# Intraoperative Monitoring, Resuscitation, and Transfusions
# An Interactive Case-Based Pro/Con Debate

## Learning Objectives for the Panel

1) Define goal directed fluid therapy and end-points for fluid resuscitation
2) Understand the arguments for and against the use of non-invasive cardiac output monitors to guide intraoperative fluid resuscitation
3) Discuss the use of vasopressors vs additional fluid resuscitation for the treatment of intraoperative hypotension
4) Examine the evidence for the rationale use of blood transfusions
5) Debate the real world justification for and against intraoperative blood transfusion
6) Outline and discuss the arguments for and against the use of Factor VII, prothrombin complex concentrates (PCCs) and anti-fibrinolytic therapy

## Discussion Outline

**A 65 year-old male with liver metastases is scheduled for an elective hepatectomy. The surgery is scheduled for 4 hours and the surgeon reports a planned EBL of approximately 500ml.**

**Should we utilize a non-invasive cardiac output monitor for the case?**

Yes:

Please review

Rational fluid management in today's ICU practice.
Bartels K, Thiele RH, Gan TJ.
Crit Care. 2013;17 Suppl 1:S6. doi: 10.1186/cc11504. Epub 2013 Mar 12. Review.
PMID: 23514431
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No:

In the last few decades, an important paradigm shift has occurred in intensive and acute care medicine, including the operating room. During this time, a growing body of work has implicated over-resuscitation, particularly with crystalloid solutions, as a risk factor in many post-op complications, including several varieties of organ dysfunction. Although the specific endpoints of resuscitation are still a subject of intense debate, it does seem clear that both over- and under-resuscitation should be avoided. It has also been demonstrated quite convincingly that one of the old standbys for monitoring fluid status, the central venous pressure, has very little correlation with cardiac output, or more importantly, volume responsiveness. Some authors have rediscovered and re-validated older clinical exam techniques, such as the passive straight leg raise—but these are impractical in the operating room. The reigning gold-standard cardiac output monitor, the pulmonary artery catheter (PAC), has fallen out of favor in clinical settings after several studies found that they had an unacceptable complication rate,

and that even when placed successfully, they were often misinterpreted at the bedside and did not improve outcomes. Several relatively new monitoring devices have recently come into clinical use (having been heavily marketed) based on the promise of close correlation with the PAC, and simplified, real time assessment of both cardiac output and volume responsiveness—thereby helping the clinician to keep a challenging patient in the sweet-spot between over and under-resuscitation. Unfortunately, currently available minimally-invasive and non-invasive cardiac output monitors, while promising and extremely appealing, are plagued by important limitations that make their interpretation more challenging and less trustworthy than they might appear. In this section, several recent reviews and clinical studies will be analyzed, highlighting specific weaknesses of these technologies and critically appraising the data supporting their use. In some cases, the devices are more invasive than might be considered acceptable, some require expensive and non-standard catheters. Several require intermittent calibration. A key limitation of many of these techniques is that they lose validity in sicker patients—for example those with vasodilatory shock, poor distal perfusion, arrhythmias, poor lung compliance, or right heart dysfunction. Finally, although some small studies have suggested improved outcomes using protocolized care based on these monitors, others have found no difference, and in fact, that routine use of these monitors leads to increased administration of IV fluids. In any case, large, well-designed clinical trials are needed before we can confidently say that these monitors are truly ready for prime-time.

**2 hours into the case, EBL is 500ml and the patient is hypotensive despite 3 Liters of volume resuscitation.**

**Should we treat the hypotension with continued fluid boluses?**

Yes:

Please review

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**No:**

Having just argued against the over-emphasis on non-invasive cardiac output monitoring, in this section, I will take the slightly contradictory position of warning against the dangers of empiric fluid over-resuscitation and in favor of judicious use of vasopressors. It could be argued that the answer to the question of fluid or pressor is a simple one once we “know the numbers.” In this line of thinking, we should use fluid resuscitation for hypovolemia, vasopressors for vasodilation, and inotropes for a so-called “pump problem” and the right answer lies in figuring out which of these problems is paramount in the patient in front of us. The challenge, unfortunately, is that we often have to make decisions in the absence of full information. Even when we have access to every number, pressure, and index in routine clinical use, the complex interactions between volume, perfusion, and vascular tone may preclude a

simple answer. This section will review the available data on the limitations of the "fluid first" mode of responding to hypotension, oliguria, and tachycardia. We will then turn our attention toward outlining some practical guidelines regarding when to start vasoactive infusions. Finally, we will conclude with a review of recent literature comparing the different vasoactive medications and highlighting current best-evidence regarding indication, drug choice, dose, and duration of pressor therapy.

**4 hours into the case, the EBL is now 1000mL and the patient is hypotension. ABG reveals a HGB of 8.**

**Should we transfuse the patient with PRBCs?**

Yes:

Data from clinical trials in the 1990's suggests that blood transfusions do not improve outcomes of critically ill patients, and that the risks of transfusions may outweigh the benefits. However, recent data suggests that with leukocyte reduction that transfusion in critically ill patients actually improves outcomes. In the acute management of trauma patients in the postoperative management of cardiac surgery patients transfusing blood to hemoglobin goals improves outcomes. I will review recent clinical trials that suggest that anemia is a poor prognostic sign and that transfusion is not only but also improves patient outcomes.

No:

Since the 1980's there has been a paradigm shift towards an era of blood conservation strategy. This has evolved from a number of different revelations related to anemia and transfusion. Large retrospective studies, primarily in Jehovah's Witness patients and other patients that have refused blood transfusion have shown that the majority of patients can tolerate low levels of anemia. This is most likely due to a number of natural compensatory mechanisms such as increased HR, increased SV, peripheral and coronary vasodilation, increased extraction ratio, shift in the oxygen-hemoglobin dissociation curve, and shunting of blood to essential organs. There are inherent risks associated with transfusion that include but are not limited to infection, circulatory overload (TACO), acute lung injury (TRALI), and immune modulation, with little benefit coming from the transfusion of packed red blood cells. Other studies such as the TRICC trial have demonstrated that the majority of patients can tolerate hemoglobin levels in the 7-10 range without untoward side effects. More recent reviews, such as the ASA's "Practice Guidelines for Perioperative Blood Management" do not provide convincing evidence that transfusion is warranted in these circumstances.

**5 hours into the case, EBL is 4 liters. The patient was transfused 6U PRBCs, 6U FFP, and 1U PLTs. The surgeon tells you that the patient is oozing.**

**Should we transfuse Factor 7 or a Prothrombin Complex Concentrate or an Antifibrinolytic?**

Yes:

The administration of recombinant Factor VII has only been shown to be an effective strategy in a few isolated circumstances, such as trauma, refractory GI bleeding, and possibly in the coagulopathy of liver

disease. The over resuscitation of bleeding patients with blood products can lead to end organ damage, immune modulation, volume overload, and end organ dysfunction. Activated factors improve limit transfusion and in selected patients should be considered as part of a targeted resuscitation strategy.

No:

The administration of recombinant Factor VII has only been shown to be an effective strategy in a few isolated circumstances, such as trauma, refractory GI bleeding, and possibly in the coagulopathy of liver disease. However, studies do not support the routine use of Factor VII in “routine surgical oozing.” Rather, replacement of coagulation factors and transfusion of plasma and cryoprecipiate or platelets should be guided by lab data supporting coagulopathy or viscoelastic studies showing abnormalities in the clotting system. Recent studies looking at the off-label use of recombinant Factor VII have not shown a mortality difference. Other studies looking at anti-fibrinolytic agents such as aprotinin, TXA, or epsilon aminocaproic acid have not shown a difference in the amount of RBC’s transfused or mortality beyond a small subset of patients, for example, orthopedic total joint surgery patients. Other studies have noted a significant increase in the prothrombotic complications associated with these drugs.