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Afterload Assessment With or Without Central Venous Pressure: A Preliminary Clinical Comparison

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Abstract A clinical comparison, of two methods of afterload assessment, has been made. The first method, systemic vascular resistance index (SVR_i), is based upon the traditional formula for afterload which utilizes central venous pressure (CVP), as well as cardiac index (C_i), and mean arterial blood pressure (MAP). The second method, total systemic vascular resistance index ($TSVR_i$), also uses MAP and C_i. However, $TSVR_i$ ignores the contribution of CVP. This preliminary examination, of 10 randomly-selected ICU patients, has shown a high degree of correlation (ranging from 90 to 100%) between SVR_i and $TSVR_i$ ($P < 0.0001$). Furthermore, there was also a high degree of correlation (ranging from 94 to 100%) noted between the hour-to-hour change in SVR_i with the hour-to-hour change in $TSVR_i$ ($P < 0.0001$). The results, of this pilot study, support the premise that the use of CVP may not always be necessary for afterload evaluation in the clinical setting. Minimally-invasive means of measuring both C_i and MAP, without CVP, may be adequate for use in assessing afterload.

Keywords Systemic vascular resistance index · Total systemic vascular resistance index · Central venous pressure · Pulmonary artery occlusion catheter

Introduction

Examining SVR_i

Clinicians have typically used systemic vascular resistance index (SVR_i) as a measure of afterload in the management of critically ill patients (Melo and Peters 1999) as well as those undergoing extensive cardiac, vascular, and thoracic surgical procedures (Barash et al. 2009). SVR_i is defined as (Li 2000, 2004):

$$SVR_i = \frac{(MAP - CVP)}{C_i} \cdot 80 \quad (1)$$

where MAP is mean arterial pressure, CVP is central venous pressure, and C_i is cardiac index. Note that the constant 80 allows for SVR_i to be expressed with the dimensions of: dyne s cm^{-5} m^{-2} or "resistance units". *A list of abbreviations, dimensions, and associated terminology is shown in* Table 1.

Traditionally, the determination of SVR_i has required invasive hemodynamic monitoring with a pulmonary artery occlusion catheter (PAOC) to provide measurements of both C_i and CVP. Note that MAP can be obtained noninvasively, with a blood pressure cuff, or in a minimally-invasive manner with a peripherally-placed arterial catheter.[1]

[1] Typically, the radial artery is used for this purpose.

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Table 1 List of abbreviations, dimensions, and terminology

Abbreviation	Meaning	Dimensions
BM_i	Body mass index[a]	kg m^{-2}
CAD	Coronary artery disease	
C_i	Cardiac index[b]	l min^{-1} m^{-2}
CVP	Central venous pressure	mmHg
DBP	Diastolic blood pressure	mmHg
DM	Diabetes mellitus	
EDM	Esophageal Doppler monitor	
EF	Ejection fraction	%
HTN	Hypertension	
MAP	Mean arterial pressure	mmHg
OSA	Obstructive sleep apnea	
PAOC	Pulmonary artery occlusion catheter	
SBP	Systolic blood pressure	mmHg
SVR_i	Systemic vascular resistance index[b]	dyne s cm^{-5} m^{-2} (resistance units)
$TSVR_i$	Total systemic vascular resistance index[b]	dyne s cm^{-5} m^{-2} (resistance units)

[a] Based upon patient height

[b] Based upon patient body surface area

The clinical utility of PAOCs has been questioned as outcome studies have generally not supported their use (ASA 2003; Sandham et al. 2003; NHLBI 2006; Warzawski et al. 2003). Furthermore, minimally-invasive and non-invasive means of measuring C_i have emerged. These technologies include the esophageal Doppler monitor (EDM) or CardioQ® (Deltex Medical, UK) and the FloTrac® (Edwards Lifesciences, USA).[2] Note that the EDM determines C_i using the velocity of blood flow in the distal aorta (Dark and Singer 2004). Whereas the FlowTrac® requires a peripheral arterial catheter to measure pulse pressure and uses a "pulse contour analysis" method to estimate cardiac output (Manecke 2005). The assumptions made with pulse contour analysis may be erroneous as clinical comparisons, of this device with the EDM, have demonstrated significant inconsistencies in stroke volume determination (Chatti et al. 2009).

Measurement of the change in electrical impedance or "bioimpedance" of the thorax, or whole-body, as a function of a time, has also been developed to non-invasively assess C_i (Barin et al. 2000; Ouzounian et al. 1996). Unfortunately, this method has also demonstrated inconsistent correlation with traditional methods of measuring cardiac output (Imhoff et al. 2000; Simon et al. 2009).

None of these minimally-invasive techniques, of C_i evaluation, allow for CVP measurement. An ideal method of afterload assessment would therefore also be minimally invasive and allow for *continuous* measurements. It should be noted that PAOCs only allow for determination of SVR_i on an *intermittent* basis.

[2] Atys Medical (France) also manufactures an EDM. However, it is not currently available in the USA.

Examining $TSVR_i$

Another method of afterload assessment can be defined as total systemic vascular resistance ($TSVR_i$) in which the contribution of CVP is ignored (Wang et al. 2008; Atlas 2008):

$$TSVR_i = \frac{MAP}{C_i} \cdot 80 \tag{2}$$

Thus, the purpose of this study was to assess the correlation of SVR_i to $TSVR_i$ using the data acquired from PAOCs. Therefore, the contribution of CVP, to afterload assessment, could be made. In addition, the hour-to-hour changes in both SVR_i and $TSVR_i$ were also examined. This was done to determine how changes in CVP effect changes in afterload.

Ultimately, the use of minimally-invasive and non-invasive devices could possibly be utilized, for the assessment of afterload, without the potential complications associated with pulmonary artery cannulation. These complications are summarized in Table 2 (ASA 2003).

Differential Analysis

A differential examination of both SVR_i and $TSVR_i$ was also made to assess the hour-to-hour changes in these values. It should be noted that clinicians frequently examine the "trend" in afterload over time. Thus, the hour-to-hour changes in SVR_i and $TSVR_i$ are defined as:

$$\Delta SVR_i(k) = SVR_i(k+1) - SVR_i(k), \quad k = 1, 2 \ldots (N-1) \tag{3}$$

and

Table 2 List of complications associated with PAOCs (ASA 2003)

Complication	Incidence in most studies (%)
Arterial puncture	≤3.6
Pneumothorax	0.3–1.9
Minor dysrthymias	>20
Severe dysrthymias (ventricular tachycardia or fibrillation)	0.3–3.8
Pulmonary artery rupture	0.03–0.7
Positive catheter-tip cultures	≥19
Catheter-related sepsis	0.7–3.0
Venous thrombosis	0.5–3.0
Pulmonary infarction	0.1–2.6
Valvular/endocardial vegetations or endocarditis	2.2–7.1

$$\Delta \mathrm{TSVR}_i(k) = \mathrm{TSVR}_i(k+1) - \mathrm{TSVR}_i(k), \quad k = 1, 2 \ldots (N-1) \tag{4}$$

Note that k ranges from the first hour to the hour preceding the final hour $(N-1)$. Where N represents the final hour. The mathematical relationship, between $\Delta \mathrm{SVR}_i$ and $\Delta \mathrm{TSVR}_i$, using both an algebraic method as well as the definition of the total differential, is documented in the Appendix.

Methods

Data Acquisition and Analysis

After institutional review board approval, the medical records of 10 randomly-selected patients, whose treatment had included indwelling PAOCs, were examined. All patients had been admitted to the Cardiothoracic Intensive Care Unit (CTICU) at University Hospital/Newark. Note that this study was strictly a retrospective chart review of preexisting data. Each patient's demographic information is shown in Table 3.

Hemodynamic data, from all 10 patients, were initially obtained each hour for at least 24 h. These data included: C_i, CVP, and MAP. Note that MAP was determined from either an indwelling arterial catheter or a non-invasive blood pressure cuff. For the purposes of this study, MAP was calculated using (Li 2000, 2004):

$$\mathrm{MAP} = \frac{2}{3}\mathrm{DBP} + \frac{1}{3}\mathrm{SBP} \tag{5}$$

As this was a preliminary or "pilot" study, no attempt to stratify data, based upon patient acuity, severity, or survival, was made. Furthermore, the hemodynamic influence, of any of each patient's medications, such as sedatives or vasoactive medications, was also not included.

SVR_i and TSVR_i were calculated, for each hour, using Eqs. 1 and 2 respectively. Similarly, $\Delta \mathrm{SVR}_i$ and $\Delta \mathrm{TSVR}_i$ were also calculated, on an hourly basis, using Eqs. 3 and 4.

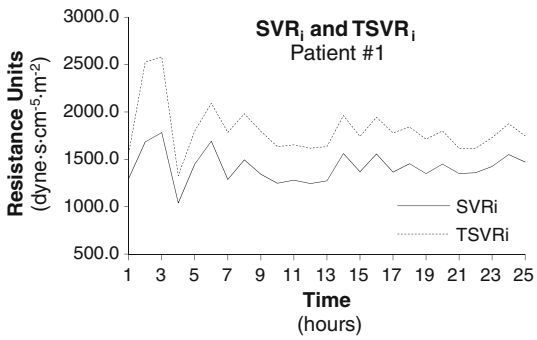

Fig. 1 SVR_i and TSVR_i calculated, on an hourly basis, using the data from patient #1

Table 3 Demographic data of the ten randomly-selected cardiothoracic ICU patients

Patient	Age (years)	Gender	Weight (kg)	Height (m)	BM_i (kg m^{-2})	HTN	CAD	DM	Tobacco use	OSA	EF	Procedure
1	46	Male	116	1.85	33.64	+	+	+			50%	Ascending aortic aneurysm
2	65	Male	124	1.80	38.07	+		+		+	55–60%	Esophagectomy
3	64	Male	79	1.72	26.30	+	+		+		55%	Coronary artery bypass graft(s)/mitral valve replacement
4	76	Male	73	1.70	25.06	+	+	+	+		75%	Coronary artery bypass graft(s)
5	72	Female	57	1.57	22.86	+		+	+		70%	Aortic valve replacement
6	47	Female	94	1.65	34.61	+	+	+			25–30%	Coronary artery bypass graft(s)/mitral valve replacement
7	53	Male	104	1.52	44.91	+		+		+	56–60%	Mitral valve repair
8	70	Male	106	1.72	35.58	+	+	+	+		50%	Coronary artery bypass graft(s)
9	62	Male	70	1.72	23.41		+		+		40–45%	Coronary artery bypass graft(s)
10	57	Female	80	1.75	25.99	+	+		+		50%	Mitral valve replacement

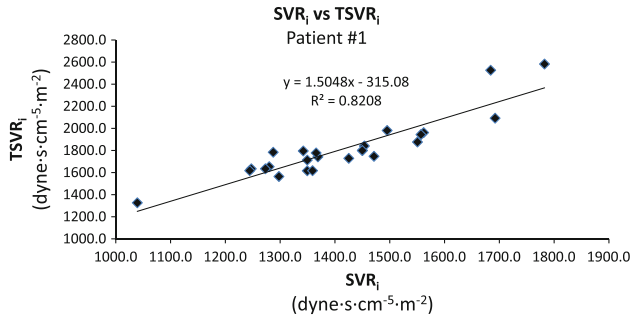

Fig. 2 A linear correlation, between SVR_i and $TSVR_i$, is illustrated using the data from patient #1

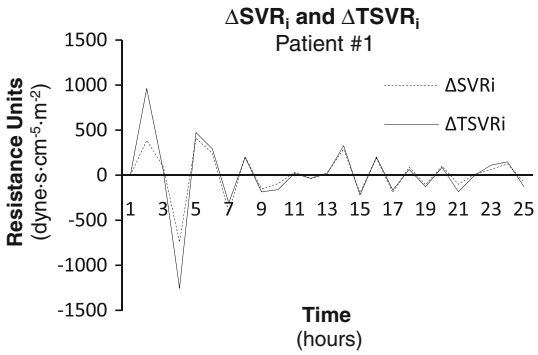

Fig. 3 Hour-to-hour changes in SVR_i and $TSVR_i$ using the data from patient #1. These changes are referred to as ΔSVR_i and $\Delta TSVR_i$ respectively

Statistical analysis was accomplished, on a patient-by-patient basis, using a Pearson product-moment correlation coefficient (Kutner et al. 2004; Kreyszig 1999). In addition, a Fisher's z-transformation was also used to determine statistical significance, and associated confidence intervals, from the correlation coefficients derived from each patient's data (Hawkins 1989). Furthermore, it was assumed that all data points were from a bivariate normal distribution.

Results

Based upon this preliminary study, SVR_i and $TSVR_i$ are closely correlated. This is exemplified in both Figs. 1 and 2 which graphically demonstrate the data from patient #1. The correlation coefficients, from all ten patients' data, ranged from 90 to 100%. Furthermore, all correlation coefficients were statistically significant ($P < 0.0001$).

Similarly, ΔSVR_i and $\Delta TSVR_i$, which represent the hour-to-hour changes in SVR_i and $TSVR_i$, were also highly correlated. These correlation coefficients ranged from 94 to 100% ($P < 0.0001$). Figures 3 and 4, which are also based upon the data from patient #1, illustrate this relationship.

All correlation coefficients and their associated 95% confidence intervals are documented in Table 4.

Discussion

The assessment of afterload remains an important aspect of hemodynamic monitoring during anesthesia and critical care situations. Specifically, clinical conditions such as: septicemia, adrenal insufficiency, and pancreatitis frequently require afterload measurement (Melo and Peters 1999). In addition, cardiac, thoracic, and vascular surgical procedures also may necessitate intraoperative and postoperative afterload assessment (Barash et al. 2009).

Furthermore, severely injured patients, particularly those with neurogenic shock (Bilello et al. 2003) or with systemic inflammatory response syndrome (SIRS) (Cremer et al. 1996; Carrel et al. 2000; Kohsaka et al. 2005), may require afterload measurements. Other conditions requiring possible afterload assessment include: hypertensive crisis, congestive heart failure, aortic dissection, hepatic transplantation, burn injury, and the management of pheochromocytoma (Barash et al. 2009).

Fig. 4 A linear correlation, between $\Delta TSVR_i$ and ΔSVR_i, is documented using the data from patient #1

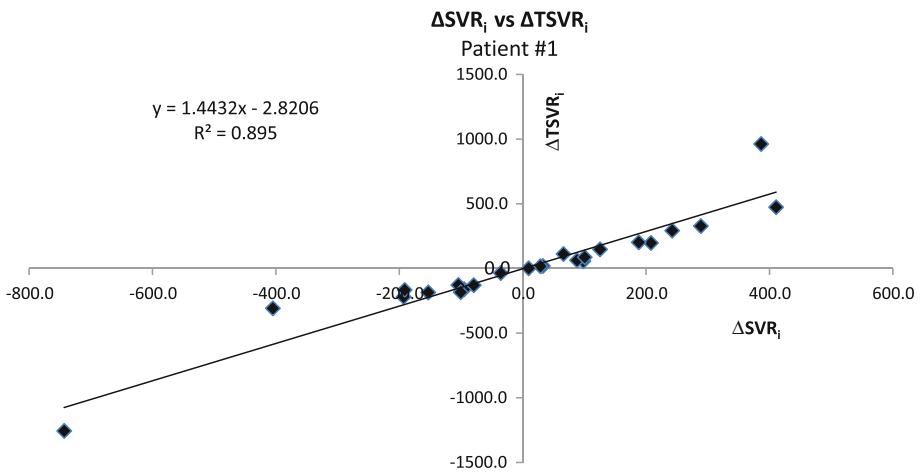

Table 4 The correlation coefficient, R (in bold), and the corresponding 95% confidence intervals for both $TSVR_i$ versus SVR_i and $\Delta TSVR_i$ versus ΔSVR_i

	Patient									
	1	2	3	4	5	6	7	8	9	10
$TSVR_i$ versus SVR_i	**0.91*** (0.79, 0.96)	**0.90*** (0.77, 0.95)	**0.92*** (0.83, 0.97)	**0.99*** (0.97, 0.99)	**0.97*** (0.93, 0.99)	**1.00*** (0.99, 1.00)	**0.96*** (0.91, 0.98)	**0.93*** (0.87, 0.97)	**0.99*** (0.98, 1.00)	**0.94*** (0.88, 0.97)
$\Delta TSVR_i$ versus ΔSVR_i	**0.95*** (0.87, 0.98)	**0.96*** (0.91, 0.98)	**0.94*** (0.85, 0.97)	**0.99*** (0.99, 1.00)	**0.97*** (0.94, 0.99)	**1.00*** (0.99, 1.00)	**0.97*** (0.93, 0.99)	**0.97*** (0.93, 0.98)	**1.00*** (0.99, 1.00)	**0.99*** (0.98, 1.00)

* $P < 0.0001$

However, the use of PAOCs has been associated with questionable benefit as well as significant morbidity, mortality, and economic cost (ASA 2003; Domino et al. 2004; Stewart et al. 1998). In particular, the rising incidence of antibiotic-resistant nosocomial infections may further question, and limit, the use of these highly invasive hemodynamic monitors (Blot et al. 2005; Richards et al. 2000).

Minimally-invasive hemodynamic monitors, specifically the EDM, have become well established as reliable, safe, and accurate (Madan et al. 1999; DiCorte et al. 2000; Dark and Singer 2004). Furthermore, the EDM can be placed either orally, in intubated patients, or nasally, in awake patients (Atlas and Mort 2001; Dodd 2002). Whereas the FlowTrac® requires a peripheral arterial cannula. It should be noted that both of these instruments provide approximations of cardiac output. However, in clinical trials, the EDM has been shown to provide reproducible and reliable measurements of cardiac output (Singer et al. 1989; Valtier et al. 1998). In addition, the EDM is extremely accurate in measuring *changes* in cardiac output (DiCorte et al. 2000; Dark and Singer 2004).

Therefore, the ability to assess afterload, with minimally-invasive devices, without CVP measurement, could offer significant benefit in patient safety. This preliminary study has shown that CVP appears to play a minor role in afterload assessment. Furthermore, the hour-to-hour changes in afterload appear to be minimally affected by the exclusion of CVP.

However, specific clinical conditions may nonetheless require CVP measurement for management purposes. These would include such pathological states as: superior vena cava syndrome, congenital cardiac anomalies, intracardiac tumors, and cardiac tamponade. These conditions can create significant increases in CVP by restricting venous blood flow (Magder 2005).

Furthermore, jugular venous distention (JVD), which is a diagnostic "hallmark" of CHF, is also associated with an increase in central venous pressure (Drazner et al. 2001). However, invasive monitoring of CVP is rarely necessary for the clinical diagnosis and management of this common condition (Senni et al. 1998).

Certain clinical situations may artificially alter CVP. The use of positive end-expiratory pressure (PEEP), for patients with severe pulmonary disease, is one example (Magder 2005). In addition, the simultaneous use of the central venous cannula, for intravenous fluid administration *during* CVP measurements, may produce erroneous data (Ho et al. 2005). Furthermore, CVP may not be meaningful for patients who are in the prone or lateral decubitus positions (Soliman et al. 1998). A malpostioned pressure transducer can also significantly change CVP measurements (Magder 2005). Therefore, in these clinical situations, $TSVR_i$ may be advantageous over SVR_i due to the possible *inaccuracy* of CVP.

Continuous afterload assessment would also be useful in "highly critical" situations. These would possible include patients on potent vasoactive medications for treatment during shock or sepsis. Patients undergoing removal of a pheochromocytoma may also benefit from this.

Lastly, other hemodynamic parameters and terminology, associated with afterload assessment, exist. These include: pulse wave velocity, impedance, and characteristic impedance (Segers et al. 2007; Nichols and O'Rourke 2005; Milnor 1989). However, SVR and SVR_i remain the "mainstays" of clinical afterload measurement within the hospital-based clinical community.

Conclusions

This preliminary study has favorably compared, and correlated, afterload assessment with CVP measurement (SVR_i) to afterload assessment without CVP measurement ($TSVR_i$). Further research, which would include prospective trials to assess the benefits, risks, and limitations of $TSVR_i$ measurement, appear indicated. In addition, these studies could be carried out with minimally-invasive hemodynamic monitors. A clinical comparison, of various means of afterload assessment, could then be implemented. Thus, a comparison of SVR_i to $TSVR_i$, with respect to patient outcome, complications, and cost, could be made.

Appendix

The Interrelationship Between ΔSVR_i and $\Delta TSVR_i$

ΔSVR_i is calculated using the definition of SVR_i from Eq. 1:

$$\Delta SVR_i = \left[\frac{(MAP_2 - CVP_2)}{C_{i_2}}\right] \cdot 80 - \left[\frac{(MAP_1 - CVP_1)}{C_{i_1}}\right] \cdot 80 \tag{1A}$$

Rearrangement yields:

$$\Delta SVR_i = \left[\frac{MAP_2}{C_{i_2}} - \frac{MAP_1}{C_{i_1}}\right] \cdot 80 + \left[\frac{CVP_1}{C_{i_1}} - \frac{CVP_2}{C_{i_2}}\right] \cdot 80 \tag{2A}$$

Substituting the definition of $TSVR_i$ from Eq. 2 results in:

$$\Delta SVR_i = \Delta TSVR_i + \left[\frac{CVP_1}{C_{i_1}} - \frac{CVP_2}{C_{i_2}}\right] \cdot 80 \tag{3A}$$

Realizing that:

$$\frac{CVP_1}{C_{i_1}} \approx \frac{CVP_2}{C_{i_2}} \quad \text{thus:} \left[\frac{CVP_1}{C_{i_1}} - \frac{CVP_2}{C_{i_2}}\right] \approx 0 \tag{4A}$$

Finally:

$$\Delta SVR_i \approx \Delta TSVR_i \tag{5A}$$

This concept can also be illustrated using the definition of the total differential:

$$\Delta SVR_i = \frac{\partial SVR_i}{\partial MAP}\Delta MAP + \frac{\partial SVR_i}{\partial C_i}\Delta C_i + \frac{\partial SVR_i}{\partial CVP}\Delta CVP \tag{6A}$$

and

$$\Delta TSVR_i = \frac{\partial TSVR_i}{\partial MAP}\Delta MAP + \frac{\partial TSVR_i}{\partial C_i}\Delta C_i \tag{7A}$$

Since $\frac{\partial SVR_i}{\partial CVP}\Delta CVP \approx 0$ then $\Delta SVR_i \approx \Delta TSVR_i$. Also: $\frac{\partial SVR_i}{\partial MAP} \approx \frac{\partial TSVR_i}{\partial MAP}$ and $\frac{\partial SVR_i}{\partial C_i} \approx \frac{\partial TSVR_i}{\partial C_i}$.

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