Hemodynamic Monitoring: Review Questions

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QUESTIONS

Choose the single best answer for each question.

1. A 44-year-old man with hypertension and diabetes is admitted to the intensive care unit (ICU) for severe pancreatitis. Soon thereafter, he develops respiratory distress requiring intubation. His blood pressure is 80/30 mm Hg, heart rate is 120 bpm, and respiratory rate is 24 breaths/min. His serum creatinine level is 2.5 mg/dL (baseline, 1.0 mg/dL), and his arterial lactate level is 3.8 mg/dL. An electrocardiogram shows sinus tachycardia with occasional ventricular premature beats, and chest radiograph shows bilateral fluffy infiltrates. The ventilator is set on assist-control mode; tidal volume, 500 mL; respiratory rate, 12 breaths/min, FiO2, 100%; and positive end-expiratory pressure, 10 cm H2O. Pulse oximetry shows SaO2 of 90%. Data from a pulmonary artery catheter (PAC) show central venous pressure (CVP), 24 mm Hg; pulmonary artery pressure, 35/20 mm Hg; pulmonary artery wedge pressure (PAWP), 12 mm Hg; and cardiac output (CO), 4.5 L/min. At this point, what is the best management option for this patient?

(A) Intravenous (IV) furosemide 40 mg
(B) IV β-blocker
(C) IV amiodarone
(D) IV fluid bolus
(E) IV dobutamine

2. A 58-year-old man with liver failure from hepatitis C cirrhosis is admitted to the surgical ICU after liver transplantation. The patient is intubated and has a perioperatively placed PAC. He is 5 ft 8 in, weighs 78 kg, and his vital signs are: blood pressure, 98/50 mm Hg; mean arterial pressure, 66 mm Hg; heart rate, 108 bpm; and temperature, 36°C. Results of laboratory tests show: leukocyte count, 12.0 × 10³/mm³; hemoglobin level, 9.0 g/dL; international normalized ratio, 2.3; arterial lactate level, 5 mg/dL (increased from 3.4 mg/dL immediately postoperatively); sodium, 134 mEq/L; potassium, 4.5 mEq/L; bicarbonate, 23 mEq/L; blood urea nitrogen, 20 mg/dL; creatinine, 1.4 mg/dL; and glucose, 123 mg/dL. Results of an arterial blood gas sampling show: pH, 7.36; PaCO2, 38 mm Hg; Po2, 120 mm Hg; and SaO2, 95%. Results of mixed venous blood gases show: pH, 7.32; PacO2, 50 mm Hg; Po2, 43 mm Hg; and SvO2, 72%. PAC data are: CVP, 8 mm Hg; PAWP, 12 mm Hg; CO, 4.0 L/min; and systemic vascular resistance, dynes/sec/cm–5. Which of the following statements regarding this patient’s CO is correct?

(A) CO is adequate
(B) CO is not adequate
(C) CO is too high
(D) CO is falsely low
(E) CO cannot be accurately determined in transplant patients

3. To clarify the utility of the PAC to improve patient outcome, several major studies have been conducted. These studies have demonstrated which of the following?

(A) Perioperative PAC use benefits high-risk surgical patients
(B) PAC use does not benefit patients with acute respiratory distress syndrome (ARDS)
(C) PAC use harms patients with septic shock
(D) PAC use should not be allowed in the ICU setting
(E) PAC use benefits patients with septic shock

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ANSWERS AND EXPLANATIONS

1. (D) IV fluid bolus. The patient has severe pancreatitis with shock and is developing ARDS and multi-organ failure. Fluid resuscitation is needed at this time. Diuretics (eg, furosemide) would exacerbate hypoperfusion and shock by decreasing intravascular volume. Although the CVP is elevated, numerous studies have shown that CVP is an inaccurate measurement of intravascular volume and fluid responsiveness. Neither CVP nor PAWP provide accurate information regarding the left ventricle’s filling and fluid responsiveness. A better measure of ventricular volume (ie, preload) is bedside echocardiography. For this patient, an empty left ventricle with decreased left ventricular end-diastolic area and end-systolic obliteration with normal or increased contractility would indicate the need for further fluid resuscitation. β-Blockers would decrease heart rate and blood pressure and worsen CO, which is dependent on tachycardia due to the patient’s low stroke volume (37.5 mL/beat). Occasional ventricular ectopy does not require pharmacologic management, and amiodarone may worsen hypotension. If after receiving adequate IV fluid resuscitation the patient remains hypotensive and in shock, beginning an inopressor would be indicated. However, in the setting of tachycardia and hypotension from pancreatitis or sepsis, dobutamine often worsens hypotension and tachycardia and may precipitate serious cardiac arrhythmias or cardiovascular collapse.†1–3

2. (B) CO is not adequate. The normal CO range is 4 to 6 L/min, and the normal cardiac index is 2 to 4 L/min/m². Determining whether a patient’s CO is adequate depends on the patient’s clinical condition and laboratory assessments, such as lactate, mixed venous oxygen saturation (MVO₂), and the mixed venous-arterial PCO₂ gradient. In patients with hyperdynamic states (eg, sepsis, liver failure), CO is often elevated above the normal range (7–10 L/min); therefore, the CO of 4.0 L/min obtained in the case patient is relatively low. The elevated and rising lactate level (normal, < 2.0 mg/dL) suggests that either the oxygen delivery to the tissues is inadequate to meet oxygen demand and/or that lactate clearance by the newly transplanted liver is decreased. It is commonly assumed that low MVO₂ levels indicate inadequate oxygen delivery to the tissues; however, high or even normal MVO₂ levels may be found in the presence of inadequate oxygen delivery. In this patient, the MVO₂ level and SvO₂ are in normal range, but the patient is anemic with a relatively low CO. Because of the patient’s cirrhosis and liver failure, the MVO₂ level is raised to normal due to arteriovenous shunting, and thus MVO₂ should not be used to evaluate CO. Mixed venous-arterial PCO₂ gradient can be used to evaluate CO as well. In this case, the venous-arterial PCO₂ gradient is significantly elevated at 12 mm Hg (normal, < 5–6 mm Hg), suggesting that CO is relatively low. The venous-arterial PCO₂ may be elevated in patients with high CO (hyperdynamic) if they are also hypermetabolic (eg, high fever, sepsis, malignant hyperthermia). This patient was afebrile and not hypermetabolic.†2,4

3. (B) PAC use does not benefit patients with ARDS. A recent multicenter randomized trial examined the use of PAC in patients with septic shock, ARDS, or both and found that PAC use did not affect mortality or morbidity in either condition.5 Another large, multicenter, randomized trial examined perioperative PAC use in high-risk surgical patients undergoing major surgery and found no evidence of benefit from PAC-guided treatment compared with standard care.5 Potential explanations for this lack of benefit include inaccurate and variable interpretation of PAC data, variable endpoints and treatments for hemodynamic instability, and reliance on CVP and PAWP to guide or dictate treatment. Furthermore, the PAC is a monitoring technology as opposed to a therapeutic technology; for a monitoring technology to change outcome, the disease state it is used to monitor must have an effective treatment. Critically ill patients with multi-organ failure, ARDS, or shock do not yet have treatments as effective as those for cardiac conditions in which another monitoring technology, the electrocardiogram, clearly improves outcomes (eg, early detection of ischemia treated with angioplasty).5,6

REFERENCES


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