Continuous Right Ventricular End-Diastolic Volume (CEDV) Measurements in Surgical Patients

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The critically ill post-surgical or traumatically injured patient presents a significant challenge for the intensivist due to the presence of changes in ventricular compliance and increases in intrathoracic or intra-abdominal pressure. Accurate assessment and effective resuscitation of these patients commonly requires the use of advanced hemodynamic calculations and monitoring such as is afforded by insertion of a pulmonary artery catheter. Traditional estimates of intravascular volume status, such as pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP), have been widely shown to correlate poorly with changes in cardiac output in the critically ill. Volumetric measurements of preload, such as the right ventricular end-diastolic volume index (RVEDVI), are superior to PAOP and CVP in assessing preload status and can predict patient outcome. The recent introduction of continuous end-diastolic volume (CEDV) measurements provides the clinician with previously unavailable information and insight into the complex and constantly changing hemodynamic response of patients to critical illness. Volumetric pulmonary artery catheters and RVEDVI / CEDV measurements should be utilized in the critically ill surgical patient who requires invasive hemodynamic monitoring.

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Introduction

Intravascular volume is frequently decreased in the critically ill post-surgical or traumatically injured patient as a result of hemorrhage and "third space" fluid losses. Subsequent development of sepsis or other shock state can similarly affect not only the patient's intravascular volume or "preload" status, but also their cardiac contractility and peripheral vascular "afterload". These changes directly impact upon oxygen delivery and end organ perfusion. Inadequate resuscitation and restoration of cellular oxygen delivery can result in ischemia, anaerobic metabolism, and development of multiple system organ dysfunction and failure with its high attendant mortality rate. Critically ill patients whose intravascular volumes are optimized to ensure that end organ perfusion and oxygen transport balance is restored demonstrate decreased organ failure, improved survival, and increased functional status (1-4).

Accurate assessment of intravascular volume is essential to the care of these critically ill patients. Traditional hemodynamic estimates of resuscitation adequacy such as blood pressure, heart rate, and urinary output are nonspecific and, with the introduction in 1970 of the pulmonary artery catheter by Swan and Ganz, have been replaced by invasive estimates of preload status such as pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP). Over the ensuing three decades, these intracardiac filling pressures have come to be widely regarded as the "gold standard" for preload assessment. Pressure-based estimates of intravascular volume, however, do have limitations which if unrecognized can lead to inappropriate and potentially detrimental therapy (5). This is particularly true in surgical and trauma patients who commonly demonstrate altered ventricular compliance and elevated intrathoracic and intra-abdominal pressures as occur in acute lung injury (ALI), sepsis, intra-abdominal hypertension (IAH), and abdominal compartment syndrome (ACS).
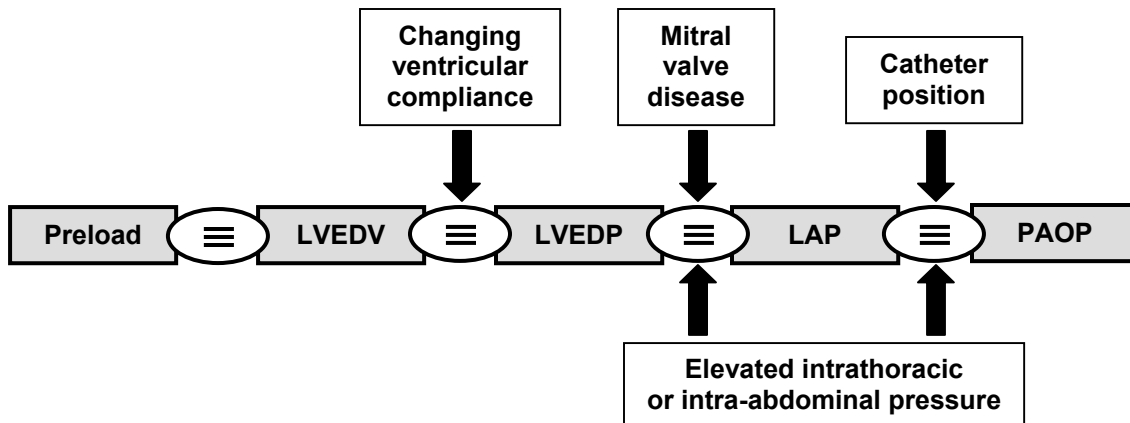
Figure 1: "The PAOP Assumption": Why intracardiac filling pressures do not accurately estimate preload status
LVEDV = left ventricular end-diastolic volume; LVEDP = left ventricular end-diastolic pressure;
LAP = left atrial pressure; PAOP = pulmonary artery occlusion pressure.
(Adapted from Cheatham ML. Right ventricular end-diastolic volume measurements in the resuscitation of trauma victims. Int J Crit Care 2000; 7:165-176.)

Preload Assessment

By the Frank-Starling principle, ventricular preload is defined as myocardial muscle fiber length at end-diastole. Ideally, the appropriate clinical correlate would be left ventricular end-diastolic volume (LVEDV), but this parameter is not easily measured on a serial basis. Assuming that ventricular compliance remains constant, changes in ventricular volume *should* be reflected by changes in ventricular pressure. Thus, pressure-based parameters such as left ventricular end-diastolic pressure (LVEDP), left atrial pressure (LAP), and PAOP have been utilized clinically as surrogate estimates of intravascular volume (Figure 1).

Although likely valid in normal healthy individuals, the multiple assumptions required to utilize PAOP and CVP as estimates of preload status are not necessarily true in the critically ill. Such patients commonly demonstrate significant aberrations in their cardiopulmonary physiology that can interfere with the accuracy of PAOP and CVP measurements as estimates of intravascular volume status. Each of these assumptions will be addressed in turn.

First, ventricular compliance is constantly changing in the critically ill, resulting in a variable relationship between pressure and volume (10-12). Changes in intracardiac pressure no longer directly reflect changes in intravascular volume, reducing the accuracy of pressure-based estimates of volume status. Disparate ventricular function as a result of acute respiratory failure-induced changes in right ventricular afterload and intrathoracic pressure may also explain, in part, the poor correlation noted between right and left sided filling pressures in the critically ill (11-14).

Second, elevated intrathoracic pressure, as a result of ALI and decreased chest wall compliance, has been demonstrated to increase PAOP and CVP measurements by an amount that is difficult to predict, further confounding the validity of intracardiac filling pressure measurements (15-17). Multiple studies have demonstrated that reliance on such measurements to guide fluid resuscitation in patients with elevated intrathoracic pressure may lead to under-resuscitation and inappropriate administration of diuretic medications (5,11,13,15,17,18). The use of either positive end-expiratory pressure (PEEP) or inverse-ratio ventilation to treat ALI-associated hypoxemia has also been demonstrated to interfere with accurate measurement of PAOP and CVP resulting in potentially misleading values that can lead to inappropriate therapy (15). Juxtacardiac pressure-corrected PAOP measurements have not been shown to improve this parameter's validity in estimating preload status in such patients (16,17).

Third, elevated intra-abdominal pressure or "intra-abdominal hypertension" (IAH) has recently been noted to increase intrathoracic pressure and complicate accurate interpretation of PAOP and CVP measurements (3-5,15,18-20). IAH can occur in the presence of visceral edema, tumors, hemoperitoneum, retroperitoneal hemorrhage, pelvic fracture, ischemic or perforated intestine, ascites, pancreatitis, burns, and abdominal packing as part of a so-called "damage control laparotomy" (19). Despite significantly elevated PAOP and CVP values,

continued fluid resuscitation commonly results in improved cardiac output and organ perfusion in patients with elevated intra-abdominal and intrathoracic pressures (3,15,18). Failure to appropriately fluid resuscitate patients based upon erroneously elevated PAOP and CVP measurements may result in under-resuscitation and may worsen end-organ perfusion and failure as these elevated intracardiac filling pressures are artificial and do not reflect the patient's true intravascular volume status (3,15,18).

Fourth, mitral valve disease can confound the use of PAOP as an estimate of intravascular volume status. Mitral valve stenosis and regurgitation both interfere with the relationship between LAP and PAOP making the latter less indicative of left ventricular preload. The presence of mitral valve disease typically results in elevated PAOP values that may lead to under-resuscitation.

Fifth, to effect a change in patient outcome, pulmonary artery catheters must be placed correctly and the derived information utilized appropriately. Several studies demonstrate that less than one-half of physicians inserting these devices are able to recognize a PAOP tracing and confirm appropriate catheter placement (21,22). This suggests that many pulmonary artery catheters may be improperly positioned, potentially providing erroneous information regarding the patient's preload status. Verification of catheter tip location using lateral chest radiographs or blood sampling has been described, but such practices are time consuming, costly, and rarely performed. Further, pulmonary artery catheter-derived hemodynamic data can only improve patient care and outcome when clinicians thoroughly understand both the appropriate utilization as well as the potential measurement errors associated with use of these parameters.

Volumetric Pulmonary Artery Catheters

In the 1980s, a new generation of pulmonary artery catheter was introduced which allowed calculation of both right ventricular ejection fraction (RVEF) and right ventricular end-diastolic volume index (RVEVDVI) (2,3,5-10,13,15,23-30). The RVEF provides information about the patient's right ventricular contractility and afterload. Knowledge of the patient's RVEF allows calculation of the RVEDVI using the following equation (where SVI = stroke volume index):

$$RVEDVI = SVI / RVEF$$

The RVEDVI is a true volumetric, as opposed to pressure-based, estimate of intravascular volume whose measurement accuracy is not subject to the effects of changing ventricular compliance and increased intrathoracic or intra-abdominal pressures as are PAOP or CVP. Initially, this technology still depended upon the traditional method of intermittent thermal injectate boluses (typically iced saline), but provided valuable hemodynamic data not previously available. In the late 1990s, the current generation of volumetric or "continuous cardiac output" (CCO) pulmonary artery catheters was introduced. These catheters differ significantly from the original "Swan-Ganz" pulmonary artery catheter in several important respects:

- A rapid response thermistor (response time 50-70 milliseconds) allows blood temperature changes to be detected more promptly and with increased accuracy
- Correlation with the patient's electrocardiogram tracing allows identification of the patient's R-R interval and measurement of beat-to-beat changes in stroke volume rather than minute-to-minute changes in cardiac output
- A surface-mounted thermal energy coil safely heats the pulmonary artery blood by 40-100 millidegrees Centigrade in a pseudo-random "on" and "off" pattern. The resulting changes in blood temperature are used to construct a continuously updated thermodilution curve of the patient's stroke volume and cardiac output. This technology obviates the need for intermittent manual injectate boluses, reduces nursing time, negates issues of injection-ventilator synchrony, and facilitates continuous assessment of patient hemodynamics.

CCO measurements have been shown to be equal in accuracy to intermittent iced injectate boluses as well as indocyanine green dye dilution techniques (36-38). The clinical accuracy of the CCO technique has been validated in comparisons to traditional radionuclide techniques (23,24), biplane angiography (26,27), and 2-D echocardiography (25). These traditional techniques can be difficult and sometimes impossible to perform in the critically ill patient and, with the exception of echocardiography, cannot be used serially to guide therapy as can CCO determinations (24,26). CCO measurements are relatively inexpensive, safe, accurate, and reproducible, allowing serial determinations of hemodynamic function to guide therapeutic interventions (1-3,15,23,24,28-30).
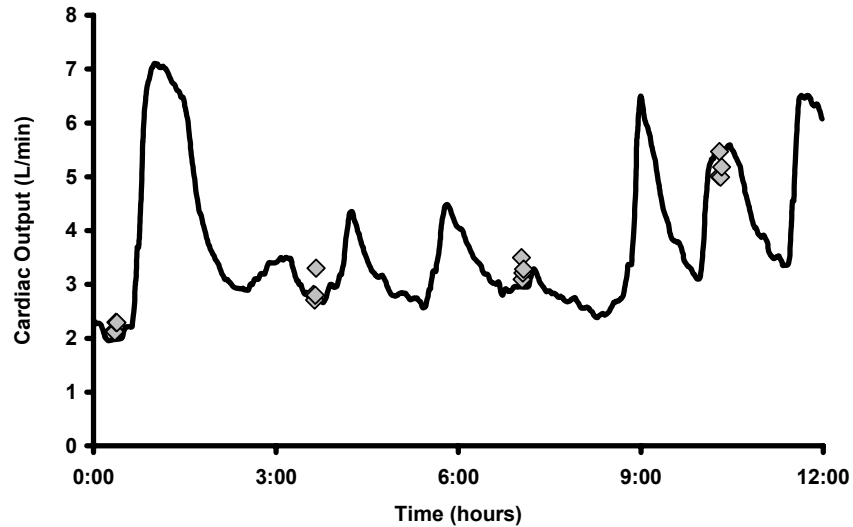
Figure 2: Continuous vs. Intermittent Cardiac Output During Patient Resuscitation

Continuous cardiac output (solid line) vs. intermittent cardiac output measurements (diamonds) during the initial resuscitation of a critically ill patient. Continuous cardiac output technology provides significant insight into the dynamic nature of the critically ill patient previously unavailable with conventional cardiac output techniques.
(Adapted from Cheatham ML. Right ventricular end-diastolic volume measurements in the resuscitation of trauma victims. Int J Crit Care 2000; 7:165-176.)

The Advantages of Continuous Cardiac Output
Since the inception of the pulmonary artery catheter, the average of three to five intermittent cardiac output measurements, obtained every four to twelve hours, has been assumed to accurately portray a patient's cardiovascular state during the intervening time. It is now recognized, however, that these intermittent measurements are only hemodynamic "snapshots" that poorly illustrate the "moving picture" of the patient's response to injury and subsequent resuscitation.

The CCO technology has several advantages over standard thermodilution techniques. First, many of the factors that may alter the accuracy of intermittent thermodilution measurements (such as injectate volume and temperature, injection technique, and injectate timing with regards to ventilation) do not play a role in the determination of CCO measurements. Thus, CCO techniques are more accurate than standard thermodilution methods (39). Second, by obviating the need for tedious thermal injectate boluses, measurement of cardiac output is possible without the significant volume load incurred by serial thermodilution measurements (36). Third, and most importantly, CCO monitoring provides the clinician with a new cardiac output measurement every 60 seconds allowing immediate assessment of patient

response to therapeutic interventions and potentially allowing more rapid and effective resuscitation.

Clinical experience with this new technology has further confirmed the dynamic and constantly changing cardiopulmonary state exhibited by these critically ill patients (Figure 2). Such hemodynamic changes may either be missed completely by intermittent thermodilution measurements or not identified until potentially devastating events have occurred. CCO technology is capable of identifying these potentially untoward changes in hemodynamic function, allowing appropriate interventions to be made at an earlier point in time. With the addition of mixed venous oximetry, this new technology provides clinicians with a continually updated, on-line assessment of oxygen transport balance and systemic perfusion by which to guide patient resuscitation, reduce organ dysfunction and failure, and improve patient outcome.

Application of Volumetric Technology
Over the past decade, multiple authors have noted the advantages of volumetric preload measurements in resuscitation of the critically ill (1-3,5-8,15,19,29,30). RVEDVI has been shown to be an accurate indicator of "preload recruitable" increases in cardiac index (CI) in a variety of patient populations and disease
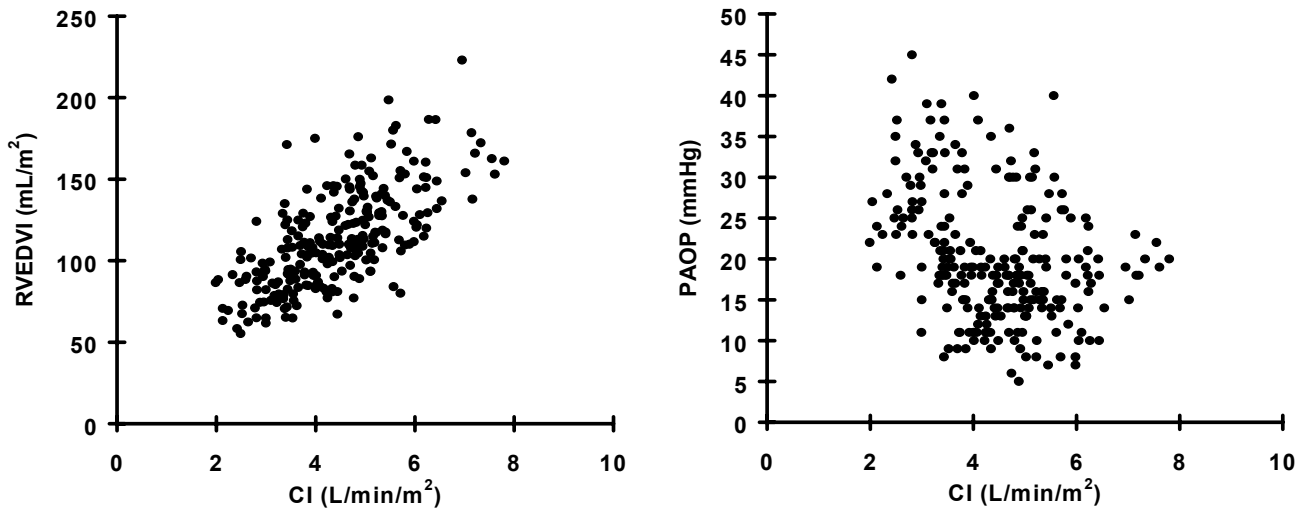
Figure 3: Cardiac Index vs. RVEDVI and PAOP in Post-Surgical Patients
CI correlates significantly with RVEDVI, but inversely with PAOP in patients with elevated intra-abdominal or intrathoracic pressure following surgical procedures. CI - cardiac index, RVEDVI - right ventricular end-diastolic volume index, PAOP - pulmonary artery occlusion pressure
(Adapted from Cheatham ML. Right ventricular end-diastolic volume measurements in the resuscitation of trauma victims. Int J Crit Care 2000; 7:165-176.)

processes including hemorrhagic, cardiogenic, neurogenic, and septic shock; ALI; pulmonary hypertension; IAH; and ACS (3,5,7,9,15,16,28,29). These studies have routinely identified a significant correlation between RVEDVI and CI and a lack of correlation between PAOP and CI during preload assessment of patients undergoing resuscitation (2,3,5,7,9,15,29).

Eddy et al. evaluated the value of RVEF as a predictor of survival following traumatic injury (10). Survivors were noted to maintain normal RVEF values post-injury while nonsurvivors exhibited a decrease in RVEF 3-6 hours post-injury that could not be improved with resuscitation. Eddy further described that traditional intracardiac filling pressure measurements were not reliable in this patient population due to decreased ventricular compliance and distortion of the normal pressure-volume relationship (10).

Subsequently, Diebel et al., Durham et al., and Chang et al. independently evaluated the use of RVEDVI as an estimate of intravascular preload in surgical and trauma patients (5,6,8,30). In each study, a significant correlation was demonstrated between CI and RVEDVI with a poor correlation between CI and both PAOP and CVP (5,6,8,9). Diebel further described that PAOP measurements provided potentially misleading information regarding preload status in 52% of their study patients (5).

Diebel et al. and Cheatham et al. assessed the impact of airway pressure and positive end-expiratory pressure (PEEP) on preload assessment in surgical and trauma patients (7,15). At levels of PEEP as high as 50 cm H_2O, CI consistently maintained a highly significant correlation with RVEDVI, while PAOP and CVP were frequently found to exhibit *inverse* correlations with CI, directly challenging the Frank-Starling principle (7,15). PAOP and CVP values as high as 60 mmHg were documented in the presence of clinical and echocardiographic evidence of intravascular volume depletion (15). Thus, these studies demonstrated that elevations in intrathoracic pressure with subsequent transmission to the pulmonary capillaries can dramatically and erroneously increase measured PAOP and CVP values.

Cheatham et al. applied volumetric technology to the preload assessment of surgical and trauma patients with elevated intra-abdominal pressure and demonstrated similar findings to those of patients with acute respiratory failure on high level PEEP (3,15). CI was noted to correlate significantly with RVEDVI and inversely with both PAOP and CVP (3) (Figure 3). Ridings et al. further confirmed the detrimental impact of elevated intra-abdominal pressure on the accuracy of PAOP and CVP measurements (18). Such findings are of particular importance as preload adequacy is an essential part of the resuscitation of patients who exhibit intra-abdominal hypertension. Intracardiac

filling pressure measurements are therefore of limited value in the hemodynamic assessment of this increasingly recognized clinical entity (19).

"Optimal" RVEDVI / CEDVI

The initial studies describing the use of volumetric pulmonary artery catheter technology described "optimal" RVEDVI values of approximately 130-140 mL/m^2, above which patients were felt to no longer respond to further volume administration with increases in CI (5,6,8). As clinical experience with volumetric technology has increased, these "optimal" values have been disputed and found to oversimplify what is actually a complex and dynamic relationship between preload, contractility, and afterload (3,15,19,40). Ongoing work has demonstrated that the patient's RVEF must be taken into consideration when assessing the adequacy of RVEDVI as a resuscitation endpoint (1,29,40).

Sarnoff et al. described cardiac contractility as a series of "ventricular function curves" (31). Each of these curves is associated with both an ejection fraction, which describes the ventricle's contractility, and an end-diastolic volume, which identifies the plateau of the patient's ventricular function curve. Resuscitation to this plateau end-diastolic volume is widely believed to optimize a patient's intravascular volume, cardiac function, and end-organ perfusion (8).

As demonstrated by Eddy, ventricular function and compliance are constantly changing in the critically ill (10). As ventricular function changes, the patient "shifts" from one RVEF-defined Starling curve to another with identification of a new, optimal plateau RVEDVI as a resuscitation endpoint (1,19,29). Thus, each RVEDVI must be considered in the context of the simultaneous RVEF measurement to determine whether the patient's right ventricular function is increasing, decreasing, or stable. In the presence of unchanging right ventricular contractility and afterload (as evidenced by a stable RVEF), RVEDVI assessment is relatively straightforward, as the target RVEDVI remains unchanged. In the critically ill patient with deteriorating right ventricular contractility or increasing right ventricular afterload, however, RVEDVI assessment becomes more complex.

By the Frank-Starling principle, as RVEF changes as a result of alterations in right ventricular contractility and afterload, the heart shifts to a different ventricular function curve and plateau RVEDVI must change proportionally assuming a constant intravascular volume state (Figure 4). Thus, whereas a RVEDVI of 100 mL/m^2 is considered normal for a RVEF of 0.40, a RVEDVI of 200 mL/m^2 would be required for a RVEF of 0.20 assuming intravascular volume has not changed. Thus, since ventricular compliance (and therefore RVEF) is subject to change in the critically ill, there cannot be a single value of RVEDVI that can be considered the goal of resuscitation for all patients. Each patient must be resuscitated to restore end-organ perfusion and function rather than to a single, arbitrary RVEDVI, PAOP, or CVP value.
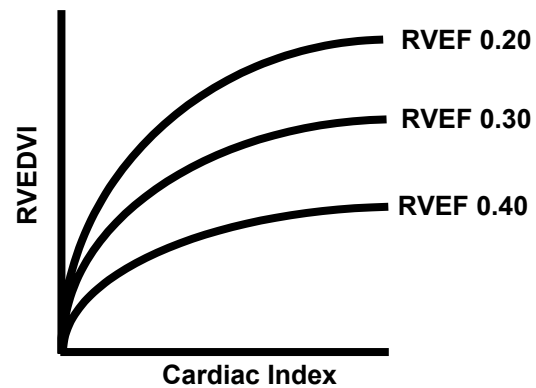

Figure 4: Ventricular Function Curves by RVEF
RVEDVI / CEDV must be interpreted in conjunction with the patient's RVEF. RVEF - right ventricular ejection fraction, RVEDVI - right ventricular end-diastolic volume index. (Adapted from Cheatham ML. Right ventricular end-diastolic volume measurements in the resuscitation of trauma victims. Int J Crit Care 2000; 7:165-176.)

Assuming a normal RVEF, what RVEDVI (or CEDVI if measured using the new CCO technology) is sufficient to optimize a traumatically injured patient's volume status? Several studies have demonstrated that certain threshold values of RVEDVI / CEDVI correlate with improved patient outcome following surgery or injury. Chang et al. prospectively documented a decreased incidence of multiple organ failure and death in patients who received aggressive volume resuscitation with maintenance of a RVEDVI greater than 110 mL/m^2 (mean RVEF 0.39) compared to those resuscitated to a RVEDVI less than 100 mL/m^2 (mean RVEF 0.39) (2). Miller et al. similarly identified improved visceral perfusion by gastric tonometry and a reduction in organ dysfunction, organ failure, and patient mortality by maintaining a RVEDVI of 120 mL/m^2 (mean RVEF 0.33) compared to 100 mL/m^2 (mean RVEF 0.34) (1). Cheatham et al. identified significantly higher RVEDVI values in patients who survived intra-abdominal hypertension and open abdominal decompression with a mean RVEDVI of 133 mL/m^2 for survivors (mean RVEF 0.37) compared to 105 mL/m^2 for non-survivors (mean RVEF 0.36) (3).

Based upon these studies, a reasonable resuscitation protocol is to initially fluid resuscitate patients to a "RVEF-corrected" RVEDVI or CEDVI of 100 mL/m^2, assuming a normal RVEF is approximately 0.40. Patients with lower RVEF measurements are resuscitated to proportionally higher RVEDVI values as per Figure 4. A patient with a RVEF of 0.30, for example, would be considered to have a *target* RVEDVI of 150 mL/m^2 and a patient with a RVEF of 0.20, a RVEDVI of 200 mL/m^2. Markers of systemic and regional perfusion adequacy such as urinary output and normalization of base deficit and arterial lactate are followed closely. If, after achieving such levels of intravascular volume, the patient continues to demonstrate signs of malperfusion, the target RVEDVI should be increased to 120-130 mL/m^2 (based upon the work of Chang et al. and Cheatham et al.) while simultaneously initiating vasoactive medications as necessary to optimize ventricular contractility and

RVEF-Corrected "Target" RVEDVI / CEDVI

RVEF	"Normal"	Critically ill
.20	200 mL/m^2	240 mL/m^2
.30	150 mL/m^2	180 mL/m^2
.35	125 mL/m^2	150 mL/m^2
.40	100 mL/m^2	120 mL/m^2
.50	50 mL/m^2	60 mL/m^2

Figure 5: RVEF-corrected "target" RVEDVI / CEDVI
These values should serve as an initial goal for resuscitation. Patients should be resuscitated to that RVEDVI / CEDVI value that restores end-organ perfusion and normalizes markers of resuscitation adequacy. This table should serve only as a guideline to direct initial patient resuscitation; subsequent resuscitation should be based upon the patient's hemodynamics and response to therapy.

afterload. The RVEF measurement is useful in determining the need for and choice of vasoactive infusion as it represents the relationship between ventricular contractility and afterload. Patients with a high RVEF will usually respond to fluid administration alone while those with a low RVEF almost invariably benefit from early administration of inotropic support. Restoration of adequate intravascular volume must precede institution of vasoactive medications, however, in order to avoid visceral malperfusion and acidosis (1). It is important to recognize that patients should be volume resuscitated to the RVEDVI that restores end organ perfusion and function and normalizes the patient's markers of resuscitation

adequacy. Patients may achieve these endpoints at RVEDVI / CEDVI values below their RVEF-corrected target value. Unnecessary over-resuscitation past this RVEDVI / CEDVI value will not benefit the patient.

Mathematical Coupling
Mathematical coupling, the interdependence of two variables when one is used to calculate the other, has been proposed to account for the significant correlation between CI and RVEDVI measurements (32,33). The relationship between two such variables is due, in part, to their common derivation and shared measurement error (34). Since RVEDVI is calculated using stroke volume, CI and RVEDVI are, by definition, mathematically coupled variables. Chang, Durham, and Nelson have separately addressed the potential impact of mathematical coupling on the reliability of RVEDVI as a measurement of preload adequacy. Chang independently measured cardiac output via indirect calorimetry and demonstrated a significant correlation between mathematically uncoupled CI and thermodilution RVEDVI (9). Durham used mathematical modeling to correct for the shared measurement error introduced by mathematical coupling and found CI to remain significantly correlated with RVEDVI (8). Nelson compared CI with RVEDVI measurements determined using two different thermodilution technologies and further confirmed the significant correlation between mathematically uncoupled CI and RVEDVI (35). Mathematical coupling does not negate the validity of RVEDVI as a predictor of intravascular volume status.

Conclusions
Volumetric estimates of preload status such as RVEDVI and CEDVI are of significant value in the assessment of traumatically injured patients. RVEDVI / CEDVI is especially useful in surgical patients with changing ventricular compliance and elevated intra-abdominal or intrathoracic pressure in whom traditional intracardiac filling pressure measurements such as PAOP and CVP may be artificially elevated and difficult to interpret. Reliance on such pressures to guide resuscitation can lead to inappropriate therapeutic decisions, under-resuscitation, and organ failure. CCO technology has the potential to significantly improve the speed and accuracy of patient resuscitation following surgery or traumatic injury. Right ventricular function pulmonary artery catheters and serial measurement of RVEDVI / CEDVI and RVEF should be considered in surgical and traumatically injured patients who require invasive hemodynamic monitoring.

Case Presentation
A 57 year-old male with a history of coronary artery disease, hypertension, congestive heart failure and previous left hemicolectomy for adenocarcinoma of the colon presents to the Emergency Room with progressive abdominal distension, pain, and obstipation. His physical examination and radiographic evaluation suggests an acute abdomen with evidence of complete small bowel obstruction.

During placement of a nasogastric tube to decompress the patient's abdomen, the patient vomits and aspirates feculent material. The patient is intubated for acute respiratory failure with further feculent material being suctioned from his endotracheal tube. He is placed on broad-spectrum antibiotics and taken to the operating room.

Intraoperatively, the patient is found to have a complete small bowel obstruction secondary to adhesions from his prior hemicolectomy. In addition, a 30 cm segment of small bowel has become necrotic as a result of the obstruction. A segmental small bowel resection is performed and the patient's abdomen is closed with some difficulty due to his significant abdominal distention.

The patient is admitted to the intensive care unit and resuscitation is initiated. The patient remains hypotensive and hypoxemic requiring increased levels of both positive end-expiratory pressure (PEEP) and FiO_2. A chest radiograph is obtained demonstrating a typical acute respiratory distress syndrome (ARDS) pattern. Due to his age, comorbidities, and failure to respond to initial resuscitation, a volumetric CCO pulmonary artery catheter is inserted. His initial hemodynamic parameters are listed in Table 1.

Question:
What is the most likely diagnosis?
  A) Hypovolemic shock
  B) Cardiogenic shock
  C) Septic shock
  D) Intra-abdominal hypertension

The patient is given two liters of crystalloid over the ensuing 1½ hours resulting in the significantly improved hemodynamic data measured at 01:30. His pulmonary function, however, continues to deteriorate. By 08:00, his hemodynamics are as depicted. His abdomen is now rigid and inspiratory pressures are rising. His intra-abdominal pressure is measured at 42 mmHg.

Question
Which of the following would be <u>most</u> appropriate?
  A) Fluid administration
  B) Diuretic administration
  C) Dopamine infusion
  D) Abdominal decompression

Table 1: Hemodynamic Parameters

	00:00	**01:30**	**08:00**	**10:00**
BP (mmHg)	82/43	122/65	70/30	130/70
PAP (mmHg)	30/21	45/30	55/41	40/31
HR (beats/min)	120	105	140	95
CI (L/min/m^2)	1.4	2.8	1.5	3.5
SVRI (dynes•cm^2/sec^{-5})	2514	2400	2311	2057
PAOP (mmHg)	18	24	40	12
CVP (mmHg)	12	15	28	10
PIP (cm H2O)	40	45	60	42
PEEP (cm H_2O)	8	10	10	10
FiO$_2$ (fraction)	0.60	1.0	1.0	0.40
SaO$_2$ (fraction)	0.92	0.94	0.82	1.0
SvO$_2$ (fraction)	0.60	0.68	0.47	0.72
RVEF (fraction)	0.40	0.42	0.20	0.39
RVEDVI (mL/m^2)	45	102	70	136
IAP (mmHg)	---	---	42	13
UOP (mL/hr)	5	30	0	60

BP – blood pressure, PAP – pulmonary artery pressure, HR – heart rate, CI – cardiac index, SVRI – systemic vascular resistance index, PAOP – pulmonary artery occlusion pressure, CVP – central venous pressure, PIP – peak inspiratory pressure, PEEP – positive end-expiratory pressure, FiO$_2$ – fraction of inspired oxygen, SaO$_2$ – arterial oxygen saturation, SvO$_2$ – mixed venous oxygen saturation, RVEF – right ventricular ejection fraction, RVEDVI – right ventricular end-diastolic volume index, IAP – intra-abdominal pressure, UOP – urinary output

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References

1. Miller PR, Meredith JW, Chang MC. Randomized, prospective comparison of increased preload versus inotropes in the resuscitation of trauma patients: Effects on cardiopulmonary function and visceral perfusion. *J Trauma* 1998; 44:107-113.
2. Chang MC, Meredith JW: Cardiac preload, splanchnic perfusion, and their relationship during resuscitation in trauma patients. *J Trauma* 1997; 42:577-584.
3. Cheatham ML, Safcsak K, Block EFJ, Nelson LD. Preload assessment in patients with an open abdomen. *J Trauma* 1999; 46:16-22.
4. Meldrum DR, Moore FA, Moore EE, Franciose RJ, Burch JM. Prospective characterization and selective management of the abdominal compartment syndrome. *Am J Surg* 1997; 174:667-673.
5. Diebel LN, Wilson RF, Tagett MG, et al: End-diastolic volume: a better indicator of preload in the critically ill. *Arch Surg* 1992; 127:817-822.
6. Diebel L, Wilson RF, Heins J, et al: End-distolic volume versus pulmonary artery wedge pressure in evaluating cardiac preload in trauma patients. *J Trauma* 1994; 37:950-955.
7. Diebel LN, Myers T, Dulchavsky S: Effects of increasing airway pressure and PEEP on the assessment of cardiac preload. *J Trauma* 1997; 42:585-591.
8. Durham R, Neunaber K, Vogler G, et al: Right ventricular end-diastolic volume as a measure of preload. *J Trauma* 1995; 39:218-224.
9. Chang MC, Black CS, Meredith JW: Volumetric assessment of preload in trauma patients: addressing the problem of mathematical coupling. *Shock* 1996; 6:326-329.
10. Eddy AC, Rice CL, Anardi DM: Right ventricular dysfunction in multiple trauma victims. *Am J Surg* 1988; 155:712-715.
11. Calvin JE, Driedger AA, Sibbald WJ: Does the pulmonary capillary wedge pressure predict left ventricular preload in critically ill patients? *Crit Care Med* 1981; 9:437-443.
12. Packman MI, Rackow EC: Optimum left heart filling pressure during fluid resuscitation of patients with hypovolemic and septic shock. *Crit Care Med* 1983; 11:165-169.
13. Martyn JA, Snider MT, Farago LF, et al: Thermodilution right ventricular volume: a novel and better predictor of volume replacement in acute thermal injury. *J Trauma* 1981; 21:619-626.
14. Civetta JM, Gabel JC, Laver MB: Disparate ventricular function in surgical patients. *Surg Forum* 1971; 22:136-139.
15. Cheatham ML, Nelson LD, Chang MC, Safcsak K. Right ventricular end-diastolic volume index as a predictor of preload status in patients on positive end-expiratory pressure. *Crit Care Med* 1998; 26:1801-1806.
16. Safcsak K, Fusco MA, Miles WS, et al: Does transmural pulmonary artery occlusion pressure (PAOP) via esophageal balloon improve prediction of ventricular preload in patients receiving positive end-expiratory pressure (PEEP)? *Crit Care Med* 1995; 23(1) suppl:A244.
17. Qvist J, Pontoppidan H, Wilson RS, et al: Hemodynamic responses to mechanical ventilation with PEEP. *Anesth* 1975; 42:45-55.
18. Ridings PC, Bloomfield GL, Blocher CR, Sugerman HJ. Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. *J Trauma* 1995; 39:1071-1075.
19. Cheatham ML. Intra-abdominal hypertension and abdominal compartment syndrome. *New Horiz* 1999; 7:96-115.
20. Cullen DJ, Coyle JP, Teplick R, Long MC. Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill patients. *Crit Care Med* 1989; 17:118-121.
21. Iberti TJ, Fischer EP, Leibowitz AB, Panacek EA, Silverstein JH, Albertson TE. A multicenter study of physicians' knowledge of the pulmonary artery catheter. Pulmonary Artery Catheter Study Group. *JAMA* 1990; Dec 12;264(22):2928-32.
22. Trottier SJ, Taylor RW. Physicians' attitudes toward and knowledge of the pulmonary artery catheter: Society of Critical Care Medicine membership survey. *New Horiz* 1997 Aug;5(3):201-6.
23. Vincent JL, Thirion M, Brimioulle S, et al: Thermodilution measurement of right ventricular ejection fraction with a modified pulmonary artery catheter. *Intensive Care Med* 1986;12:33-38.
24. Dhainaut JF, Brunet F, Monsallier JF, et al: Bedside evaluation of right ventricular performance using a rapid computerized thermodilution method. *Crit Care Med* 1987; 15:148-152.
25. Jardin F, Brun-Ney D, Hardy A, et al: Combined thermodilution and two-dimensional echocardiographic evaluation of right ventricular function during respiratory support with PEEP. *Chest* 1991; 99:162-168.
26. Urban P, Scheidegger D, Gabathuler J, et al: Thermodilution determination of right ventricular volume and ejection fraction: a comparison with biplane angiography. *Crit Care Med* 1987; 15:652-655.
27. Voelker W, Gruber HP, Ickrath O, et al: Determination of right ventricular ejection fraction by thermodilution technique - a comparison to

biplane cineventriculography. *Int Care Med* 1988;14:461-466.

28. Jardin F, Gueret P, Dubourg O, et al: Right ventricular volumes by thermodilution in the adult respiratory distress syndrome. *Chest* 1988; 88:34-39.

29. Cheatham ML, Safcsak K, Nelson LD: Right ventricular end-diastolic volume index is superior to cardiac filling pressures in determining preload status. *Surg Forum* 1994; pp. 79-81.

30. Chang MC, Blinman TA, Rutherford EJ, et al: Preload assessment in trauma patients during large-volume shock resuscitation. *Arch Surg* 1996;131:728-731.

31. Sarnoff SJ, Berglund E. Starling's law of the heart studied by means of simultaneous right and left ventricular function curves in the dog. *Circulation* 1954; 9:706.

32. McNamee JE, Abel FL: Mathematical coupling and Starling's law of the heart. *Shock* 1996; 6:330.

33. Civetta JM, Kirton O, Civetta-Hudson J, et al: Mathematical coupling: why correlations of right ventricular function are so good? *Crit Care Med* 1995; 23(1) suppl:A126.

34. Stratton HH, Feustel PJ, Newell JC. Regression of calculated variables in the presence of shared measurement error. *J Appl Physiol* 1987; 62:2083-2093.

35. Nelson LD, Safcsak K, Cheatham ML, Block EFJ. Mathematical coupling does not explain relationship between right ventricular end-diastolic volume (RVEDV) and thermodilution cardiac output (TDCO). *Crit Care Med* 1999; 27(1):A151.

36. Boldt J, Menges T, Wollbruck M, et al. Is continuous cardiac output measurement using thermodilution reliable in the critically ill patient? *Crit Care Med* 1994; 22:1913-1918.

37. Munro HM, Wood CE, Taylor BL, Smith GB. Continuous invasive cardiac output monitoring - the Baxter/Edwards Critical-Care Swan Ganz® Intellicath™ and Vigilance™ system. *Clinical Intensive Care* 1994; 5:52-55.

38. Haller M, Zollner C, Briegel J, Forst H. Evaluation of a new continuous thermodilution cardiac output monitor in critically ill patients: A prospective criterion standard study. *Crit Care Med* 1995; 23:860-866.

39. Burchell SA, Yu Mihae, Takiguchi SA, et al. Evaluation of a continuous cardiac output and mixed venous oxygen saturation catheter in critically ill surgical patients. *Crit Care Med* 1997; 25:388-391.

40. Cheatham ML. Right ventricular end-diastolic volume measurements in the resuscitation of trauma victims. *Int J Crit Care* 2000; 7:165-176.