary artery bypass grafting; AVR, aortic valve replacement

Table 5.3 Hemodynamic data of patients during baseline, head-up tilt and fluid loading

	Baseline		HUT			+ 500 ml		
	Mean	SD	Mean	SD	p1	Mean	SD	p2
Pa (mmHg)	88.8	17.9	77.3	17.0	< 0.001	97.9	15.3	0.003
Psys (mmHg)	128.5	21.9	107.2	16.9	0.001	143.3	17.7	0.004
Pdias (mmHg)	69.0	17.7	62.4	17.9	0.001	75.2	15.6	0.040
PP (mmHg)	59.5	14.7	44.8	9.9	0.016	68.1	12.1	0.076
Pcv (mmHg)	7.1	2.0	4.4	1.8	0.001	10.4	1.3	0.001
CO ($l \bullet min^{-1}$)	5.8	1.6	4.8	1.2	0.006	7.0	1.7	0.004
HR (min^{-1})	88	14	87	15	0.574	86	10	0.475
Pmsf (mmHg)	19.7	3.9	16.2	3.0	0.001	28.3	3.6	< 0.001
Parm (mmHg)	18.4	3.7	15.4	3.1	0.001	27.1	4.0	< 0.001
Pmsa (mmHg)	14.7	2.7	10.9	2.0	< 0.001	19.2	1.1	< 0.001

Values are means ± SD; n = 11 patients. Pa, mean arterial pressure; Psys, systolic arterial pressure; Pdias, diastolic arterial pressure; PP, pulse pressure; Pcv, central venous pressure; CO, cardiac output; HR, heart rate; Pmsf, mean systemic filling, pressure; Parm arm equilibrium pressure; Pmsa, model analogue mean circulatory pressure. Statistical comparison, p1, paired t-test between baseline and head-up tilt condition (HUT) and p2, paired t-test between baseline and after fluid loading condition (+ 500 ml).

Agreement of methods

For all measurements Pmsf and Parm displayed a non-significant bias of -1.0 ± 3.08 mmHg (p = 0.062), COV of 15% and with LOA of -7.3 and 5.2 mmHg (figure 5.2B). The biases for Pmsf and Parm were: baseline -1.3 ± 3.4, HUT -0.8 ± 3.2, VOL -1.2 ±

2.8 mmHg. For all measurements Pmsf and Pmsa displayed a bias of -6.0 ± 3.1 mmHg ($p < 0.001$), COV of 17% and LOA of -12.4 and 0.3 mmHg (figure 5.3B). The biases for Pmsf and Pmsa were: baseline -5.0 ± 2.8, HUT -5.3 ± 3.2, VOL -8.1 ± 2.7 mmHg. Mean Pmsf, Parm and Pmsa across all three states were 20.9 ± 5.6, 19.8 ± 5.7 and 14.9 ± 4.0 mmHg, respectively.

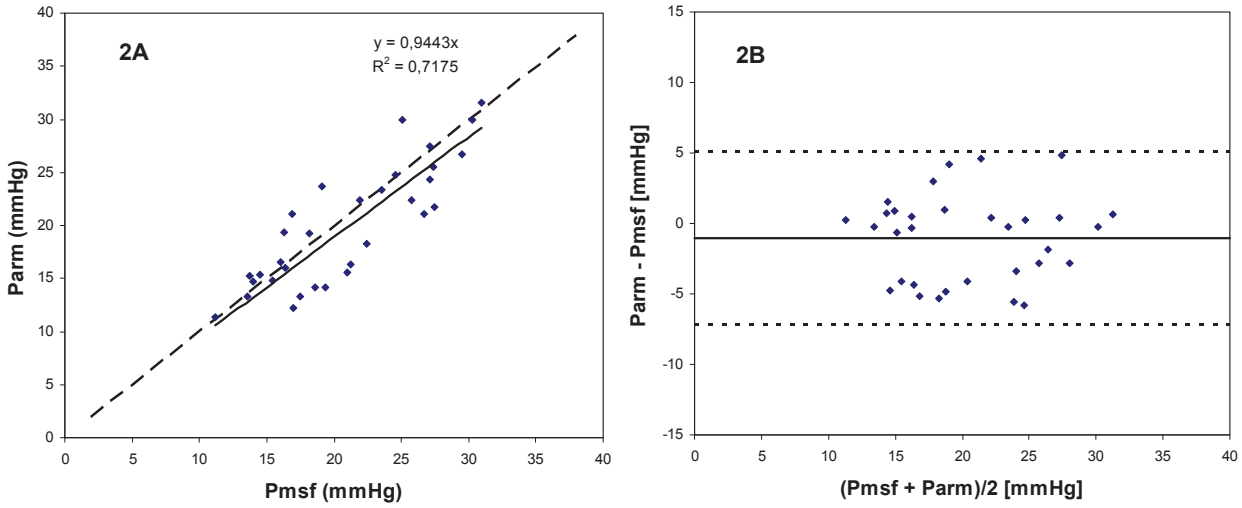

Figure 5.2 Regression (A) and Bland-Altman analysis (B) of arm equilibrium pressure (Parm) and mean systemic filling pressure (Pmsf).
In panel A, the solid line is the regression line and the dashed line is the line of identity. In Panel B, the solid line indicates the bias and the dashed lines are the limits of agreement.

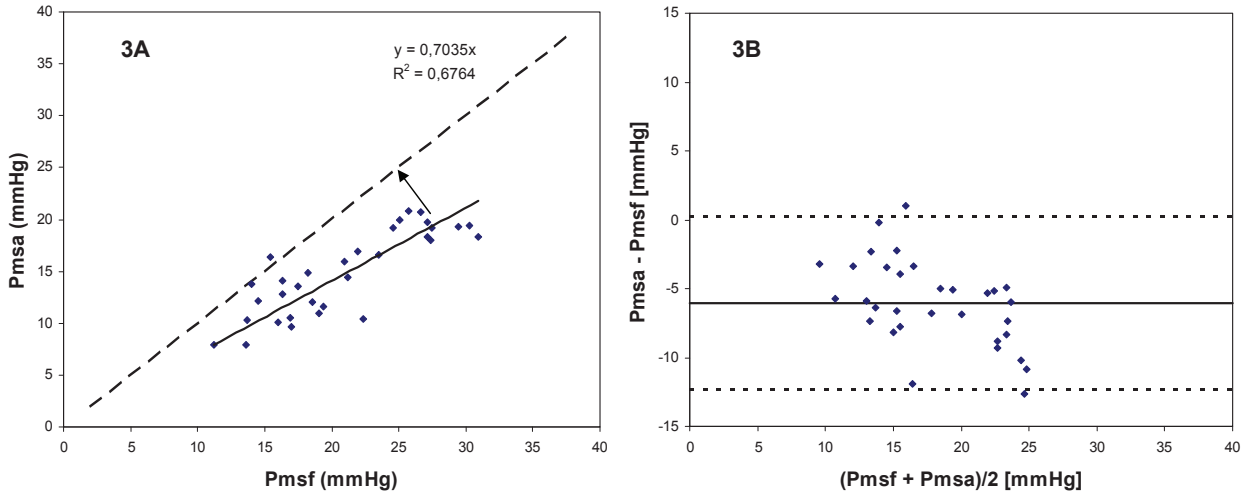

Figure 5.3 Regression (A) and Bland-Altman analysis (B) of model analogue pressure (Pmsa) and mean systemic filling pressure (Pmsf).
In panel A, the solid line is the regression line and the dashed line is the line of identity. In Panel B, the solid line indicates the bias and the dashed lines are the limits of agreement.

Changes of Parm (∆Parm) and Pmsa (∆Pmsa) versus changes in Pmsf (∆Pmsf) are shown in figure 5.4. Both ∆Parm and ∆Pmsa regressed significantly ($p < 0.001$) with ∆Pmsf (slope = 0.85, R = 0.896 and slope = 0.53, R = 0.871, respectively). The cross

tabulation agreement of positive and negative changes in each of the methods for HUT and VOL displayed directionally balanced concordance for all data pairs for both ΔParm and ΔPmsa versus ΔPmsf.

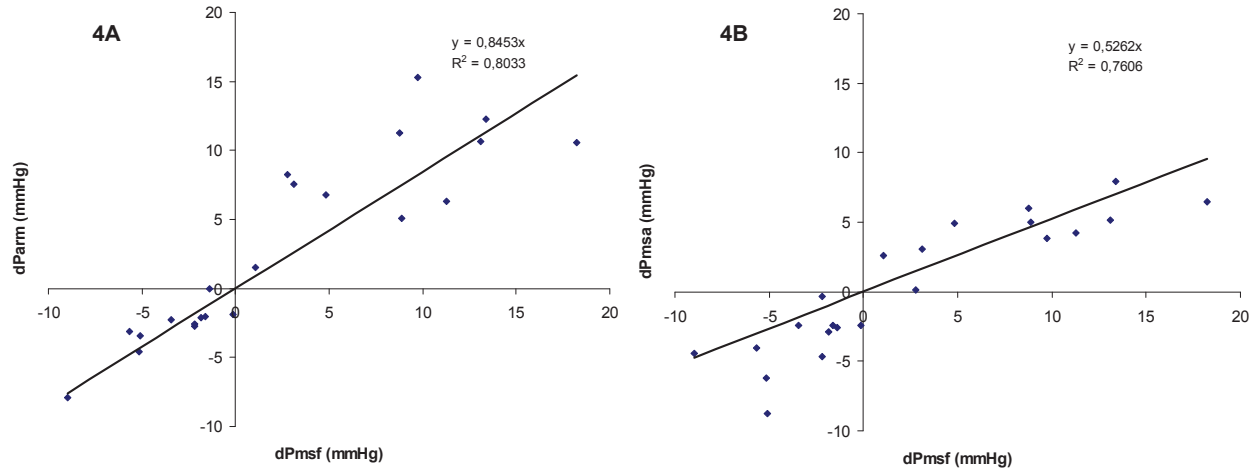

Figure 5.4 Changes in mean systemic filling pressures.
Changes in mean systemic filling pressure by arm equilibrium pressure (Parm) (A) and by model analogue (Pmsa) (B) plotted against changes in mean systemic filling pressure by inspiratory hold procedures (Pmsf). The regression line is indicated by a solid line.

Repeatability of Parm

Bland-Altman analysis for Parm duplicate measurements during both baseline and VOL revealed a bias of 0.03 ± 1.02 mmHg, LOA from -2.04 to 2.09 mmHg and COV of 5%. No difference was found between the first and second of the duplicate Parm measurements (p = 0.915).

Discussion

Our study demonstrates that estimates of Pmsf measured 30 seconds after arm stop-flow (Parm) are interchangeable with Pmsf calculated using inspiratory hold maneuvers in mechanically ventilated postoperative cardiac surgery patients. Furthermore, changes in volume status by HUT and VOL are similarly tracked by Pmsf, Parm and Pmsa. These data support the hypothesis formulated, but not previously validated, by Anderson[8], that during steady-state flow conditions the arm is representative of the entire circulation, such that a rapid vascular occlusion will result in its stop-flow Pa approximating Pmsf. Thus, both Pmsf and Parm can be used at the bedside to measure effective circulating blood volume. Furthermore, Pmsa can reliably tract changes in effective circulating blood volume status.

The use of both Parm and Pmsa has practical advantages over our previously validated inspiratory hold maneuver Pmsf approach. Neither requires positive-pressure breathing or multiple simultaneous measures of Pcv and CO during inspiratory hold maneuvers, and both can be rapidly and repeatedly measured sequentially as treatment or time

progresses. Parm requires only the peripheral arterial catheter. Pmsa requires both central venous and peripheral arterial catheters. Thus, these two novel approaches markedly increase the applicability of assessment of effective circulating blood volume to a broader patient population.

Methodological consideration

Radial artery pressure. Shortly after cardiopulmonary bypass, radial artery pressure can be significantly less than aortic pressure[13-15], but this difference disappears after about 60 minutes, coinciding with hemodynamic stabilization.[13] Our study started after approximately 2 hours after cardiopulmonary bypass in stable patients. Therefore, we believe that mean radial artery pressure reliably reflected central aortic pressure. We recently documented in a porcine model of acute endotoxemia[16] that similar central to regional arterial pulse pressure changes occur. However, the value of Pmsf is not dependent on the calibration of the pulse contour method as long as a linear change in CO is followed by a linear change in CO derived from pulse contour. Indeed, Pcv at CO equal to zero is not even influenced by a calibration factor.

Arm stop-flow procedure. In the pilot stop-flow study described above, we observed that a plateau pressure developed in both arterial and venous pressures after 20-30 seconds as predicted by Anderson.[8] However, a further decrement in both Pa and Pv developed after 35-40 seconds, indicating probable hypoxia-induced vasodilation. We also observed the best repeatability and lowest standard deviations between the arterial and venous pressure at 25-30 seconds of stop-flow, which was the time we used in this study. Furthermore, stop-flow durations longer than 5 minutes are needed to produce reactive hyperemia in the human forearm.[17,18] Thus, if stop-flow maneuvers are limited to < 1 minute, regional blood flow will also normalize after an additional 1 minute.[19] The rapid cuff inflator inflates in less than 0.3 seconds.[20] In this time there is only a brief cessation of venous return prior to arterial stop-flow equal to approximately one heart beat. We expect this overestimation to be negligible because the amount of inflow is small compared to the total distal arm blood volume. Finally, since longer vascular occlusion maneuvers are routinely used to assess dynamic changes in tissue O_2 saturation without complications[21], we feel that this much shorter vascular occlusion maneuver is safe.

Model analogue Pmsa. No clinical evaluation of Pmsa against other methods to measure Pmsf has been done so far. The validity of the Pmsa algorithm was successfully tested using a closed loop control of fluid replacement during continuous hemodiafiltration.[22] Our data support these findings because ∆Pmsf and ∆Pmsa faithfully track each other.

Pmsf. We showed that Modelflow pulse contour CO was interchangeable with pulmonary artery and aortic flow probe derived CO in swine[23], and that Modelflow-

derived Pmsf was interchangeable with flow probe-derived Pmsf with a COV for duplicate measurements of 6.1%. Still, we report mean baseline Pmsf values of 19.7 mmHg in our cardiac surgical patients, which are higher than Pmsf values reported between 7-12 mmHg in animal studies.[24-27] Using the same inspiratory hold technique and pulse contour analysis we found Pmsf values of 10.38 ± 1.09 mmHg in pigs.[23] A primary difference between the prior animal studies and our patient observations is the difference in baseline Pcv. In the animals studies this value is close to zero whereas Pcv in our patient population is on average 7.1 mmHg. The pressure gradient for venous return (Pmsf minus Pcv) in our study (12-13 mmHg) is therefore similar to the pressure gradient for venous return in the animal studies. Thus, our Pmsf values are coupled with the increased Pcv. However, high values of Pmsf still predispose to peripheral edema formation.

Jellinek *et al*.[28] and Schipke *et al*.[29] estimated Pmsf in patients during episodes of apnea and ventricular fibrillation, and found a mean Pmsf value of 10.2 mmHg and 12 mmHg, respectively. However, both studies were done on highly anesthetized non-volume resuscitated subjects. Our method of estimation of Pmsf differs considerably from stopping flow by ventricular fibrillation, and our method allows an estimation of Pmsf with intact circulation, applicable in the intensive care unit.[5,30]

Agreement between Parm, Pmsa and Pmsf. We found agreement between Pmsf and Parm (figure 5.2) and ∆Pmsf and ∆Parm were concordant in all interventions (figure 5.4). Therefore, both methods should equally measure and follow changes in effective circulating blood volume. There was poor agreement between Pmsa and Pmsf. The large bias makes the methods non-interchangeable. However, the full concordance between ΔPmsf and ΔPmsa indicates that the Pmsa method is very applicable to track changes in effective circulating blood volume, as indeed was documented by Parkin *et al*. in dialysis patients.[22]

Finally, effective circulating blood volume is a functional measure, not an absolute one. In our study the vasoactive medication was not changed. Changing vasomotor tone will alter unstressed volume, stressed volume and compliance. Any treatment that alters unstressed volume will also alter effective circulating blood volume independent of changes in blood volume, as was demonstrated by Guyton *et al*.[4]

Can either Parm or Pmsa replace the Pmsf method in the bedside assessment of effective circulating blood volume? Based on the established argument of Critchley and Critchley[31], a new method may replace an older established method if the new method itself has errors not greater than the older method. The Parm method showed a non-significant bias when compared to Pmsf. A single measurement of Pmsf has a COV of about 6%.[23] We found a 5% repeatability for Parm. Thus, our data support the argument that Parm may replace inspiratory hold-maneuver generated Pmsf.

A significant bias ($p < 0.001$) was observed between Pmsf and Pmsa, precluding the substitution of raw Pmsa values for Pmsf. However, based on the linearity of Pmsf and

Pmsa (figure 5.3A) one can adjust the Pmsa values using a calibration factor of 1.42 (i.e. the reciprocal of the slope of the regression 0.704). After this calibration is applied to Pmsa values, indicated in figure 5.3A by an arrow from the regression line to the line of identity, the bias reduces to zero. The expected precision of the calculation of Pmsa is approximately 10% (this COV is largely caused by the COV in Pcv measurement, a value of 10 mmHg can be 9.51 or 10.49 mmHg). Although this 10% is higher than the 6% for Pmsf, after recalibration the Pmsa model analogue may replace Pmsf. It must be emphasized that the correction factor only describes our postoperative cardiac surgery population and will require similar validation in other patient groups.

Study limitations. The number of patients included in the study is relatively low. However, we still found a significant difference between Pmsa and Pmsf. With a larger study population the difference between Pmsf and Parm could have become significant. However, the absolute difference of -1.0 mmHg is not clinically relevant. We included patients with preserved left ventricular function, after relatively simple cardiac surgery, and excluded patients with previous myocardial infarction and/or congestive heart failure (New York Heart Association class 4). These patients are known to have markedly increased vascular tone with an associated decreased proportional unstressed vascular volume. Thus, caution needs to be used when extrapolating the accuracy of these comparisons to other patients groups. During the study, we made no changes in medication. Therefore, we cannot report on the values and comparison of Pmsf, Parm and Pmsa during changes in vasoactive medication, which influences vascular elastance, resistance and conductance properties. A fundamental limitation of the Parm technique is the need to measure arterial pressure from a radial arterial site. In patients with sepsis or on high levels of vasopressors, radial artery compliance may not reflect central arterial compliance, although mean Pa remains accurate.[16] Therefore, in these patients it is not clear if Parm or Pmsa will tract Pmsf. Still, under those conditions, the diagnosis of decreased effective circulating blood volume is rarely an issue.

Conclusions

The equilibrium pressure in the arm during stop-flow (Parm) and inspiratory hold maneuver-derived Pmsf values are interchangeable in mechanically ventilated postoperative cardiac surgery patients. Thus, the mean systemic filling pressure can be simply measured at the bedside by measuring arterial pressure during upper arm stop-flow, without the need of inspiratory hold maneuvers or central venous or pulmonary artery catheters. Furthermore, changes in effective circulatory volume are accurately trended by changes in both Parm and Pmsa.

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