Systolic Pressure Variation in Hemodynamic Monitoring After Severe Blast Injury

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Fluid management in patients following blast injury is a major challenge. Fluid overload can exacerbate pulmonary dysfunction, whereas suboptimal resuscitation may exacerbate tissue damage. In three patients, we compared three methods of assessing volume status: central venous (CVP) and pulmonary artery occlusion (PAOP) pressures, left ventricular end-diastolic area (LVEDA) as measured by transesophageal echocardiography, and systolic pressure variation (SPV) of arterial blood pressure. All three patients were mechanically ventilated with high airway pressures (positive end-expiratory pressure 13 to 15 cm H_2O, pressure control ventilation of 25 to 34 cm H_2O, and I:E 2:1). Central venous pressure and PAOP were elevated in two of the patients (CVP 14 and 18 mmHg, PAOP 25 and 17 mmHg), and were within normal limits in the third (CVP 5 mmHg, PAOP 6 mmHg). Transesophageal echocardiography was performed in two patients and suggested a diagnosis of hypovolemia (LVEDA 2.3 and 2.7 cm^2, shortening fraction 52% and 40%). Systolic pressure variation was elevated in all three patients (15 mmHg, 15 mmHg, and 20 mmHg), with very prominent dDown (23, 40, and 30 mmHg) and negative dUp components, thus corroborating the diagnosis of hypovolemia. Thus, in patients who are mechanically ventilated with high airway pressures, SPV may be a helpful tool in the diagnosis of hypovolemia. © 1999 by Elsevier Science Inc.

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Introduction

Blast injury predisposes the lung to edema formation, and therapy usually includes fluid restriction. On the other hand, bone fractures, abdominal injuries, and burns, which frequently accompany blast injury, require intensive fluid resuscitation.[1,2] Therefore, fluid management in patients who suffer from combined injuries following blast can pose a great challenge.[3] Assessment of the adequacy of fluid resuscitation in multiple trauma patients cannot depend solely on clinical judgment, but also may require direct measurement of filling pressures with either central venous of pulmonary artery (PA) catheter. Measurement of left ventricular end-diastolic area (LVEDA) with transesophageal echocardiography (TEE) can be of assistance in assessing the volume status of such patients.[4-6] During the last decade, arterial waveform analysis has been introduced as a monitoring tool for evaluation of different hemodynamic conditions.[7-11]

Systolic pressure variation (SPV) is the difference between the maximal and minimal systolic blood pressure (SBP) during a cycle of positive pressure ventilation (PPV).[7,8] The SPV can be further divided into the dUp and dDown components by comparison with apneic SBP. The SPV and dDown have been proven to be good predictors of hypovolemia in an experimental set-up and in patients during the perioperative period.[7,8,10] The dUp is an augmentation of the SBP during mechanical inspiration and usually has a positive value even, in hypovolemic patients.[8,10]

In the present study, three methods (PA catheter, TEE,

Table 1. Demographic, Clinical, Respiratory, and Hemodynamic Data

	Patient 1	Patient 2	Patient 3
Age (yrs)	55	34	19
Burns (% body area)	30	30	18
Broncho-pleural fistula	Present	Present	Absent
Vasopressor support	Noradrenaline 1 µg/kg/min	Dopamine 10 µg/kg/min	Noradrenaline 2 µg/kg/min
Pressure control above PEEP (cm H_2O)	34	25	25
PEEP (cm H_2O)	13	14	15
Frequency (breaths/min)	22	20	20
pH	7.33	7.27	7.29
$PaCO_2$ (mmHg)	37	48	42
PaO_2/FIO_2	54	43	61
Lactate (mM/L)	2.4	6.8	NA
Heart rate (bpm)	146	150	150
Mean blood pressure (mmHg)	105	55	70
CVP (mmHg)	14	18	5
PAOP (mmHg)	25	17	6
MPAP (mmHg)	36	30	18
Cardiac output (L/min)	2.7	6.1	2.7
LVEDA (cm^2)	2.3	NA	2.7
SF (%)	52	NA	40
SPV (mmHg)	15	15	20
dUp (mmHg)	−8	−25	−10
dDown (mmHg)	23	40	30

PEEP = positive end-expiratory pressure; $PaCO_2$ = arterial carbon dioxide tension; PaO_2 = arterial oxygen tension; FIO_2 = inspired oxygen concentration; NA = not available; CVP = central venous pressure; PAOP = pulmonary artery occlusion pressure; MPAP = mean pulmonary artery pressure; LVEDA = left ventricular end-diastolic area, measured at end expirium (normal range ≥5.5); SF = shortening fraction (normal range = 50%); SPV = systolic pressure variation.

and SPVs) were used to assess the volume status of three patients with complicated blast injury, who remained hemodynamically unstable despite intensive fluid resuscitation.

Case Report

Three patients with blast injuries sustained in an enclosed space were studied 12 hours following injury. Their demographics are presented in *Table 1*. All three patients suffered from blast lung injury manifested by bilateral lung infiltrates, extensive subcutaneous emphysema, hypoxemia, and bronchopleural fistula (*Table 1*). Blast injury also was manifested by bilateral ear drum perforation in all patients. Other injuries included various levels of third-degree burns (*Table 1*). The three patients were mechanically ventilated using inverse ratio (2:1) pressure control ventilation and an inspired oxygen concentration (FIO_2) of 0.8 to 1.0 (*Table 1*). Ventilatory parameters and relevant laboratory data are presented in *Table 1*. All three patients were treated with catecholamines due to hemodynamic instability (*Table 1*). Increased SPV with a dramatic dDown and significant levels of negative dUp were noted in all three of the patients (*Table 1, Figure 1*). Transesophageal echocardiographic findings in two of the patients corroborated the diagnosis of hypovolemia: a small hypercontractile left ventricle at the midpapillary transgastric view (*Table 1*). On the other hand, filling pressures were elevated in two of the patients, and in the normal range in

the third patient. In these three patients, SPV correlated with volume status as measured by TEE in two patients and with the clinical state of shock in all three (*Table 1*), whereas the filling pressures did not correlate with these findings (*Table 1*). None of the patients had signs of myocardial ischemia, either on electrocardiogram or on the TEE study.

Discussion

Hemodynamic instability in the blast injured patient may be caused by several mechanisms; hypovolemia, coronary artery air embolization, and myocardial contusion.[1,3] In such patients with acute lung injury, fluid overload can aggravate pulmonary edema, whereas inadequate volume replacement in the patient with hemorrhagic shock may hinder tissue perfusion and lead to multiorgan dysfunction. It is therefore essential to accurately assess the volume status of the patient with multiple trauma due to blast injury. In the patients described, direct measurement of intravascular filling pressures [both central venous pressure (CVP) and pulmonary artery occlusion pressures (PAOP)] proved unreliable when compared with direct visualization of the left ventricle by TEE. More accurate estimation of filling pressures during ventilation with high intrathoracic pressures, such as nadir PAOP, requires temporary disconnection from the ventilator, which might be harmful in severely hypoxemic patients.[12] There were no signs of direct myocardial injury. Thus, the elevated
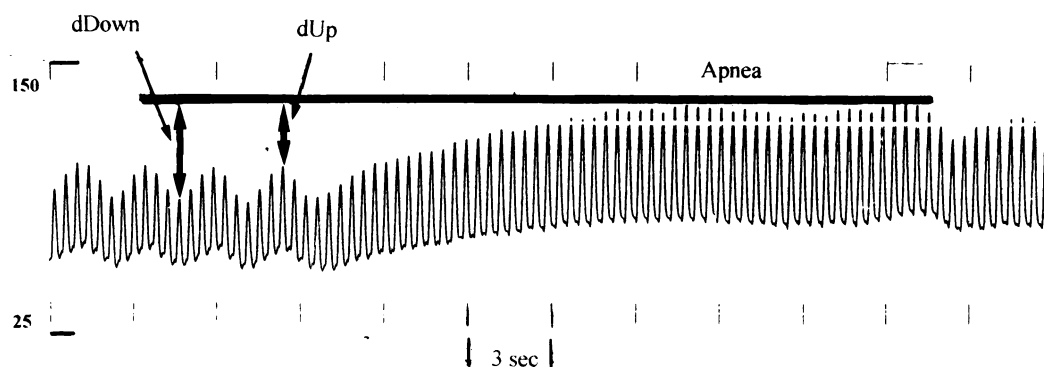
Figure 1. Arterial blood pressure recording of a mechanically ventilated patient with blast lung injury. Systolic pressure variation values are moderately elevated (15 mmHg). A short apnea reveals continuous hemodynamic depression induced by mechanical ventilation, which is expressed by a significant dDown component (40 mmHg) and a negative dUp (−25 mmHg).

filling pressure measurements probably reflected increased intrapleural pressure due to the high ventilatory pressures and limited expiratory period, which may lead to air trapping, and do not represent an increase in preload.[13]

In the presently described patients, the total respiratory compliance was decreased, although it could not be measured because of significant air leak. The transmission of ventilatory pressure to the pleural space is dependent on lung and chest wall compliance. Decreased chest wall compliance without a concurrent decrease in lung compliance results in the greatest transmission of pressure to the pleural space.[13] Even though there are no data to support it, it could be postulated that extensive subcutaneous emphysema may reduce chest wall compliance, making the SPV more accurate than either CVP or PAOP in reflecting the volume status of such patients. Recently, it was shown that relative hypovolemia can be unmasked by increasing the inspiratory:expiratory (I:E) ratio in mechanically ventilated patients, thus augmenting SPV.[14] Inverse I:E ratio ventilation may cause an additional increase of SPV and decrease in preload, which might result in decreased cardiac output (CO). However, in these severely hypoxemic patients, this ventilation was warranted so as to provide minimal acceptable oxygenation.

Arterial waveform analysis, on the other hand, correlated with the findings of central hypovolemia as seen by TEE. In the euvolemic mechanically ventilated patient, SPV is about 10 mmHg and consists equally of the dUp and dDown components, as compared with the apneic pressure.[8,10] The dDown component corresponds to the transient decrease in venous return to the right ventricle during the inspiratory phase of PPV. It is therefore augmented in states of increased preload dependency of the CO, such as hypovolemia.[7,8,10] The dUp reflects, on the other hand, a transient augmentation in the left ventricular stroke volume by the mechanical breath, due to a transient increase in left ventricular preload and a decrease in left ventricular afterload.[15,16] Augmented SPVs, associated with increased dDown have been found in

hypovolemia and correlated well with changes in CO.[7,8] On the other hand, the findings of significant negative dUp also suggests the presence of hyperinflation.[17] To assist in the diagnosis of negative dUp, it is imperative to perform a prolonged enough apnea for the determination of baseline SBP, as the heart may be compressed by the hyperinflated lungs throughout the respiratory cycle (*Figure 1*).

In the present report, we demonstrated the importance of combining several modalities of hemodynamic monitoring in critically ill patients with multiple trauma due to blast injury. The use of arterial waveform analysis is simple to perform, relatively noninvasive, and may provide an accurate estimate of preload. It can be concluded that arterial waveform analysis can serve as a complementary method for the estimation of intravascular volume in the hemodynamically unstable patient being ventilated with high airway pressures.

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The Use of Systolic Pressure Variation in Hemodynamic Monitoring During Deliberate Hypotension in Spine Surgery

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Abstract

The systolic pressure variation (SPV), which is the difference between the maximal and minimal systolic blood pressure (SP) during one ventilatory cycle, was studied in ten patients during posterior spine fusion. To minimize the blood loss, deliberate hypotension to a mean blood pressure of 50 mmHg was introduced by a continuous infusion of sodium nitroprusside. SPV was further divided into two components, delta up and delta down, using SP during a short apnea as a reference point. All hemodynamic parameters were measured at the beginning of anesthesia, 15 minutes after induction of hypotension, before cessation of nitroprusside infusion, and 15 minutes after the end of the hypotensive period.

During the hypotensive period (166 ± 53 min), cardiac output (CO) decreased significantly from 4.83 ± 1.36 L/min to 3.86 ± 1.07 L/min ($p < 0.05$). Heart rate (HR), central venous pressure (CVP), and pulmonary capillary wedge pressure (PCWP) did not change during this period and bore no correlation to the changes in CO.

The only variables that changed during the hypotensive period, in addition to CO, were SPV (from 13.1 ± 4.9 mmHg to 16.9 ± 5.1 mmHg, $p < 0.02$), and delta down (from 6.0 ± 3.8 mmHg to 9.9 ± 6.3 mmHg, $p < 0.05$). The delta down segment was the only hemodynamic variable whose changes during the hypotensive period showed a significant ($p < 0.018$) correlation with the changes in OC. Delta down reflects the degree of decrease in left ventricular stroke output in response to a positive pressure breath, and thus is a sensitive indicator of preload. An increase in SPV and its delta down component during deliberate hypotension may signify the inadequacy of circulating blood volume and reflect decreased CO more effectively than can conventional hemodynamic variables.