A Comparison of Systolic Blood Pressure Variations and Echocardiographic Estimates of End-Diastolic Left Ventricular Size in Patients After Aortic Surgery

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As suggested by experimental studies, systolic pressure variation (SPV), the difference between maximum and minimum values of systolic blood pressure after a single positive pressure breath, may be a fair indicator of left ventricular preload. SPV was quantified in 21 patients who had undergone abdominal aortic surgery and were sedated under mechanical ventilation. The aim of the study was to assess the ability of this parameter to qualitatively estimate left ventricular preload measured using transesophageal echocardiography. All patients had preoperative radionuclide ejection fraction >45%. Postoperative mechanical ventilatory patterns were the same for all patients: tidal volume = 10 mL/kg; respiratory frequency = 12–14 breaths/min; and zero end-expiratory pressure mode. Left ventricular dimensions at end-diastole correlated well with the magnitude of both SPV (r = 0.80) and its delta down (dDown) component (i.e., the degree by which systolic pressure decreases with each mechanical breath) (r = 0.83). Once the first measurement was completed, volume loading with two increments of 250 mL of human albumin 5% was performed in all but three patients. Each volume loading step caused a significant increase in the end-diastolic area (EDa) index (7.0 ± 1.6 to 8.5 ± 1.6 cm^2/m^2) and cardiac index (CI) (3.1 ± 0.9 to 4.1 ± 0.9 $L \cdot min^{-1} \cdot m^{-2}$) and a concomitant significant decrease in the SPV (8.6 ± 4.5 to 6.1 ± 3.7 mm Hg) and its dDown component (5.9 ± 4.1 to 2.9 ± 2 mm Hg). The initial (preinfusion) dDown values showed a significant linear correlation to the increase in EDa (r = 0.63) and CI (r = 0.55) in response to the infusion of 500 mL of colloid solution. Thus the higher the initial dDown, the greater was the change in EDa and CI after volume loading. These findings confirm the results of animal studies that demonstrate the usefulness of SPV to estimate left ventricular filling. They suggest that, in mechanically ventilated patients after vascular noncardiac surgery, pressure waveform analysis is a simple means of cardiovascular assessment that provides reliable information concerning preload estimation and the response to left ventricular volume loading.

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The accurate estimation of effective intravascular volume is of great importance in patients after major vascular procedures. The main goal of achieving an adequate preload is to improve the ability of the left ventricle to cope with alterations in contractility or afterload and maintain cardiac output (1). Because major vascular procedures are associated with aortic clamping and unclamping, blood loss, large amounts of intravenous fluids, and underlying cardiovascular pathology, patients may have either inadequate or excessive preload postoperatively (2).

The assessment of the preload is now done mainly by measuring the pulmonary artery occlusion pressure (PAOP). The limitations of using pulmonary artery catheter pressure as an indicator of left ventricular (LV) end-diastolic volume, which is a true measure of preload, are well documented (3,4). Recent experimental studies have introduced a new method for assessing preload, based on the analysis of changes in arterial pressure waveform during mechanical ventilation (5–8). The increase in intrathoracic pressure during a mechanical breath causes a decrease in venous return that is expressed by a decrease in arterial pressure, mainly due to the reduction in LV stroke output. The degree by which the systolic pressure decreases after a fixed tidal volume, termed delta down (dDown), has been shown to be a sensitive indicator of hypovolemia

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(5). The absence of such decrease is indicative of hypervolemia and/or congestive heart failure (8).

These animal studies, which demonstrate the usefulness of the systolic pressure variation (SPV) to estimate LV filling, might lead to clinically useful information. We have quantified the SPV in patients who were sedated and whose lungs were being mechanically ventilated immediately after abdominal aortic surgery. Our goal was to assess the ability of the SPV to reflect the preload status using LV filling estimated by a transesophageal echocardiogram (TEE). This approach is based on the fact that LV short-axis areas provide accurate values for LV size that correlate well with those obtained by angiographic or scintigraphic methods (9–12).

Methods

After institutional approval of the study protocol, patients scheduled to undergo abdominal aortic surgery in our institution were asked to participate in the study. Pre- and intraoperatively, the patients were managed according to standard clinical practice used in the vascular surgery unit for all aortic surgical patients. Therefore, all patients 1) underwent gated radionuclide angiography to determine ejection fraction before surgery, 2) had a pulmonary artery catheter inserted for perioperative monitoring, and 3) were maintained under fentanyl sedation for the first three postoperative hours in a specially organized recovery room. Contraindications for enrollment in the study included the following: 1) chronic β-adrenergic block therapy, 2) clinical history of congestive heart failure (New York Heart Association Classes III and IV), 3) preoperative radionuclide LV ejection fraction less than 40% (normal level for our laboratory is 65 ± 6%), 4) disabling respiratory disease, and 5) the need for antihypertensive therapy (systolic blood pressure greater than 160 mm Hg) at the end of the surgical procedure or postoperatively before hemodynamic measurements were performed.

Of the 27 patients initially enrolled in the study, 6 were excluded. Adequate short-axis LV images could not be obtained in 3 patients, and intravenous antihypertensive therapy was administered in 3 others. In the 21 patients studied, surgical procedures included infrarenal aortic repair because of infrarenal aneurysm (n = 9) or occlusive atheromatous disease (n = 12).

The final patient group included 18 males and 3 females. Weight ranged from 57 to 105 kg. All patients were studied 30 min to 1 h postoperatively in a recovery room specially devoted to vascular surgical patients.

A cannula was inserted into the radial artery, and a flow-directed pulmonary artery thermodilution catheter was positioned via the right internal jugular vein. Thereafter, anesthesia was induced with fentanyl, flunitrazepam, and pancuronium bromide. Anesthesia was maintained by incremental doses of fentanyl, as well as nitrous oxide in oxygen and isoflurane.

During the surgical procedure, fluid infusion was left to the discretion of the anesthesiologist in charge of the patient. No echocardiographic monitoring was used during the surgical procedure.

After surgery, patients were transferred, with their tracheas intubated and their lungs ventilated, to the recovery room. Postoperative sedation was provided by intravenous administration of fentanyl (300–500 μg/h) over at least the first 2–3 h to allow positive pressure ventilation with heated humidified oxygen in air (F_{IO_2} 0.4) during the rewarming period. Respiratory frequency was 12–14 breaths/min with tidal volume set at 10–15 mL/kg. A zero end-expiratory pressure mode of ventilation was used. The use of additional fentanyl or benzodiazepine to maintain sedation was left to the discretion of the physicians caring for the patients in the recovery room. If systolic blood pressure was greater than 160 mm Hg for more than 2 min, labetalol or nicardipine was given intravenously and the patient was excluded from the study.

Upon arrival in the recovery room, a Diasonics 3–5 MHz phased array ultrasonic transducer, fitted to the end of a standard gastroscope, was inserted into the patient's esophagus. A Diasonics ultrasonograph was used for two-dimensional echocardiographic imaging. The probe was positioned to obtain a LV short-axis image at the midpapillary muscle level. The transducer of the TEE was not moved during the study.

The experimental protocol started 20–60 min after the patient's arrival in the recovery room. A complete set of hemodynamic measurements and echocardiographic images was obtained, with the PAOP being determined at end-expiration from a tracing provided by the Marquette monitor. A mean value of pulmonary capillary wedge pressure measured over three respiratory cycles was considered. Cardiac output was measured in triplicate. Simultaneously the systemic arterial blood pressure curve, obtained from the radial artery catheter, was recorded together with the airway pressure on a Gould ES 1000 strip chart recorder.

Once the first set of measurements was completed, a bolus of 250 mL of 5% human albumin was infused over 20 min twice in each patient, except in those who had a PAOP greater than 16 mm Hg. A complete set of hemodynamic and echocardiographic measurements was performed after each 250-mL bolus of the colloid infusion.

The arterial pressure waveforms and TEE recordings were analyzed by independent observers (blood pressure records by AP, TEE tapes by FLB and MS), in a

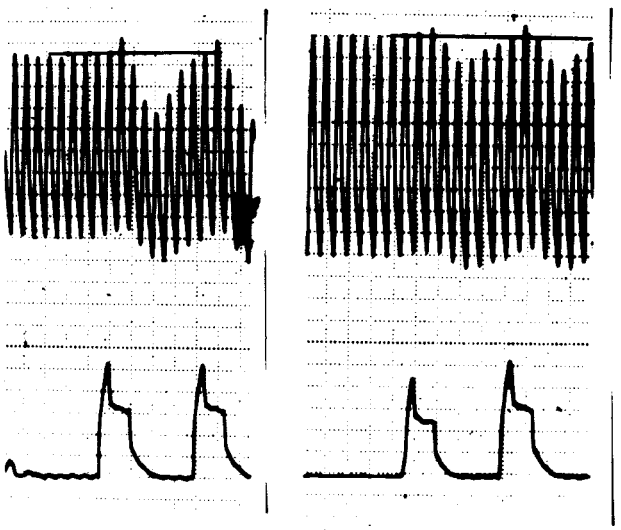

Figure 1. Systemic arterial blood pressure curve and airway pressure before (left: systolic pressure variation [SPV] = 20 mm Hg) and after (right: SPV = 10 mm Hg) volume loading. A short period of apnea affords the opportunity to calculate the delta down component of the SPV.

blinded fashion with the reviewers unaware of any clinical data.

Area analysis of TEE data consisted of measurements of end-diastolic area (EDa) and end-systolic area (ESa). Ejection fraction area (EFa) was calculated using the standard formula (EDa − ESa)/EDa. Areas were determined for four consecutive beats by outlining the endocardium with a light pen using the leading-leading edge technique. All patients were in normal sinus rhythm, and no rhythm disturbances were noted during the echocardiographic measurement period. The mean value of the four measurements of EDa and ESa was divided by the surface body area of the patient to obtain indexed LV areas at end-systole and at end-diastole (13). The accuracy of this approach for area determination has been previously demonstrated (12,13).

Pressure waveform analysis was done in the following manner: the SPV, which is the difference between the maximal systolic pressure and minimal systolic pressure during one cycle of a mechanical breath, was measured from the strip chart. The mean of the SPV values during five consecutive breaths was calculated for the control state and after each volume load. The value of the systolic blood pressure during a period of 10 s of apnea was used as a reference pressure for the calculation of the dDown and delta up (dUp) (Figure 1). The dDown is the difference between the value of the minimum systolic pressure during one cycle of a mechanical breath and the systolic pressure during apnea. The dUp is the difference between the maximum systolic pressure and the systolic pressure during apnea. The SPV is the sum of the dUp and dDown.

Table 1. Hemodynamic and Echocardiographic Data Upon the Patients' Arrival in the Intensive Care Unit (n = 21) (Mean ± SD)

MAP (mm Hg)	85 ± 12	(63–103)
SAP (mm Hg)	126 ± 17	(87–155)
HR (beats/min)	82 ± 10	(52–101)
CI ($L \cdot min^{-1} \cdot m^{-2}$)	3.2 ± 0.9	(1.9–5.6)
PAOP (mm Hg)	7 ± 4	(1–20)
SVR ($dynes \cdot s^{-1} \cdot cm^{-5}$)	910 ± 180	(650–1250)
EDaI (cm^2)	7.2 ± 2.0	(3.1–11.5)
EFa (%)	63 ± 15	(31–82)
SPV (mm Hg)	8.5 ± 4.5	(3–20)
Delta down (mm Hg)	5.9 ± 4.1	(1–16)
Delta up (mm Hg)	2.8 ± 1.2	(1–5)
Peak paw (cm H_2O)	13 ± 2	(10–15)
Respiratory compliance mL/cm H_2O)	47 ± 5	(40–55)
Temperature (°C)	34 ± 1	(32.5–36)

Ranges appear in parentheses.
MAP = mean arterial pressure; SAP = systolic arterial pressure; HR = heart rate; CI = cardiac index; PAOP = pulmonary artery occlusion pressure; SVR = systemic vascular resistance; EDaI = end-diastolic area; EFa = ejection fraction area; SPV = systolic pressure variation.

Because the arterial pressure has an additional nonrespiratory low frequency fluctuation [Mayer waves (14)], the dUp and dDown were determined only during the respiratory cycle that immediately preceded or succeeded the apnea period (Figure 1). Dynamic respiratory compliance was calculated by dividing tidal volume by peak airway pressure recorded together with blood pressure.

Conventional regression analysis and two-way analysis of variance were used when appropriate. A P value less than 0.05 was considered statistically significant. All values are presented as mean ± SD.

Results

The final patient group included 21 patients (ages 49–75 yr, mean 63 yr), with a mean preoperative ejection fraction of 60% (range 45%-79%).

All patients tolerated the study well. The initial postoperative values of the echocardiographic and hemodynamic parameters in the intensive care unit are shown in Table 1. Central body temperature measured using the pulmonary artery catheter ranged from 32.5 to 36°C. Peak airway pressure, recorded together with blood pressure, ranged from 9 to 15 cm H_2O.

A significant inverse linear relationship was found between the initial values of the indexed EDa and both the SPV ($-0.36x + 10$, $r = 0.80$) and the dDown ($-0.40x + 9.5$, $r = 0.83$). (Figure 2). A weaker correlation was found between the dDown and the initial EFa ($r = 0.49$), lower EFa values being associated with smaller dDown values.

By contrast, no significant linear relationship was found between the initial indexed EDa and the initial PAOP (Figure 2).

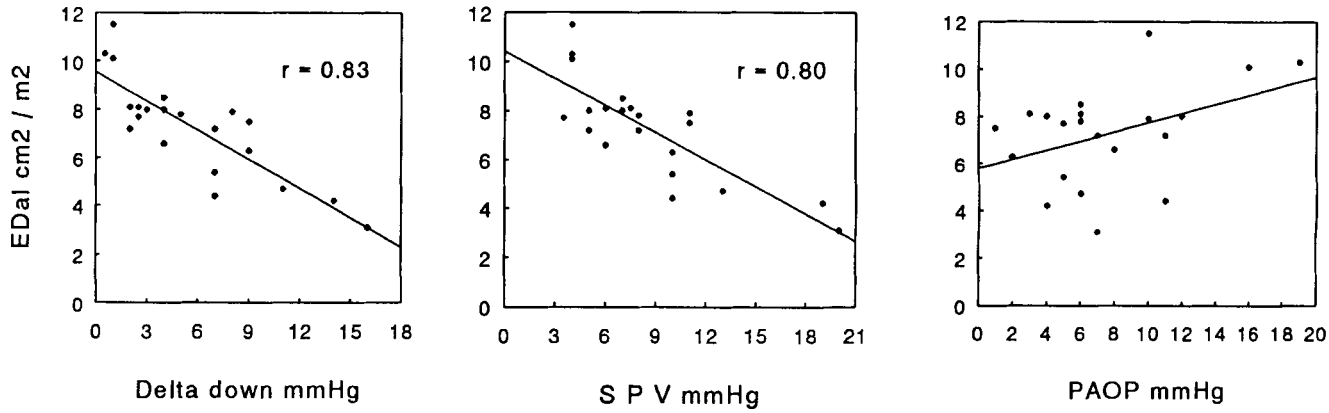

Figure 2. Relationship between initial end-diastolic area (indexed) (EDaI) and 1) the delta down component of the systolic pressure variation (SPV) (left), 2) SPV (middle), and 3) pulmonary artery occluded pressure (PAOP) (right).

An overt marked hypovolemic state, characterized by complete emptying of the left ventricle at end-systole and an indexed EDa less than 5 cm^2/m^2, was evident in three patients. These three patients were the only ones in whom a dDown greater than 10 mm Hg and a SPV greater than 12 mm Hg were noted.

Once the first set of measurements was completed, volume loading with two increments of 250 mL human albumin 5% was performed in all but three patients. One of these patients showed signs of marked hypovolemia (complete systolic emptying associated with an EDa index lower than 5 cm^2). This led to suspicion of intraabdominal hemorrhage, and the patient was returned to the operating room. In the two other patients, volume loading was not performed because of postoperative PAOP values of 18 and 20 mm Hg and EFa values of 31% and 32%, respectively. In these two patients, EDa index values in the high range (10.4 and 11.7) associated with low values of dDown (1 and 0.5 mm Hg) were found.

In the remaining 18 patients, each step of the volume loading caused a significant increase in the EDa and CI and a concomitant significant decrease in the SPV and dDown (Table 2, Figure 3). No significant change in PAOP was noted in response to colloid infusion.

The initial (preinfusion) dDown values, i.e., the degree by which the systolic blood pressure decreased with each mechanical breath, showed a significant linear correlation to the increase in EDa ($r = 0.63$) and CI ($r = 0.55$) in response to the infusion of 500 mL of colloid solution (Figure 4). Thus the higher the initial dDown, the greater was the change in EDa and CI after volume loading.

Table 2. Hemodynamic and Echocardiographic Data Before and After Volume Loading ($n = 18$) (Mean ± SD)

	Before loading	Colloid infusion	
		250 mL	500 mL
MAP (mm Hg)	84 ± 12	90 ± 10	90 ± 12
SAP (mm Hg)	126 ± 18	133 ± 17	137 ± 15
HR (beats/min)	79 ± 10	79 ± 11	82 ± 11
CI ($L \cdot min^{-1} \cdot m^{-2}$)	3.1 ± 0.9	3.5 ± 0.19*	4.1 ± 0.9*
PAOP (mm Hg)	7 ± 3	8 ± 3	10 ± 3
EDaI (cm^2/m^2)	7.0 ± 1.6	7.9 ± 1.2*	8.5 ± 1.6*
EFa (%)	65 ± 10	68 ± 9	69 ± 10
Temperature (°C)	34.4 ± 1	34.6 ± 1	34.6 ± 1
Respiratory compliance (mL/cm H_2O)	45 ± 4	45 ± 4	45 ± 4

MAP = mean arterial pressure; SAP = systolic arterial pressure; HR = heart rate; CI = cardiac index; PAOP = pulmonary artery occlusion pressure; EDaI = end-diastolic area; EFa = ejection fraction area.
* $P < 0.05$ compared with the preceding value.

Discussion

This study was designed to assess the ability of SPV to qualitatively estimate LV preload in patients who were sedated and whose lungs were being mechanically ventilated after aortic surgery. These patients were chosen because the need for information about LV filling is great in this population.

This study demonstrated that LV preload assessed by a TEE correlates well with the magnitude of both SPV (dDown plus dUp) and dDown. Moreover, our study confirms the limitations of the pulmonary artery catheter to reliably reflect hypovolemia (2,4,13). We show no correlation between PAOP and EDa and found that PAOP did not reflect the response of EDa and CI to volume loading.

The analysis of the morphology of the arterial pressure curve during positive pressure ventilation is of great clinical interest for the detection of a low preload state. The changes in the arterial pressure waveform during mechanical ventilation become meaningful

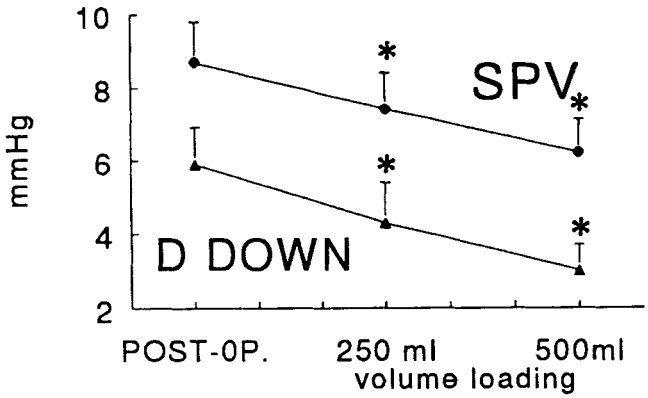

Figure 3. Changes in systolic pressure variation (SPV), and delta down (D DOWN) in 18 patients who received a 500-mL colloid infusion postoperatively (POST-OP.). *$P < 0.05$.

when the normal cardiovascular consequences of a mechanical breath are fully understood. The main cardiovascular effect of an increase in the intrathoracic pressure is the reduction of venous return with relative emptying of the right ventricle. At the same time, the filling of the left ventricle is increased because of the squeezing of the pulmonary vasculature (15,16), leading to the early inspiratory increase in the LV stroke output and the systolic blood pressure (dUp).

A few heartbeats after the beginning of the mechanical breath, the decreased right ventricular output reaches the left heart. The LV stroke output declines, diminishing the systolic pressure and creating the dDown. The dDown is thus a reflection of the decrease in venous return during a mechanical breath. When the baseline preload is low, any further reduction of venous return with each mechanical breath accounts for the typical increase in the dDown value and its correlation with varying degrees of preload (5–8). These considerations explained the correlation between EDa, an accurate index of preload, and dDown, which was demonstrated in this study. The low EDa index values may be due to either a patient hypovolemic state or a decreased LV compliance associated with a moderate decrease in the effective intravascular volume. These states, the treatment of which requires volume loading, were associated with wide SPV.

A LV hypertrophy is common in hypertensive patients and in those undergoing aortic surgery. Because the LV compliance is decreased, LV filling requires an elevated left atrial pressure. In this case, even a minor decrease in venous return, due to moderate hypovolemia, may lead to a major reduction in LV size at end-diastole which rapidly results in altered LV ejection (17,18).

A SPV greater than 12 mm Hg coincided invariably with an abnormally low EDa (less than 5 cm^2/m^2) associated with complete emptying of the left ventricle, an echocardiographic pattern that is highly suggestive of fully inadequate intravascular volume. Moreover, not only did the initial dDown correlate with the initial EDa, it also correlated significantly with the increase in the EDa and the CI after volume loading. This, indeed, is the main value of the dDown, as it is a reflection of the response of the LV output to reduced preload. When the cardiac output is greatly preload-dependent, as in hypovolemia, the dDown is increased (5). When the cardiac output is not influenced by a reduction in preload, as in hypervolemia or congestive heart failure, the dDown is minimal (8). It is clear, then, that the dDown offers information not unlike the slope of a ventricular function curve, hence its ability to predict the response of the CI to volume loading. Measuring the dDown may obviate the need to perform graded volume loading. With each of the two infusions of 250 mL of colloid solution, the SPV and the dDown, like the CI and the EDa, changed significantly. Other hemodynamic variables, such as blood pressure, heart rate, and PAOP, failed to reflect this significant change in volume status. This emphasizes the value of SPV and dDown when assessing preload. However, in clinical practice, the magnitude of SPV will never give an all-or-nothing response which indicates whether the patient is hypovolemic. Rather, it will afford an accurate stratification

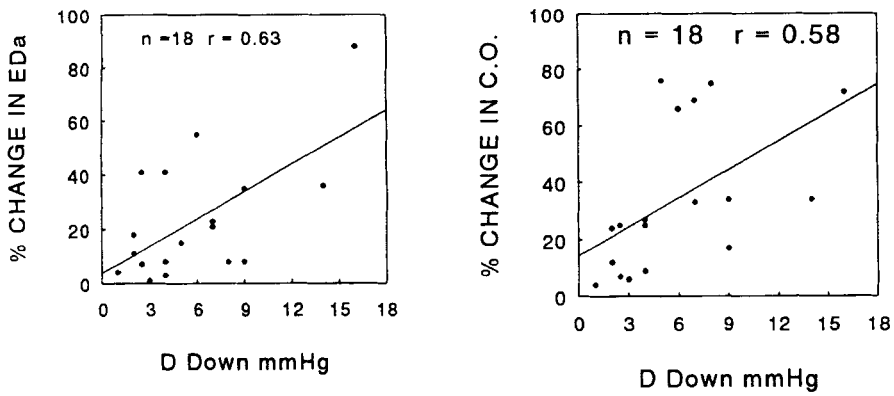

Figure 4. Relationship between initial delta down (D Down) component of the systolic pressure variation and the increase in end-diastolic area (EDa) (left) and cardiac output (C.O.) (right) in response to volume loading.

of a patient's preload state. This will guide the postoperative volume loading of these patients, in whom the complex interaction of anesthesia, surgery, and preexisting cardiac disease requires knowledge of LV volume at end-diastole.

The assessment of a new hemodynamic parameter is usually done by correlating it to existing, well-known parameters which serve as "gold standards." Choosing such a standard may be a problem, as different variables may measure somewhat different phenomena and may have their own inherent problems of methodology, technique, and interpretation. Several studies demonstrated the limitations of PAOP in assessing LV preload, and obviously PAOP could not be used as a gold standard in our study. The comparison of the SPV and dDown to the cardiac output in a heterogeneous group of patients would not result *a priori* in any significant correlation, as a low cardiac output may be associated with either a high dDown (hypovolemia) or a low dDown (heart failure). Moreover, the cyclic fluctuations of the cardiac output during mechanical ventilation may lead to erroneous results, especially when the injection of the cold solution is timed to a fixed point in the mechanical breath cycle.

We have therefore chosen the LV EDa, as determined by a TEE, as a reference index for the SPV and dDown. The LV EDa is the best means for the determination of LV preload in surgical patients, in whom the complex interaction of anesthesia, surgery, and pre-existing cardiac disease requires knowledge of LV volume at end-diastole. Strong correlation between EDa or end-diastolic volume and CI has been observed (13). Besides, several studies demonstrate that LV volume and ejection fraction can be well estimated from LV areas obtained by a single cross-sectional TEE image (12). All these studies led to consider that EDa is a fair index of LV preload. In patients undergoing abdominal aortic surgery, Clements et al. (10) compared short-axis images of the left ventricle obtained by a TEE with those obtained by radionuclide angiography. These authors concluded that TEE short-axis imaging at the midpapillary muscle level is adequate for monitoring LV preload. This study also confirmed the inadequacy of pulmonary artery catheter monitoring to assess LV filling. In patients who had undergone coronary artery bypass surgery, Urbanowicz et al. (19) found that TEE-derived EDa correlated reasonably well with LV end-diastolic volume (19). However, moderate changes in LV volume were not consistently reflected by changes in cross-sectional area obtained with TEE. In this study, LV volumes at end-diastole were determined by combining values of ejection fraction, obtained by blood pool scintigraphy, with thermodilution cardiac output. Since the limitations of the thermodilution technique to determine stroke volume in patients after cardiac surgery have been pointed out, the inaccuracy noted between EDa and LV end-diastolic volume in Urbanowicz et al.'s study might have resulted from the inadequacy of the thermodilution technique, and not from the inability of TEE to determine LV volumes. Recently, the estimation of LV volume by using currently available standard TEE imaging planes has been validated in humans (11). Two-dimensional echocardiographic estimates of LV end-diastolic volume obtained from TEE scans accurately predict corresponding measurements derived from ventricular angiography.

In conventional hemodynamics, preload is often estimated by measuring PAOP. Our study confirms that PAOP is not an accurate index of preload. The lack of correlation between PAOP and end-diastolic LV dimensions has previously been demonstrated in post-coronary artery bypass graft patients and nonsurgical patients in the coronary care unit (20). Our study emphasizes that, in particular, PAOP cannot identify patients who experience a low preload status under mechanical ventilation. Three factors may explain why the PAOP may be unreliable in the assessment of LV end-diastolic pressure during hypovolemia in ventilated patients. According to West's concept, the catheter reflects alveolar pressure and not pulmonary intravascular pressure after balloon occlusion in areas where alveolar capillaries collapse when extravascular pressure exceeds intraluminal pressure (21). Therefore, a hypovolemic state is expected to produce Zone I or II conditions, even at zero end-expiratory pressure (21). Thereby, the pressure measured is a reflection of alveolar rather than intravascular pressure. Second, atrial contraction contributes significantly to LV filling during hypovolemia, increasing the gradient between the LV and pulmonary arteriolar pressure gradients (18–22). Finally, if tachycardia develops, the premature closure of the mitral valve increases the left-atrial-to-LV pressure gradient, while the shortened pressure equilibration time leads to a pressure gradient between the left atrium and the pulmonary artery (18). In addition, in the presence of a hypertrophic cardiomyopathy which decreases LV compliance, hypovolemia may be accompanied by an unchanged or even elevated PAOP (22). Besides, it has been shown that these patients can develop dynamic LV outflow obstruction and intraventricular pressure gradient (17). These account for the fact that pulmonary capillary wedge pressure lacks the sensitivity to detect low preload state in these patients.

All these limitations of PAOP monitoring aimed at detecting low preload states reinforce the interest of considering SPV in vascular surgical patients under mechanical ventilation.

When considering the use of SPV as a clinical monitoring tool, some factors may interfere with the consequence of the provoking maneuver (the mechanical breath) on the outcome variable (the degree of SPV).

The magnitude of breath size influences SPV. This is why the method can only be used in patients mechanically ventilated with a fixed tidal volume. An altered lung compliance may blunt the cardiovascular effect of a mechanical breath, so that SPV may underestimate hypovolemia. Although the patients with an enlarged left ventricle had a consistently low SPV, no conclusion can be drawn regarding the adequacy of their preload. The withdrawal of intrathoracic pressure support of the arterial pressure may play a role in the dDown component of the SPV. Such an effect probably has a similar influence on the patients unless they have patent congestive heart failure. Besides, in patients with markedly altered LV contractility, the integrated cardiovascular system's ability to maintain homeostasis increases preload. Although experimental studies and our clinical data indicate that such abnormalities are associated with a low SPV, it is impossible in these patients to exclude the presence of an ineffective intravascular volume.

Because SPV reflects a change in systolic pressure induced by a decrease in stroke volume, the effective arterial elastance, a parameter that describes the mechanical characteristics of the arterial bed, will influence the magnitude of SPV for a given decrease in stroke volume due to mechanical ventilation. Effective arterial elastance is independent of preload and ventricular function (23). It can be approximated by the product of total arterial resistance and heart rate (1). This explains why, if systemic vascular resistance is low, the SPV may not be a useful indicator of the preload state. No patient in the study received vasodilator antihypertensive agent before the SPV measurements, and systemic vascular resistance values calculated at the time of this measure were in the normal range. Thus the clinical usefulness of SPV is limited to circulatory disorders, the principal mechanism being an insufficient intravascular volume. If a fluctuating systemic vascular resistance is the primary disorder accounting for patient cardiovascular status, then all kinds of SPV might be seen, depending on how preload reserve is affected by the circulatory problem.

In summary, our data show that pressure waveform analysis is a useful hemodynamic variable in vascular surgical patients undergoing mechanical ventilation of their lungs after an abdominal aortic procedure. In our group of postoperative patients free of preoperative LV dysfunction, initial dDown and SPV values were the only hemodynamic variables that correlated significantly with the initial EDa. They also predicted the response of EDa and CI to volume loading and changed significantly with the change in effective blood volume. Although interpretation of pulmonary artery catheter pressure is of little value, pressure waveform analysis is a simple means of cardiovascular assessment that provides reliable information concerning the preload of patients who have undergone vascular surgery.

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