

Questions with Answers

Non Invasive hemodynamic Monitoring and Shock

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1. 55 yo female sp dilation and curettage (D&C) for uterine bleeding a week ago comes to the emergency room with mental status changes, and abdominal pain. Hr 150 in a-fib, BP 60 palp. Crystalloid infusion is started; the ER physician gets a central line. CVP is 6.

Echocardiogram in the ED shows a flat IVC, hyperdynamic heart, minimal pericardial effusion. **The most likely reason for the hypotension is:**

- a. **Hypovolemia**
- b. Left ventricular dysfunction
- c. Tamponade
- d. Pulmonary embolism

Reasoning:

Inferior vena cava (IVC) diameter and collapsibility has been shown to be a reliable method of monitoring intravascular volume in mechanically ventilated patients, patients in septic shock, and also in patients not ventilated receiving dialysis. In addition to this visualization of an empty hyperdynamic heart is an indicator of hypovolemia.

References:

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2. She goes emergently to the operating room, and a bowel perforation is found, with gross abdominal contamination. She receives 5 L of crystalloid. Her urine output during the case is minimal.

Intra operatively she had an a-line placed with a FloTrac Vigileo showing a CCO of 5 and an SVV of 33. CVP now is 15.

She arrives to the ICU intubated, and receives 1 PRBC in transit from the operating room. She has a new episode of hypotension, and her hemodynamic parameters are as follows. CCO 2.5 SVV 7 CVP 22. Echo shows a full IVC mild dilation of the right atria, sluggish global heart contraction, no pericardial effusion.

Your diagnosis is most likely:

- a. Persistent Hypovolemia
- b. Pulmonary Embolism
- c. Impossible to tell needs a pulmonary artery catheter to diagnose
- d. Sepsis induced cardiac dysfunction**

3. Your treatment at this point should include:

- a. Intravenous fluids
- b. Blood
- c. Thrombolytic Therapy
- d. Inotropes**

Reasoning:

Septic shock has been considered a “hyperdynamic” state in which the cardiac output (CO) is either normal or increased. However, several have suggested there is a decreased global contractility in septic patients.

Parker et al were the first to describe LV hypokinesis in septic shock, in which patients with severely depressed LV EF (LVEF) in whom an adequate LV stroke output could be maintained through acute LV dilatation. Jardin et al studied 90 patients with septic shock with daily bedside assessment of LV volume and LVEF using TTE, and observed that LVEF was significantly depressed in all patients, resulting in severe reduction in left ventricular stroke volume.

In the septic patient, bedside echocardiography is valuable for identification of the cause of hemodynamic instability (which may be of hypovolemic, cardiogenic, or distributive origin) and for the subsequent optimization of therapy (ie, fluid administration, inotropic or pressor infusion, or a combination of the above).

References:

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2. Parrillo, JE Pathogenetic mechanisms of septic shock. *N Engl J Med* 1993;328,1471-1477

3. Jardin, F, Fourme, T, Page, B, et al Persistent preload defect in severe sepsis despite fluid loading: a longitudinal echocardiographic study in patients with septic shock. Chest 1999;116,1354-1359
4. Vieillard-Baron, A, Prin, S, Chergui, K, et al Hemodynamic instability in sepsis: bedside assessment by Doppler echocardiography. Am J Respir Crit Care Med 2003;168,1270-1276

4. She recovers nicely requiring multiple trips to the operating room for abdominal washout to treat her intra-abdominal sepsis. DVT prophylaxis was held in multiple occasions because of this issue. She is now extubated, with an ostomy, her abdomen is closed, and her renal function is now normal. As she is getting ready to be moved to a regular floor she developed acute tachypnea, tachycardia followed by hypotension.

ABG is pH 7.436, PaCO₂ 34, PaO₂ 90, SpO₂ 96% on 3 liters O₂. EKG shows an acute Right bundle branch block, with T wave inversions in leads III, V1-4. Echocardiogram shows, dilated IVC, enlarged right ventricle, small left ventricle, and overall diminished global cardiac contractility, with minimal pericardial effusion.

The reason of the hypotension this time is most likely:

- a. Persistent Hypovolemia
- b. Pulmonary Embolism**
- c. Impossible to tell needs a pulmonary artery catheter to diagnose
- d. Acute myocardial infraction

5. The first step in the treatment of this issue should be

- a. Intravenous fluids
- b. Inotrope
- c. Intravenous anticoagulation**
- d. Coronary Catheterization

Reasoning:

Echocardiography has been shown to have a good positive predictive value for massive PE, since it can detect acute RV dilatation and dysfunction following large PE. The finding of RV dilatation and dysfunction is, however, not specific for PE, as it may be observed with a variety of other conditions. Regional RV dysfunction has been found to have a sensitivity of 77% and a specificity of 94% for the diagnosis of acute PE, with a positive predictive value of 71% and a negative predictive value of 96%. The presence of regional RV dysfunction in which the apex is spared should thus raise the level of clinical suspicion for the diagnosis of acute PE vs. the other possible causes of RV dysfunction.

References:

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2. Jardin, F, Dubourg, O, Gueret, P, et al Quantitative two-dimensional echocardiography in massive pulmonary embolism: emphasis on ventricular interdependence and leftward septal displacement. *J Am Coll Cardiol* 1987;10,1201-1206
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