The FloTrac System
Case Studies
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Extending Aortic Aneurysm with Ischemic Small Bowel Case Study

INTRODUCTION
As minimally invasive, easy-to-use devices for monitoring hemodynamically unstable patients are now available, the application of these devices should be considered for more efficient management of their hemodynamics. Arterial pressure-based cardiac output monitoring and central venous oximetry may enable clinicians to manage patients’ oxygen delivery more precisely than mean arterial pressure and central venous pressure alone.

CASE NOTES
The patient presented to the Emergency Department in moderate distress and with complaints of severe abdominal discomfort. The patient’s abdomen was tender on palpation and distended. A 9-centimeter abdominal aortic aneurysm was revealed on CT scan. Initial blood pressure was over 190 mmHg systolic and 104 mmHg diastolic, and he was transferred to the Medical Intensive Care Unit.

The patient was placed on an esmolol infusion for blood pressure control. On day 2, the patient’s mental status deteriorated and his pain increased. The surgical service was consulted and a follow-up CT scan was performed, revealing the high probability of an ischemic small bowel. This diagnosis was based on visualized changes on the CT scan as well as probable partial or complete occlusion of the IMA. Lactate levels were increasing. Based on the findings surgery was initially the treatment of choice but was refused as an option by the patient. On day 3, lactate level reached 4 mmol/liter and urine output was less than 20 cc in 2 hours. Antihypertensive therapy was discontinued, fluids were administered, and low-dose vasopressors for systemic perfusion were added. The patient was transferred to the surgical ICU. A new radial arterial line and arterial pressure-based cardiac output sensor (FloTrac sensor, Edwards Lifesciences, Irvine, CA, USA) was placed, along with a central venous oximetry catheter (PreSep catheter, Edwards Lifesciences, Irvine, CA, USA). Initial readings revealed a cardiac output of 4.6 l/min (cardiac index of 2.4 l/min/m²), heart rate was 113, and ScvO₂ value of 50% with a FiO₂ of 1.0. The patient was sedated, intubated, and mechanically ventilated. The patient’s urine output fell to <20 cc/hr with a 24 hour output of 210 cc. His BUN and creatinine rose accordingly to a high of 60 and 2.5. He underwent repeated CT scans.
Over the following 24 hours, the patient’s status was optimized with administration of fluid, packed red blood cells, and low dose vasopressin. Cardiac output increased to 5.2 l/min and ScvO₂ increased to 80%. The figure represents the trends of cardiac output (CO) and central venous saturation (ScvO₂) for the resuscitation period. Within 48 hours, the patient was extubated and the patient was transferred from the ICU on the following day (admission day 8). Upon transfer, laboratory values revealed no renal complications. Liver function was adequate and lactate levels had returned to normal. The patient was discharged from the hospital on day 12.

Table 1: Comparison of key cost parameters between average patients with this diagnosis and this case

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<thead>
<tr>
<th>Parameter</th>
<th>Average</th>
<th>Case Study</th>
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<td>Hospital length of stay</td>
<td>25</td>
<td>12</td>
</tr>
<tr>
<td>ICU length of stay</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>Ventilator days</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Renal complication rate</td>
<td>24%</td>
<td>none</td>
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Retrospective analysis revealed that the average ICU length of stay for a critically-ill patient with small bowel ischemia at this institution is 15 days and the average hospital length of stay is 25 days. Other key cost parameters are shown in Table 1. Comparing aggregate cost data with the cost for this patient revealed a gross savings of approximately $56,000.

**DISCUSSION**

The introduction of less invasive, easy-to-use hemodynamic monitoring devices increases the options available for more effectively managing critically ill patients. This case illustrates the utility of continuously monitoring the fluid status and oxygen consumption of a patient who otherwise would not have been monitored in order to avoid placing a more invasive device. Use of this increased level of monitoring and the resulting treatment regimen demonstrated a significant reduction in healthcare resources in this case study.

Submitted by:
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**INTRODUCTION**

Fluid resuscitation in severely critical patients can be challenging, particularly when the patient is in cardiogenic shock with pulmonary edema and hypoxia needing mechanical ventilation. Traditional hemodynamic monitoring parameters obtained via a pulmonary artery catheter do not address the issue of fluid responsiveness in patients on mechanical ventilation. In this case study, we describe the use of stroke volume variation (SVV) monitoring, which guided our decision to add fluids in order to improve cardiac output (CO). SVV guided fluid optimization led to a successful outcome in this seriously ill patient.

**CASE NOTES**

The patient was admitted with chief complaints of progressive dyspnea even at rest (NYHA Class IV heart failure) and swelling of feet. He was in sinus rhythm, his heart rate was 90 beats/minute, blood pressure was 108/60 mmHg, (mean arterial pressure 78 mmHg), respiratory rate was 22/minute. He was treated with intravenous diuretics, nitroglycerin, dobutamine and mask oxygen. The patient was evaluated and referred for cardiac resynchronization therapy (CRT).

**Investigations before CRT:**
- Hemogram: Hb – 11.9 gm%, total leucocyte count (TLC) – 11,000/cmm, platelets – 252,000/cmm
- Blood urea levels (BUL) – 24 mg%, serum creatinine – 1.1mg%, serum sodium – 132 mEq/L, serum potassium – 3.5 mEq/L, serum chloride – 97 mEq/L
- Blood sugar levels were well controlled on insulin
- ECG showed LBBB pattern with QRS duration of 146 msec
- Chest x-ray showed cardiomegaly with bilateral lower zone haziness suggestive of pulmonary edema

**Clinical Events**

Patient details: 32-year-old male

Medical history: Diagnoses: type 2 diabetes; idiopathic dilated cardiomyopathy with left ventricular ejection fraction (LVEF) of 20%. Receiving optimal anti-heart failure therapy of torsemide, ramipril, carvedilol, digoxin, plus insulin

- Pre-procedure echocardiographic evaluation showed dilated cardiomyopathy with global hypokinesia, LVEF 20%, Grade I mitral regurgitation with mild pulmonary hypertension with pulmonary arterial systolic pressure (PH) of 38 mm

**CRT procedure:**
The patient underwent CRT in the form of bi-ventricular pacing under general anesthesia. Drugs used during the procedure were fentanyl 100 mcg, midazolam 4 mg, and ketamine 50 mg for induction and propofol 1-1.2 mg/min for maintenance. Total procedure time was 5 hours. Patient required dopamine 5-8 mcg/kg/min throughout the procedure to maintain blood pressure. Central venous pressure (CVP) and intra-arterial blood pressure monitoring was performed throughout the procedure.

Advanced hemodynamic monitoring (such as PA catheter, arterial pulse based cardiac output, transpulmonary thermodilution methods or transesophageal echo-Doppler) was not used during the procedure.
Ventilatory requirements during the procedure were high due to cardiogenic pulmonary edema. Patient received 60 mg of furosemide during the procedure. Patient was ventilated with pressure-controlled ventilation, peak inspiratory pressure of 35 cm, PEEP of 10 cm, and FiO2 of 1. Intra-operative ABG values were: pH – 7.19, PO2 – 62 mm, PCO2 – 74 mm, HCO3 – 28.3 mmol/L. Intra-operative blood loss was 300 ml and urine output was 1200 ml. The patient received 500 ml of Ringer’s lactate during the procedure.

Post-CRT:
After the CRT procedure, the patient was moved to the Intensive Care Unit (ICU). Hemodynamic and ventilatory issues identified in the ICU were:

• HR 130/min, sinus rhythm, BP was 84/50 mmHg on dopamine 14 mcg/kg/min, MAP was 61 mmHg
• Severe hypoxia. Volume-controlled ventilation, tidal volume - 500 ml, respiratory rate – 18/min, needing PEEP of 12 cm with FiO2 of 0.7, peak inspiratory pressure reaching 41 cm with plateau pressure of 32 cm
• CVP was persistently in the 18 to 20 mmHg range
• 1000 ml negative fluid balance while in operating room

At this point, our therapeutic dilemmas were:
1. If we give fluids to correct negative fluid balance, there was risk of aggravation of pulmonary edema, as the patient was already extremely hypoxic. Secondly what would be our goal or target for fluid therapy?
2. If we escalate dopamine to achieve higher perfusion pressure, there is risk of worsening of tachycardia and further deterioration of cardiac function, plus the risk of high-dose dopamine in terms of renal hypoperfusion and subsequent renal impairment.
3. To achieve afterload reduction and improvement in cardiogenic pulmonary edema, nitroglycerin or nitroprusside could not be given, as the BP was very low (same concern about dobutamine).
4. At such low MAP, would diuresis have an effect?

We decided to monitor cardiac output, cardiac index, systemic vascular resistance index (SVRI) and stroke volume variation (SVV) for better hemodynamic monitoring to get answers to these questions:
1. Is cardiac output adequate?
2. What is peripheral vascular resistance?
3. Is this shock fluid-responsive?

A radial arterial line was connected to the FloTrac system and hemodynamic variables displayed on the Vigileo monitor (see table). There were no arrhythmias. The patient was under deep sedation and paralysis on controlled mechanical ventilation. We obtained repeated SVV values by making Vt 8 ml/kg. SVV was between 20% and 22%.

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<td>CI</td>
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<td>SVV</td>
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<td>SVRI</td>
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We felt that this information offered significant and fairly reliable evidence of preload dependency of this hemodynamic insufficiency. Therefore, we administered the first fluid bolus of 250 ml of 0.9% isotonic NaCl in 30 minutes, which resulted in marginal improvement in CO to 5 l/min, and MAP increased to 66 mm. Fluid boluses were continued until SVV dropped to < 15% on controlled ventilation. A total of 2450 ml fluids were given in the 14 hours post-operatively. MAP improved with fluid boluses and dopamine was rapidly tapered to 5 mcg/kg/min at the end of 6 hours in the ICU. HR settled to 100/min. Dobutamine was started at 5 mcg/kg/min to improve cardiac output and nitroglycerin was administered. Once hemodynamic stability was achieved, 40 mg of furosemide was given. The patient’s ventilatory requirements reduced rapidly and he was extubated 24 hours after ICU admission. Dobutamine was continued for the next 24 hours and tapered gradually. Ramipril and digoxin were restarted. BSLs were controlled with insulin infusion. Five days post-procedure, cardiac evaluation via 2D echocardiography and Doppler showed improved LV function to 25%, and PH was marginally reduced. The patient was discharged from the hospital on day 7.

DISCUSSION
We feel that CO, CI and SVV monitoring helped us immensely in improving this patient’s critical hemodynamic condition. If we had monitored only MAP, CVP or PCWP in this kind of acute cardiogenic pulmonary edema plus cardiogenic shock, fluid resuscitation would not have been attempted, and the outcome would likely have been far less successful. Instead, with the indication that the patient would respond positively, we could proceed confidently with fluid replacement. Further, the SVV parameter guided us regarding when to stop administering fluids.

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Hemodynamic Instability During Endovascular Abdominal Aortic Aneurysm Repair
Case Study

INTRODUCTION
Minimally invasive techniques are becoming common, particularly in vascular surgery. These techniques tend to avoid large incisions and fluid shifts, as well as minimizing blood loss. Nonetheless, these procedures can sometimes be associated with marked, unexpected hemodynamic changes. Arterial pressure-based cardiac output monitoring with stroke volume variation assessment can be particularly valuable in these frail patients, particularly when surgical complications occur.

CASE NOTES
The patient was a 78-year-old man with a 6 cm abdominal aortic aneurysm (AAA), presenting for endovascular repair (stent placement). His medical history was remarkable for chronic hypertension and atherosclerotic heart disease. Our plan was to provide general anesthesia with endotracheal intubation, intra-arterial pressure monitoring (radial artery) and minimally invasive cardiac output monitoring (Edwards FloTrac sensor, Edwards Lifesciences LLC, Irvine, CA). After a stable anesthetic induction, blood pressure was 136/78 and heart rate 64 BPM. Cardiac output (CO) was 4.5 L/min, and stroke volume variation (SVV) was 7%. The patient’s oxygen saturation by pulse oximeter was 99% on 50% oxygen and hematocrit was 38%. The surgeons made a small incision in the right groin area, exposing the right femoral artery. They placed a catheter sheath into the artery under direct vision, with the goal of placing a catheter with stent into the abdominal aorta under fluoroscopic guidance.
One hour into what appeared to be an uncomplicated procedure, the patient’s CO dropped to 3.1 L/min, and his SVV increased to 35% (Figure 1). His heart rate had increased to 76 BPM, and his blood pressure was 110/60. Based on this information we made the diagnosis of acute hypovolemia, fearing accidental surgical disruption of the aorta. The surgeons performed a dye study showing that the aorta was actually still intact with no leakage. They suggested we search for another diagnosis. Nonetheless, we began aggressive volume resuscitation with colloid (5% albumin) and crystalloid solutions. Covert blood loss was identified from the surgical site beneath sterile drapes with saturated bed linens (estimated blood loss about 750 ml). After the administration of three units of packed red blood cells, 500 ml albumin, and 500 ml crystalloid solutions, the patient’s heart rate, blood pressure, CO, and SVV returned to baseline values. The remainder of the surgery and hospital course were uneventful; the patient was discharged to home the following day.

**DISCUSSION**

Minimally invasive vascular surgery is associated with decreased patient morbidity and resource utilization as compared with conventional “open” surgery. The procedures, however, are typically performed on elderly patients with coexisting cardiovascular disease. Thus, careful hemodynamic monitoring with aggressive, early treatment of abnormalities is essential. In this case, the sudden drop in CO, together with the rise in SVV “tipped us off” to the rapid development of hypovolemia. In particular, the rise in SVV indicating a probable decrease in circulating blood volume. The moderate rise in heart rate and mild drop in blood pressure might themselves have given us a hint, but these findings can be nonspecific during general anesthesia, as anesthesia itself can cause such conditions. We were concerned about possible disruption of the aorta, and were pleased to learn the problem was much easier to fix: leakage around the catheterization site at the femoral artery. Nonetheless, the situation was potentially life-threatening, and use of minimally invasive CO assessment with SVV allowed rapid diagnosis and carefully titrated fluid resuscitation.

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Hemodynamic Monitoring for the High Risk Patient Undergoing a Repeat Bilateral Total Knee Replacement Case Study

INTRODUCTION

Patients with left ventricular dysfunction due to ischemic heart disease are at higher risk of perioperative complications when undergoing non-cardiac surgery. These patients are frequently on many long-term cardiogenic and vasoactive medications. As a result, they may require extensive monitoring to facilitate optimal outcome. Knowledge of hemodynamic parameters such as cardiac index (CI) and systemic vascular resistance index (SVRI) can help in managing the high risk surgical patient and provide insight to their postoperative care.

CASE NOTES

The patient presenting with severe left ventricular dysfunction required repeat bilateral knee replacements. Because of observed religious practices, the patient refused transfusion of blood or blood products. Simultaneous bilateral knee replacement was scheduled per institutional protocol.

Pre-Operative

Upon admission, the patient’s blood pressure was 110/80 mmHg and hemoglobin was 10 G/dL. Routine laboratory examination was normal. Echocardiogram showed severe left ventricular dysfunction (ejection fraction 25%), moderate mitral regurgitation, severe tricuspid regurgitation and pulmonary hypertension (65 mmHg). Coronary angiogram showed diffuse disease and the patient was not a candidate for percutaneous coronary interventions or CABG surgery. The patient’s daily medications at admission were as follows: 50 mg tablet atenolol, insulin as per blood sugar, nimusulide tablet 3x, 10 mg tablet prednisolone 2x, 2.5 mg lisonopril tablet, and allopurinol tablet. She had also been taking clopidogrel and aspirin, both discontinued one week prior to surgery. All medications except atenolol were stopped one day prior to surgery. The patient preferred general anesthesia.

Clinical Events

Patient details: 72-year-old female, height: 175 cm, weight: 92 kg, BSA: 2.08
Medical history: Hypertension for 32 years, diabetes mellitus, rheumatoid arthritis, gout, multiple orthopedic surgeries

Causes of potential anesthetic complications:

a. Long standing hypertension
b. Severe left ventricular dysfunction
c. Severe coronary artery disease
d. Chronic insulin dependent diabetes mellitus
e. Chronic steroid consumption
f. Use of anticoagulant, angiotensin converting enzyme (ACE) inhibitor medications
g. Anticipated challenges due to tourniquet application and release
h. Long duration of surgery (over four hours)
i. Inability to use blood or blood products

General anesthesia was administered while monitoring heart rate (HR), mean arterial pressure (MAP), central venous pressure (CVP), CI, core temperature, oxygen saturation, end tidal carbon dioxide, blood loss, urine output and arterial blood gas estimation. The FloTrac system was selected for continuous cardiac output monitoring because tricuspid regurgitation and pulmonary hypertension can cause the pulmonary artery catheter to report erroneous values as well as make it difficult to place.
200 mg of hydrocortisone hemisuccinate was administered intravenously one hour prior to surgery and 200 µgms of fentanyl was administered thirty minutes prior to surgery. Right internal jugular vein was cannulated with 7 French triple lumen catheter and left radial artery with 20 G cannula. CI was monitored by connecting the FloTrac sensor to the arterial line. The baseline hemodynamic data are shown in Table 1. General endotracheal anesthesia was commenced using 150 mg of propofol, 100 mg of rocuronium bromide.

### Intra-Operative

Left knee replacement was completed in a tourniquet time of 90 minutes. Upon tourniquet release, the patient became hypotensive (MAP of 40 mmHg). Dopamine was infused at 5 µgms/kg/min and CI increased immediately, while MAP and SVRI continued to be low. There was concern about the ongoing low perfusion pressure in view of the coronary artery disease. Decreased organ perfusion was evident from low urine output. Vasopressin infusion was started and increases in MAP and SVRI were noted immediately. Ultimately, improvement in tissue perfusion was noticed by an increase in urine output. The hemodynamic changes can be seen in Table 1. Following the commencement of vasopressin administration, hemodynamic parameters returned to normal and no further problems were experienced when the second tourniquet was released. The total blood loss in the intraoperative period was 350 mL as estimated by the gravimetric method.

### Related Publications


### DISCUSSION

It is becoming more and more common to see patients presenting for surgery having multiple medical problems and who are taking multiple medications. As a result, advanced hemodynamic monitoring in the perioperative period for high risk patients is pivotal for better surgical outcomes. In this case, the FloTrac system provided insight used to improve tissue perfusion through pharmacological selection and titration. Although similar benefits may be obtained using a Swan-Ganz catheter, with this patient it was anticipated that pulmonary artery catheter placement would be difficult in the presence of tricuspid regurgitation and pulmonary hypertension in addition to the potential for inaccurate data. The high CI and low SVRI seen most likely came as a result of the preoperative use of ACE inhibitors and intra-operative tourniquet release. This situation can be difficult to diagnose without CI monitoring because traditional vital signs may be misleading. ACE inhibitors are commonly used due to the overwhelming evidence showing improved outcomes when they are administered. Although most ACE inhibitors currently available are short acting, their residual action may decrease SVRI. This condition may further deteriorate in the intraoperative period with the use of inhalational anesthetic agents or tourniquet release as was seen. Using vasopressors, which can potentially cause coronary vasoconstriction, may be debatable in patients with coronary artery disease, however the patient’s low SVRI helped make that decision. This case study highlights the need for and benefits of advanced hemodynamic monitoring techniques in day-to-day practice. FloTrac system provides advanced hemodynamic information that can be used to manage complex, high risk patients such as this one. The insight into cardiac function and vascular tone as delivered by the FloTrac system gave specific and sensitive information from which the patient’s care was optimized.

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INTRODUCTION
The most commonly used method of measuring a patient’s cardiac output involves placement of a catheter in the pulmonary artery and performing thermodilution measurements. Although, this method is accurate under most clinical conditions, it is invasive. A minimally invasive and simple method of monitoring cardiac output would be invaluable in high-risk critically ill patients undergoing surgery.

Clinical Events
Patient details: 54-year-old male, 170 cm, 74 kg, BSA 1.87 m²
Medical history: Hypertension, diabetes mellitus, and coronary artery disease with poor left ventricular ejection fraction of 35%

CASE NOTES
This patient arrived in the operating room for an elective liver resection for hepatocellular carcinoma. Vital signs prior to surgery were as follows: blood pressure (BP) 150/85; heart rate (HR) 66/min; and respiratory rate 14/min. General anesthesia with endotracheal intubation was performed after induction with intravenous propofol 2 mg/kg and muscle paralysis with rocuronium 0.9 mg/kg. Monitors were placed after induction and included continuous arterial blood pressure and central venous pressure (CVP) monitoring. Initial CVP was 8 mmHg. Anesthesia was maintained with isoflurane in oxygen and nitrous oxide.

Two hours into surgery, the anesthesiologist noted changes in the patient’s vital signs. BP decreased to 95/44. Electrocardiogram showed a HR of 126/min, sinus tachycardia with no ST segment changes. CVP was 7 mmHg. Blood loss thus far was approximately 600 ml. Urine output was 30 ml from the start of surgery. The anesthesiologist administered a fluid challenge of 500 ml colloid solution with no improvement in hemo-dynamic parameters.
A cardiac output sensor (Edwards FloTrac sensor, Edwards Lifesciences, Irvine, CA, USA) was connected to the existing arterial line. Initial readings revealed a cardiac output (CO) of 1.9 L/min (cardiac index of 1.0 L/min/m²). Stroke volume (SV) was decreased at 25 ml (stroke volume index 13 ml/m²) and stroke volume variation (SVV) was 20% (Figure 1). Additional fluid boluses of 250 ml each (total 750 mL) colloid solution increased the CO to 2.5 L/min. SVV improved to 10% but SV remained low at 40 ml. BP was 99/54 and HR was 110/min.

An intravenous infusion of epinephrine was therefore started at 0.05 mcg/kg/min and gradually escalated to 0.1 mcg/kg/min according to hemodynamic parameters. CO increased to 5.1 L/min with return of vital signs to near normal values (BP 125/67, HR 102/min, CVP 6 mmHg). At the end of surgery, patient remained intubated and ventilated and was transferred to the Intensive Care Unit for postoperative care. He was extubated the following day. Laboratory tests for cardiac enzymes performed in Intensive Care on the day of surgery showed raised troponin I and creatine kinase MB fraction levels consistent with myocardial ischemia.

**DISCUSSION**

This case demonstrates the usefulness of a less invasive, easy to use continuous CO monitor in the operating room. In this clinical setting where significant blood loss is usually associated with liver resection, the initial management is commonly fluid resuscitation to replace the effective circulating blood volume. When hemodynamic parameters do not improve, persisting with additional fluid loading may be detrimental as this patient has poor left ventricular function. Injudicious and excessive fluid replacement may lead to congestive heart failure. When initial fluid challenge did not improve this patient’s BP, measurement using the FloTrac sensor revealed a low CO and SV and a SVV of more than 10%. Hypovolemia is associated with an exaggerated SVV. Despite improvement in the SVV with additional fluid, CO remained low. This was indicative of poor cardiac contractility. Therefore, the correct intervention was inotropic support with an intravenous infusion of epinephrine to improve cardiac function.

The FloTrac sensor is a less invasive hemodynamic monitoring device that can be used to monitor continuous CO, SV and SVV measurement through an arterial pressure line. It is useful in the perioperative environment and allows monitoring of high-risk patients with significant cardiac disease undergoing major surgery with potentially large fluid shifts and blood loss. It is also used to guide inotropic therapy and facilitate precise titration of cardiovascular medication in real time.

Submitted by:
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INTRODUCTION
As minimally invasive, easy-to-use devices for monitoring hemodynamically unstable patients are more readily available, the application of these devices should be considered for more efficient hemodynamic management. The Edwards FloTrac sensor arterial pressure-based cardiac output (APCO) monitors an important component of oxygen delivery. Monitoring cardiac output and appropriately managing its components (HR, preload, afterload, and contractility) allows for a more precise and appropriate treatment than traditional vital signs alone can provide. Stroke volume variation (SVV), a parameter available with the Edwards Vigileo monitor, has been shown to be a sensitive indicator of a patient’s preload responsiveness and can help guide the clinicians’ management of volume resuscitation.1

Clinical Events
Patient details: 67-year-old female, 157.5 cm, 136 kg
Medical history: Morbidly obese, coronary artery disease with prior MI, CHF, ejection fraction 20%, aortic stenosis, obstructive sleep apnea, and untreated hypertension

Figure 1
CASE NOTES
This patient experienced the emergent repair of a lacerated left anterior femoral circumflex artery secondary to intertrochanteric hip fracture and hip repair. The case was classified as an emergent ASA 4. Invasive monitoring included a 14 gauge peripheral intravenous catheter, 8.5 Fr. 4-lumen right internal jugular central venous catheter and a 20 gauge 1-1/4 inch right radial arterial line for arterial pressure and continuous cardiac output monitoring using the FloTrac sensor and Vigileo monitor. The patient experienced an estimated blood loss of more than 6 liters during a total surgical time of 4.5 hours. The patient was resuscitated with 8,000 cc of crystalloids, 750 cc fresh frozen plasma, and 2560 cc of packed red blood cells. Additional support included vasopressor (phenylephrine 50-150 µg/min) and inotropic support with (epinephrine 10-16 µg/min). Patient maintained a urine output of 108 cc/hr CVP starting at 23 and ending at 14 mmHg (lowest 7 highest 24); CI starting at 2.6 and ending at 2.3 L/min/m² (lowest 1.6 highest 2.8); SVV starting at 15% and ending at 10% (lowest 5% highest 25%).

DISCUSSION
The application of this less invasive, easy-to-use hemodynamic monitoring device allowed for the early identification of left ventricular failure and optimization of oxygen delivery during high and continuous blood loss along with compromised cardiac performance. Volume resuscitation was precisely guided by using SVV (<10%) as a guide for preload responsiveness and cardiac index (>2.2 L/m²/min) as an indication of overall cardiac performance. Note the rising SVV when excessive blood loss began followed by a drip after volume resuscitation (see hours 2-4 in figure 1). Using both continuous SVV and CI provided confidence in the direction and magnitude of therapy given.

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Minimally Invasive Monitoring Necrotizing Fasciitis Case Study

INTRODUCTION
The availability of a minimally invasive device that can be easily set up to monitor critically ill patients in an emergency situation is extremely valuable. In addition to monitoring basic hemodynamic parameters such as blood pressure and heart rate, such devices allow more sophisticated monitoring of other important cardiovascular parameters such as cardiac output, central venous oxygen saturation, stroke volume variation, and systemic vascular resistance.

Clinical Events
Patient details: 66-year-old female, 168 cm, 99 kg, 2.15 m²
Medical history: Poorly controlled diabetes mellitus, not compliant with insulin therapy; rheumatoid arthritis on long-term steroid therapy

CASE NOTES
This patient presents with a four day history of pain and swelling in her right foot. On examination, she appeared lethargic with a temperature of 38.4°C. Blood pressure (BP) was 100/55, heart rate (HR) 120/min and respiratory rate (RR) 22/min. Her right foot was edematous, indurated and tender with ascending inflammation involving her calf that was extending to her knee. A probable diagnosis of necrotizing fasciitis was made based on the history and clinical findings.

She arrived in the operating room for an emergency exploration and debridement of her right lower extremity. A fluid bolus of 500 ml Lactate Ringer’s solution was administered prior to induction of anesthesia as the patient was hypotensive and tachycardic. A rapid sequence induction was performed using intravenous etomidate 2 mg/kg and suxamethonium 1 mg/kg. The trachea was intubated and anesthesia was maintained with isoflurane in 2 L/min of oxygen and 1 L/min of nitrous oxide.
When surgical incision was made on the foot, extensive necrosis extending proximally was found in the soft tissue and muscle. Thirty minutes into surgery, the patient’s hemodynamic parameters deteriorated. BP decreased to 85/40. Electrocardiogram showed a HR of 136/min, sinus tachycardia with a 1-mm ST segment depression. Blood loss was approximately 300 ml. Despite repeated fluid challenge with colloids as well as 1 unit of packed red blood cells, the patient’s vital signs remained unchanged.

A radial arterial line with a cardiac output sensor (Edwards FloTrac sensor, Edwards Lifesciences, Irvine, CA, USA) was inserted. A central venous catheter with a central venous oxygen saturation sensor (Edwards PreSep central venous oximetry catheter, Edwards Lifesciences, Irvine, CA, USA) was also placed. Initial readings revealed a cardiac output (CO) of 3.0 L/min (cardiac index of 1.4 L/min/m²), stroke volume (SV) of 45 ml (stroke volume index of 21 ml/m²) and stroke volume variation (SVV) of 15% (Figure 1). ScvO₂ was 65%. Additional colloid solution was administered to a total volume of 700 ml. SVV improved to 8% with slight elevations in CO to 3.5 L/min and SV to 60 ml. The Edwards Vigileo monitor also showed the calculated systemic vascular resistance to be low at 550 dyne-sec/cm⁵ (systemic vascular resistance index, SVRI, of 256 dyne-sec/cm⁵/m²) using the Vigileo monitor on-board derived calculator.

An intravenous infusion of norepinephrine was therefore started at 0.04 mcg/kg/min and titrated upwards according to the patient’s hemodynamic parameters. CO improved and remained at about 4.8 L/min while vital signs began to normalize. BP was 110/68 and HR was 105/min. Surgery was completed in 1 hour. In view of patient’s critical condition, she was kept intubated and ventilated and was transferred to the Intensive Care Unit for postoperative stabilization. Intravenous penicillin G, gentamicin and metronidazole were started pending bacterial culture and sensitivity testing from the wound and blood. Over the next 24 hours, the patient’s status was optimized with a combination of antibiotics, fluid and vasopressors. CO increased to 5.1 L/min, ScvO₂ increased to 80% and SVR increased to 1300 dyne-sec/cm⁵. On the second postoperative day, the patient appeared to respond to the antibiotic therapy. She was gradually weaned off the vasopressor.

**DISCUSSION**

This case demonstrates the usefulness of a minimally invasive continuous CO monitor in a perioperative setting. In this example, the initial measurements of low CO, SV and large SVV were consistent with hypovolemia. Careful fluid resuscitation did not completely correct the hemodynamic changes as the hypotension was also contributed by the low SVR.

The FloTrac sensor was helpful in excluding cardiogenic and hypovolemic causes of shock. It was also useful for monitoring of systemic vascular resistance. An extremely low SVR shown by the monitor was consistent with septic shock. The clinical picture of septic shock was expected with necrotizing fasciitis. After adequate fluid loading, the correct intervention using intravenous norepinephrine infusion was therefore instituted to increase peripheral vascular resistance to treat hypotension. The continuous monitoring of ScvO₂ enables close tracking of the patient’s progress and the adequacy of oxygen delivery against oxygen consumption.

Submitted by:
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Minimally Invasive Monitoring
Severe Pneumonia and Sepsis
Case Study

INTRODUCTION
The use of minimally invasive monitoring in the care of ICU patients is useful in directing the appropriate course of therapy, as well as the extent of that therapy. Traditional monitoring parameters can be affected by compensatory mechanisms and therefore flow-based parameters (cardiac output, central or mixed venous oxygen saturation, lactate) are better indicators of adequate oxygen delivery. Stroke volume variation (SVV), a parameter available with minimally invasive cardiac output monitoring, has been demonstrated to be a sensitive parameter in determining a patient's preload responsiveness.

CASE NOTES
His treatment included immediate intubation and ventilation on PSIMV, with an inspiratory pressure of 25, PEEP of 10 and respiratory rate of 15. He was sedated on 40 mg propofol 1% and 2 mg of alfentanil. He made no respiratory effort during the case study hour. Following insertion and checking the position of invasive lines (left IJ CVC and right radial arterial line) along with additional flow monitoring from the Edwards Vigileo monitor and Edwards FloTrac sensor, the patient was started on noradrenaline at 0.5 mg/kg/min and given fluid challenges to achieve a stroke volume variation (SVV) < 10%. Initially, his stroke volume variation was 18%.

Clinical Events
A 78-year-old male (85 kg) was admitted with severe sepsis secondary to left lower lobe pneumonia. He had a previous history of pulmonary TB 30 years ago. On admission his respiratory rate was 42 and he was using his accessory muscles. Other observations included: Temp 39.1; MAP 58; HR 135. Urine output had been 10/20/5 over the last 3 hours.

On physical examination he was cool peripherally with clammy skin. His GCS was 10. His abdomen was distended and it was noted that he had not been absorbing his nasogastric feed. His arterial blood gases were: PH 7.30; P02 78 on FiO2 .70; PC02 41; ABE –10.3; Lactate 4.2; Na+ 127; K+ 4.7; Cl- 88

Over the next 7 hours fluids (gelofusin) were given in boluses to maintain a SVV < 10%. In addition, maintenance fluid at 1.5 ml/kg/hr was given. His urine output picked up, lactate came down and his ventilation improved. Subsequent investigations included: bronchoscope & lavage (gram – ve cocci); blood cultures; CT abdomen & pelvis – which was NAD; a blood film which suggested overwhelming sepsis. Over the next 6 days he was weaned, then successfully extubated. Antibiotic therapy was continued for 10 days.
DISCUSSION

The use of SVV, as provided by the Vigileo monitor, was helpful in guiding aggressive but appropriate volume resuscitation.

Submitted by:
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MINIMALLY INVASIVE MOTOR VEHICLE ACCIDENT TRAUMA CASE STUDY

INTRODUCTION
With new minimally invasive monitoring tools available such as the Edwards FloTrac sensor the monitoring of cardiac output is easy to apply. However, no monitoring tool will impact outcome unless it is combined with a treatment algorithm. A simple algorithm was developed to be used with this minimally invasive technology. The following is an example of the application of this algorithm in a trauma patient.

CASE NOTES
An 18-year-old male was involved in a motor vehicle accident with a prolonged extraction by emergency rescue personnel. The patient displayed shock and stupor on evaluation at the scene and was immediately intubated. Systolic blood pressure was 70 with a heart rate of 160. Abdomen was firm and pelvic fracture, including left iliac wing, was identified. Upon presentation to the Emergency Department an arterial line with a FloTrac sensor was initiated showing a stroke volume index (SVI) of 14ml/m² and a stroke volume variation (SVV) of 40-45%. The patient was resuscitated by the trauma team with aggressive volume replacement using packed red blood cells (PRBC) and crystalloid infusion and brought immediately to the operating room. While in the operating room the patient received 12 units of PRBC and 14 liters of NaCl. The patient underwent a splenectomy and superior mesenteric vein repair. Intra-operatively the patient was monitored with traditional parameters (ECG, SpO₂, EtCO₂, and arterial pressure). In addition the FloTrac sensor and Edwards Vigileo monitor were used for continuous SVI and SVV assessment. Target values of an SVV<13% and a SVI of 45 ml/m² coupled with Simplified Physiologic Protocol (Figure 1) was used intra-operatively and in the ICU. After 2 hours in surgery the patient was brought to the ICU.

Post-operatively the patient continued to be volume resuscitated with 23 liters of NaCl and 16 units of PRBC guided by SVI and SVV on the Vigileo monitor using the Simplified Physiologic Protocol (Figure 1). SVI maximum was 66ml/m². Chest exam was diffusely abnormal with PaO₂/FiO₂ ratio < 200. Furosemide was then used according to the protocol (Figure 2). The patient was discharged to a step down unit with no end organ failure on post-op day two.
DISCUSSION
Physiologic goal-directed resuscitation using a simplified treatment algorithm to guide both aggressive volume replacement and ultimately diuretic therapy resulted in the successful treatment and discharge of this severely injured young man with shock and acute lung injury.

Submitted by:
William T. McGee MD, MHA

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Tufts University Medical School
INTRODUCTION
Minimally invasive monitoring is helpful in the guidance of rapid resuscitation of trauma patients. Usual indicators of preload such as CVP may be inconclusive or misleading indicator of volume needs. The use of stroke volume index (SVI) and stroke volume variation (SVV) are helpful in determining the appropriateness and timing of volume resuscitation.

CASE NOTES
Medications: Lipitor 10 mg QD, Atenolol 50 mg QD, Amitriphyline 25 mg HS, Lanoxin .25 mg QD, Coumadin 3 mg QD, Glucosimine daily.

This elderly woman’s car went off the road at high speed for unknown reasons and became airborne. Presented to emergency department awake and alert, c/o nausea, pain with deep inspiration, complaining of left sided pain as well as abdominal pain, and hypotension refractory to volume resuscitation. Diagnosed with ruptured spleen and prepared patient for surgery. Patient is of significant surgical risk for death secondary to her current injuries and history of anticoagulation. ASA class 3. Patient typed and crossed for 4 units of packed red blood cells (PRBC) along with fresh frozen plasma (FFP).

Clinical Events
Patient details: 74-year-old female, 5’2”, 70 kg
Medical history: Chronic atrial fibrillation treated with anticoagulant, hypertension under control with treatment, and osteoarthritis

Patient underwent general anesthesia with rapid sequence induction for an emergent splenectomy. Intubated with a number 7 ETT, right radial arterial line started for frequent laboratory samples, continuous arterial pressure monitoring and continuous cardiac output monitoring using the Edwards FloTrac sensor and Edwards Vigileo monitor. Output: Blood loss estimated at 1500 cc, urine output 2000. Input: 4 units PRBC, 4 units of FFP, 450 cc Autotransfusion blood, NaCl 4 liters, and Hespan 1 liter. Patient underwent successful procedure, extubated and sent to recovery stable and awake.
DISCUSSION

Use of minimally invasive monitoring allowed for continued and aggressive volume resuscitation guided by Stroke Volume Index and Stroke Volume Variation when CVP values indicated normal filling pressures. Use of SVV and SVI gave confidence in direction and magnitude of resuscitation which resulted in a successful outcome.

Submitted by:
Michael Burns CRNA
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Sudden Blood Loss During Exploratory Laparotomy Case Study

INTRODUCTION
New minimally invasive monitoring tools can be extremely valuable when guiding fluid resuscitation in cases with anticipated or sudden unexpected blood loss. Arterial pressure-based cardiac output (APCO) monitoring and central venous oximetry enable clinicians to manage patients’ oxygen delivery more precisely than traditional vital signs alone. Stroke Volume Variation (SVV), a parameter available with APCO monitoring, has been shown to be a sensitive indicator of a patient’s preload responsiveness.

CASE NOTES
The patient experienced a sudden 2-liter blood loss due to intraoperative complications during the removal of a large peri-vaginal mass. Minimally invasive monitoring was initiated with a left radial arterial line and arterial pressure-based cardiac output (APCO) sensor (Edwards FloTrac sensor, Edwards Lifesciences, Irvine, CA, USA) along with a right internal central venous oximetry catheter (Edwards PreSep catheter, Edwards Lifesciences, Irvine, CA, USA) and monitored using a dedicated monitor (Edwards Vigileo monitor, Edwards Lifesciences, Irvine, CA, USA). Stroke Volume Variation (SVV) was used to monitor the patient’s preload responsiveness and guide blood and saline resuscitation. Normal saline and blood were infused rapidly through the PreSep catheter to achieve SVV values of less than 13% during duration of the patient’s surgery. Total volume replacement included 6 units of packed red blood cells and 4 liters of normal saline.

Clinical Events

<table>
<thead>
<tr>
<th>Clinical Events</th>
<th>Patient Details: 72-year-old female, 5'2&quot;, 72.7 kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical History:</td>
<td>Previous medical history revealed hypertension</td>
</tr>
</tbody>
</table>

For additional case studies visit [www.Edwards.com/FloTrac](http://www.Edwards.com/FloTrac)
DISCUSSION
The application of this less-invasive, easy-to-use hemodynamic monitoring device allowed the clinician to appropriately manage fluid resuscitation in the face of large and ongoing blood loss. SVV was used to establish the patient’s preload responsiveness and guide fluid resuscitation throughout the surgical procedure preventing over or under resuscitation. One aberrant SVV value of 25% was noted at 2:32 secondary to an arrhythmia. Cardiac output was optimized as the primary component of DO$_2$ and the adequacy of delivery was confirmed with ScvO$_2$.

Submitted by:
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SVV Guided Fluid Therapy in a Case of Heart Failure with Hypotension
Case Study

INTRODUCTION
Fluid resuscitation in severely critical patients can be challenging, particularly when the patient is in cardiogenic shock with pulmonary edema and hypoxia needing mechanical ventilation. Traditional hemodynamic monitoring parameters obtained via a pulmonary artery catheter do not address the issue of fluid responsiveness in patients on mechanical ventilation. In this case study, we describe the use of stroke volume variation (SVV) monitoring, which guided our decision to add fluids in order to improve cardiac output (CO). SVV guided fluid optimization led to a successful outcome in this seriously ill patient.

CASE NOTES
The patient was admitted with chief complaints of progressive dyspnea even at rest (NYHA Class IV heart failure) and swelling of feet. He was in sinus rhythm, his heart rate was 90 beats/minute, blood pressure was 108/60 mmHg, (mean arterial pressure 78 mmHg), respiratory rate was 22/minute. He was treated with intravenous diuretics, nitroglycerin, dobutamine and mask oxygen. The patient was evaluated and referred for cardiac resynchronization therapy (CRT).

Investigations before CRT:
- Hemogram: Hb – 11.9 gm%, total leucocyte count (TLC) – 11,000/cmm, platelets – 252,000/cmm
- Blood urea levels (BUL) – 24 mg%, serum creatinine – 1.1 mg%, serum sodium – 132 mEq/L, serum potassium – 3.5 mEq/L, serum chloride – 97 mEq/L
- Blood sugar levels were well controlled on insulin
- ECG showed LBBB pattern with QRS duration of 146 msec
- Chest x-ray showed cardiomegaly with bilateral lower zone haziness suggestive of pulmonary edema

Clinical Events
- Patient details: 32-year-old male
- Medical history: Diagnoses: type 2 diabetes; idiopathic dilated cardiomyopathy with left ventricular ejection fraction (LVEF) of 20%. Receiving optimal anti-heart failure therapy of torsemide, ramipril, carvedilol, digoxin, plus insulin

- Pre-procedure echocardiographic evaluation showed dilated cardiomyopathy with global hypokinesia, LVEF 20%, Grade I mitral regurgitation with mild pulmonary hypertension with pulmonary arterial systolic pressure (PH) of 38 mm

CRT procedure:
The patient underwent CRT in the form of bi-ventricular pacing under general anesthesia. Drugs used during the procedure were fentanyl 100 mcg, midazolam 4 mg, and ketamine 50 mg for induction and propofol 1-1.2 mg/min for maintenance. Total procedure time was 5 hours. Patient required dopamine 5-8 mcg/kg/min throughout the procedure to maintain blood pressure. Central venous pressure (CVP) and intra-arterial blood pressure monitoring was performed throughout the procedure.

Advanced hemodynamic monitoring (such as PA catheter, arterial pulse based cardiac output, transpulmonary thermodilution methods or transesophageal echo-Doppler) was not used during the procedure.
Ventilatory requirements during the procedure were high due to cardiogenic pulmonary edema. Patient received 60 mg of furosemide during the procedure. Patient was ventilated with pressure-controlled ventilation, peak inspiratory pressure of 35 cm, PEEP of 10 cm, and FiO\textsubscript{2} of 1. Intra-operative ABG values were: pH – 7.19, PO\textsubscript{2} – 62 mm, PCO\textsubscript{2} – 74 mm, HCO\textsubscript{3} – 28.3 mmol/L.

Intra-operative blood loss was 300 ml and urine output was 1200 ml. The patient received 500 ml of Ringer’s lactate during the procedure.

**Post-CRT:**
After the CRT procedure, the patient was moved to the Intensive Care Unit (ICU). Hemodynamic and ventilatory issues identified in the ICU were:

- HR 130/min, sinus rhythm, BP was 84/50 mmHg on dopamine 14 mcg/kg/min, MAP was 61 mmHg
- Severe hypoxia. Volume-controlled ventilation, tidal volume - 500 ml, respiratory rate – 18/min, needing PEEP of 12 cm with FiO\textsubscript{2} of 0.7, peak inspiratory pressure reaching 41 cm with plateau pressure of 32 cm
- CVP was persistently in the 18 to 20 mmHg range
- 1000 ml negative fluid balance while in operating room

**At this point, our therapeutic dilemmas were:**
1. If we give fluids to correct negative fluid balance, there was risk of aggravation of pulmonary edema, as the patient was already extremely hypoxic. Secondly what would be our goal or target for fluid therapy?
2. If we escalate dopamine to achieve higher perfusion pressure, there is risk of worsening of tachycardia and further deterioration of cardiac function, plus the risk of high-dose dopamine in terms of renal hypoperfusion and subsequent renal impairment.
3. To achieve afterload reduction and improvement in cardiogenic pulmonary edema, nitroglycerin or nitroprusside could not be given, as the BP was very low (same concern about dobutamine).
4. At such low MAP, would diuresis have an effect?

We decided to monitor cardiac output, cardiac index, systemic vascular resistance index (SVRI) and stroke volume variation (SVV) for better hemodynamic monitoring to get answers to these questions:

1. Is cardiac output adequate?
2. What is peripheral vascular resistance?
3. Is this shock fluid-responsive?

A radial arterial line was connected to the FloTrac system and hemodynamic variables displayed on the Vigileo monitor (see table). There were no arrhythmias. The patient was under deep sedation and paralysis on controlled mechanical ventilation. We obtained repeated SVV values by making Vt 8 ml/kg. SVV was between 20% and 22%.

<table>
<thead>
<tr>
<th>Hemodynamic Monitoring Values:</th>
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<tbody>
<tr>
<td>CO</td>
<td>4.7 l/min</td>
</tr>
<tr>
<td>CI</td>
<td>2.6 l/min/m²</td>
</tr>
<tr>
<td>SVV</td>
<td>20%</td>
</tr>
<tr>
<td>SVRI</td>
<td>1261</td>
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</table>

We felt that this information offered significant and fairly reliable evidence of preload dependency of this hemodynamic insufficiency. Therefore, we administered the first fluid bolus of 250 ml of 0.9% isotonic NaCl in 30 minutes, which resulted in marginal improvement in CO to 5 l/min, and MAP increased to 66 mm. Fluid boluses were continued until SVV dropped to < 15% on controlled ventilation. A total of 2450 ml fluids were given in the 14 hours post-operatively. MAP improved with fluid boluses and dopamine was rapidly tapered to 5 mcg/kg/min at the end of 6 hours in the ICU. HR settled to 100/min. Dobutamine was started at 5 mcg/kg/min to improve cardiac output and nitroglycerin was administered. Once hemodynamic stability was achieved, 40 mg of furosemide was given. The patient’s ventilatory requirements reduced rapidly and he was extubated 24 hours after ICU admission. Dobutamine was continued for the next 24 hours and tapered gradually. Ramipril and digoxin were restarted. BSLs were controlled with insulin infusion.

Five days post-procedure, cardiac evaluation via 2D echocardiography and Doppler showed improved LV function to 25%, and PH was marginally reduced. The patient was discharged from the hospital on day 7.

**DISCUSSION**

We feel that CO, CI and SVV monitoring helped us immensely in improving this patient's critical hemodynamic condition. If we had monitored only MAP, CVP or PCWP in this kind of acute cardiogenic pulmonary edema plus cardiogenic shock, fluid resuscitation would not have been attempted, and the outcome would likely have been far less successful. Instead, with the indication that the patient would respond positively, we could proceed confidently with fluid replacement. Further, the SVV parameter guided us regarding when to stop administering fluids.

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