Assessing Fluid-Responsiveness by a Standardized Ventilatory Maneuver: The Respiratory Systolic Variation Test

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Respiratory-induced changes in arterial blood pressure predict fluid responsiveness. However, the accuracy of these variables is affected by the preset tidal volume and by the early inspiratory increase in arterial blood pressure. We have therefore calculated the slope produced by the minimal systolic blood pressures (plotted against the respective airway pressures) during a ventilatory maneuver consisting of four incremental, successive, pressure-controlled breaths, termed the Respiratory Systolic Variation Test (RSVT). In 14 ventilated patients, after major vascular surgery, the slope of the RSVT decreased significantly after intravascular fluid administration and correlated with the end-diastolic area and with changes in cardiac output better than filling pressures. This preliminary study suggests that a standardized ventilatory maneuver may be useful in guiding fluid therapy in ventilated patients.

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Functional hemodynamic variables that are based on analysis of the respiratory-induced variations in the arterial blood pressure (AP), e.g., the systolic blood pressure variation (SPV) (1), the pulse pressure variation (PPV) (2), and the stroke volume variation (SVV) (3), are sensitive indicators of fluid responsiveness (FR) in ventilated patients. These variables are greatly influenced by the magnitude of the tidal volume used (4,5). In addition, they are based on the difference between the maximal and minimal left ventricular stroke volumes (LVSV) during the mechanical breath. However, it is only the decrease in LVSV that reflects FR, whereas its augmentation during early inspiration, especially when being the dominant AP variation, is associated with hypervolemia or congestive heart failure (6) and absent FR (1,7). We have therefore developed the Respiratory Systolic Variation Test (RSVT), which is a measure of the slope (RSVTs) of the decrease in the systolic AP in response to a standardized maneuver consisting of a series of successive incremental pressure-controlled breaths. In this study, we report our preliminary observations on the feasibility of using the RSVT to predict FR in ventilated patients.

Methods

We studied 14 patients after abdominal aortic surgery after the respective approval of each institution's human investigation committee and after obtaining written informed consent. Contraindications for enrollment in the study included advanced congestive heart failure and rhythm other than sinus. The study was conducted in the first 2 h after admission to the intensive care unit, with the patients on pressure-controlled mechanical ventilation (Siemens 900C ventilator) under sedation with fentanyl and no spontaneous breathing efforts.

The RSVT was performed (in triplicate) by manually adjusting the pressure-controlled knob to produce four successive breaths at inspiratory pressures of 5, 10, 15, and 20 cm H_2O, at a respiratory rate of 12/min, inspiratory/expiratory ratio of 1:2.5 and no positive end-expiratory pressure. Off-line analysis of a paper strip chart recorder included identification of the minimal value of the systolic AP after each of the

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The first author is the inventor of the RSVT (US patent no. 5,769,082) and is a member of the medical advisory board of Pulsion Medical Systems, Munich, Germany.

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four respective breath cycles (Fig. 1). These values were then plotted against the corresponding airway pressures and a line of best fit drawn through these four points, with the resulting RSVTs expressed in mm Hg/cm H_2O. Other measurements including the indexed left ventricular end-diastolic area (EDAI) by transesophageal echocardiography were performed before and after intravascular fluid administration (7 mL/kg of plasma expander over 30 min). Statistical analysis was performed by a two-tailed *t*-test, Pearson correlation, and receiver operating characteristic (ROC) curves, where appropriate.

Results

Baseline data and their changes after intravascular fluid administration are presented in Table 1. The initial RSVTs (range, 0.03–0.55 mm Hg/cm H_2O) decreased significantly after intravascular fluid administration (range, 0.01–0.35). The relative range of any triplicate RSVTs measurements did not exceed 15%. EDAI correlated better with the RSVTs (Fig. 2) than with the pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP) (r = 0.792 and 0.696, respectively) before intravascular fluid administration. Baseline RSVT values of patients whose cardiac index (CI) responded to intravascular fluid administration were significantly higher than those of nonresponders (Table 2) and correlated with the change in CI significantly better than the EDAI, PAOP, and CVP (Fig. 3). In addition, the percent of change in the CI correlated significantly (r = 0.748) with the decrease in the RSVTs after intravascular fluid administration.

The areas under the ROC curves for a ≥15% change of CI for the PAOP, EDAI, and RSVT were 0.771 (95% confidence intervals, 0.486–1.055), 0.875 (95% confidence interval, 0.683–1.067), and 0.896 (95% confidence interval, 0.726–1.065), respectively. An RSVT value ≥0.24 mm Hg/cm H_2O predicted a change ≥15% in CI with a sensitivity of 87.5% and a specificity of 83%, whereas an EDAI value ≤10.2 cm^2/m^2 predicted a change ≥15% in CI with a sensitivity of 87.5% and a specificity of 67%.

Discussion

The RSVT is a new functional hemodynamic variable that influences preload by a series of incremental tidal volumes. Our results clearly show that steeper RSVTs are associated with smaller EDAI and a significant CI increase after intravascular fluid administration. Because the size of the tidal volume complicates the interpretation of the SPV, PPV and SVV (4,5), the RSVT is a first attempt at standardizing the respiratory stimulus that serves as a basis for functional hemodynamics (8). In addition, the RSVT is a measure of the slope created by the lowest systolic values only and does not include the frequently occurring early inspiratory increase in the SV, which is more prominent in the presence of hypervolemia or congestive heart failure, denoting absent FR (1,4,6,7). The inclusion of the maximal beat in the calculation of the SPV, PPV, and SVV may therefore decrease their accuracy as

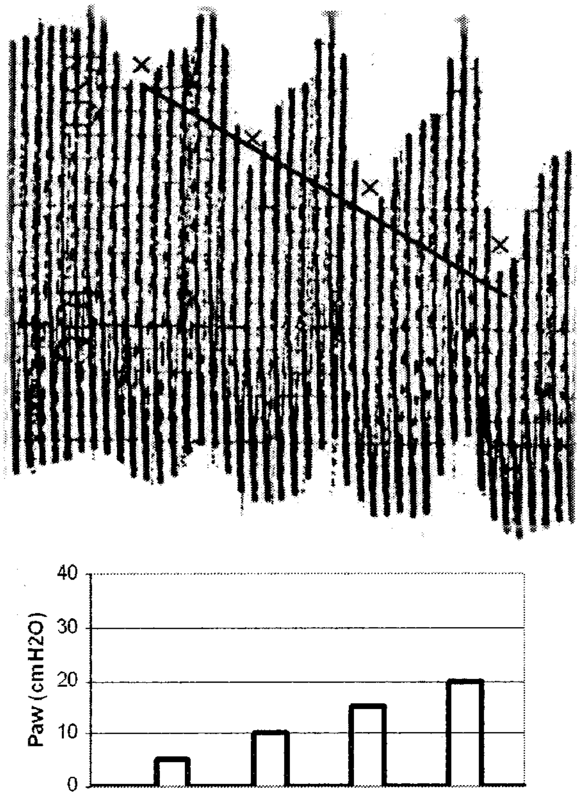

Figure 1. Lower panel: Schematic representation of the Respiratory Systolic Variation Test (RSVT) maneuver. Upper panel: The smallest four systolic pressure values (identified by the X) are plotted against their respective airway pressures to obtain the slope of the RSVT.

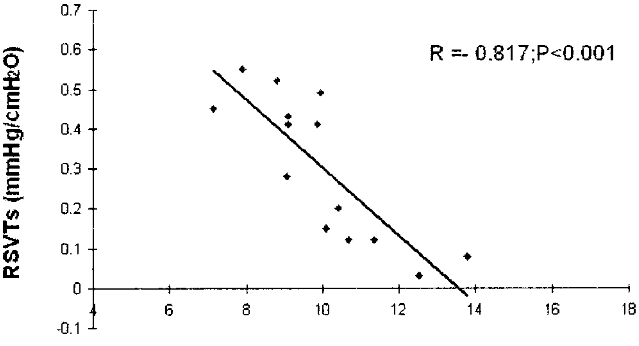

Figure 2. The relationship between end-diastolic area (EDAI) and the Respiratory Systolic Variation Test (RSVT) before intravascular fluid administration.

Table 1. Hemodynamic Data Before and After Intravascular Fluid Administration

Variable	Baseline values (Mean ± SD)	After volume loading	*P*-value
RSVTs (mm Hg/cm H_2O)	0.30 ± 0.18	0.10 ± 0.10	<0.001
EDAI (cm^2/m^2)	10.0 ± 1.7	11.5 ± 1.4	<0.0001
BPm (mm Hg)	86 ± 13	97 ± 12	<0.002
PAPm (mm Hg)	25.8 ± 8.8	32.1 ± 6.4	<0.001
PAOP (mm Hg)	11.9 ± 5.9	17.6 ± 5.8	<0.0001
CVP (mm Hg)	10.7 ± 4.0	13.4 ± 3.9	<0.05
HR (bpm)	105 ± 18	96 ± 16	<0.03
CI ($L \cdot min^{-1} \cdot m^2$)	2.78 ± 0.58	3.28 ± 0.74	<0.0001
SVI (mL/m^2)	27.6 ± 9.1	35.2 ± 9.8	<0.001
SVR ($dynes \cdot s^{-1} \cdot cm^5$)	1218 ± 297	1158 ± 276	NS
PVR ($dynes \cdot s^{-1} \cdot cm^5$)	227 ± 119	210 ± 110	NS

RSVTs = Slope of the Respiratory Systolic Variation Test; EDAI = left ventricular end-diastolic area index; BPm = mean arterial blood pressure; PAPm = mean pulmonary artery pressure; PAOP = pulmonary artery occlusion pressure; CVP = central venous pressure; HR = heart rate; CI = cardiac index; SVI = stroke volume index; SVR = systemic vascular resistance; PVR = pulmonary vascular resistance.

Table 2. Hemodynamic Variables Before Intravascular Fluid Administration of Responders (change in CI ≥ 15%) and Nonresponders

	Responders (*n* = 8)	Nonresponders (*n* = 6)	*P*-value
RSVTs (mm Hg/cm H_2O)	0.41 ± 0.14*	0.17 ± 0.13	<0.01
EDAI (cm^2/m^2)	9.1 ± 1.1	11.2 ± 1.7	<0.02
BPm (mm Hg)	79 ± 10	95 ± 10	<0.01
PAOP (mm Hg)	9.2 ± 3.8	15.3 ± 6.8	<0.052
CVP (mm Hg)	9.6 ± 3.6	12.2 ± 4.3	NS
HR (bpm)	106 ± 16	103 ± 21	NS
CI ($L \cdot min^{-1} \cdot m^2$)	2.67 ± 0.59	2.93 ± 0.59	NS
SVI (mL/m^2)	25.8 ± 8.0	30.2 ± 10.6	NS

Values are mean ± SD.

RSVTs = slope of the Respiratory Systolic Variation Test; EDAI = end diastolic area index; BPm = mean arterial blood pressure; PAPm = mean pulmonary artery pressure; PAOP = pulmonary artery occlusion pressure; CVP = central venous pressure; HR = heart rate; CI = cardiac index; SVI = stroke volume index.

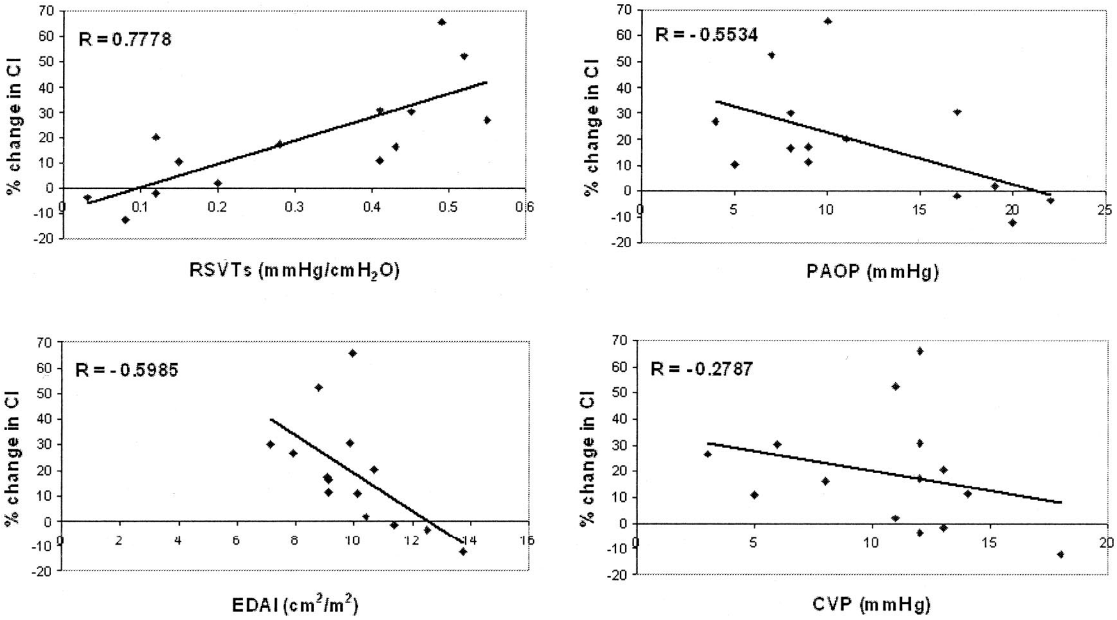

Figure 3. The correlation of baseline values of central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP), end-diastolic area (EDAI), and Respiratory Systolic Variation Test (RSVTs)to the change in cardiac index (CI) after intravascular fluid administration.

predictors of FR and may account for the recently reported reduced effectiveness of the SVV as a predictor of FR in patients with impaired left ventricular function (3).

This preliminary report suggests that the RSVT is a useful variable of FR in mechanically ventilated patients. Although performed manually in this study, this maneuver can become clinically available in the future by appropriate automation and linkage of ventilator and monitor.

References

1. Tavernier B, Makhotine O, Lebuffe G, et al. Systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. Anesthesiology 1998;89:1313–21.
2. Michard F, Boussat S, Chemla D, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J Respir Crit Care Med 2000;62:134–8.
3. Reuter DA, Kirchner A, Felbinger TW, et al. Usefulness of left ventricular stroke volume variation to assess fluid responsiveness in patients with reduced cardiac function. Crit Care Med 2003;31:1399–404.
4. Szold A, Pizov R, Segal E, Perel A. The effect of tidal volume and intravascular volume state on systolic pressure variation in ventilated dogs. Intensive Care Med 1989;15:368–71.
5. Reuter DA, Bayerlein J, Goepfert MS, et al. Influence of tidal volume on left ventricular stroke volume variation measured by pulse contour analysis in mechanically ventilated patients. Intensive Care Med 2003;29:476–80.
6. Preisman S, DiSegni E, Vered Z, Perel A. Left ventricular preload and function during graded hemorrhage and retranfusion in pigs: analysis of arterial pressure waveform and correlation with echocardiography. Br J Anaesth 2002;88:716–8.
7. Vieillard-Baron A, Chergui K, Augarde R, et al. Cyclic changes in arterial pulse during respiratory support revisited by Doppler echocardiography. Am J Respir Crit Care Med 2003;168:671–6.
8. Perel A. The value of functional hemodynamic parameters in hemodynamic monitoring of ventilated patients. Anaesthesist 2003;52:1003–4.