Instructions: For each question, select the most correct answer.

1. A 65-year-old woman admitted for aneurysmal subarachnoid hemorrhage has dyspnea and substernal chest pain two days after admission. ECG shows anterior lead ST elevations. Troponin is mildly elevated. However, there is no angiographic evidence of obstructive coronary disease or acute plaque rupture. She then develops hypotension (blood pressure 80/60 mm Hg) and is found to have a moderate left ventricular outflow tract obstruction.

Which of the following treatments is contraindicated for this patient?
A. Alpha-agonists
B. Beta-blockers
C. Inotropic agents
D. Intra-aortic balloon pump

2. A 53-year-old man with a history of IV drug abuse is admitted to the ICU after a Whipple procedure. He regularly takes methadone. Acutely, he takes hydromorphone for acute analgesia and antibiotic prophylaxis with cefazolin. The QTc from the morning 12-lead ECG is 510 ms. Later that morning, the rhythm shown below is observed.
Which of the following is the most appropriate next intervention?
A. Discontinue methadone.
B. Continue all current drugs and monitor ECG twice daily.
C. Start amiodarone.
D. Administer bicarbonate.

3. A 65-year-old woman with a history of diabetes, hypertension, depression, and anxiety is evaluated in the emergency department for severe substernal chest pain with nausea and vomiting. She recently lost her husband to cancer and just found out that her son was killed in a car accident this morning. On arrival, vital signs are: temperature 37°C (98.6°F), heart rate 120 beats/min, blood pressure 90/60 mm Hg, respiratory rate 24 breaths/min, oxygen saturation 92% on 6 liters/min nasal cannula. Physical examination is notable for crackles in the lung bases and cool extremities. ECG shows ST elevation in leads V1-V3 with T wave inversions in leads I and aVL. Chest radiograph shows mild pulmonary edema.

She is started on aspirin, clopidogrel, and a heparin infusion, while being given a 1-liter IV normal saline bolus as she is taken up to the catheterization laboratory for a percutaneous coronary intervention. She is noted to have small amounts of plaque with less than 30% stenosis in her left anterior descending and right coronary arteries. Left ventriculography reveals apical ballooning with vigorous contraction of the basal segment. Echocardiography shows evidence of left ventricular outflow tract obstruction (see below). During the procedure, her oxygen saturations drop to 70% despite a nonrebreather mask, and her blood pressure is now 70/40 mm Hg, with a heart rate of 130 beats/min, and she is confused.
Which of the following is the most appropriate hemodynamic support strategy for this patient?
A. Intubate and start dobutamine and norepinephrine.
B. Intubate and start IV esmolol and phenylephrine.
C. Intubate and give a 1-liter IV normal saline bolus.
D. Begin bilevel positive airway pressure, and start dobutamine and norepinephrine.

A 54-year-old man presents with advanced heart failure. He had a left ventricular assist device placed two days ago as a bridge to transplantation. His intraoperative course was uneventful and he was extubated 24 hours ago. His pump speed has remained at 9,600 revolutions/min. Power has remained at 5.7 watts. His last documented flow was 4.8 L/min. His heart has remained in normal sinus rhythm. The on-duty physician is called to the bedside by the nurse, who has now noted an acute decrease in cardiac output (pump flow). There have been no complications with the left ventricular assist device machinery or connections.

Which of the following findings would be consistent with the emergent situation of cardiac tamponade?

<table>
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<tr>
<th>CVP</th>
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Abbreviations: CVP = central venous pressure, PAP = pulmonary artery pressure, PAOP = pulmonary artery occlusion pressure, MAP = mean arterial pressure, LV = left ventricle, RV = right ventricle, LA = left atrium, RA = right atrium

Two hours after a right carotid endarterectomy, a 75-year-old man is increasingly somnolent and has a right-sided headache when aroused. His blood pressure is 180/100 mm Hg, heart rate 70 beats/min, and respiratory rate 18 breaths/min.

Which of the following is the most appropriate intervention?
A. Administer labetolol to control hypertension.
B. Obtain a head CT to rule out intracranial bleeding.
C. Administer a narcotic for analgesia.
D. Obtain transcranial Doppler study to assess possible vasospasm.
6. A 55-year-old man with hypertension has chest pain and is brought to the emergency department by his wife, where he has a cardiac arrest, and cardiopulmonary resuscitation (CPR) is immediately initiated with compressions and masked ventilation. An endotracheal intubation is performed, and colorimetric end-tidal carbon dioxide (CO\textsubscript{2}) testing is used to aid in confirmation of endotracheal tube placement. There is mild color change on the end-tidal CO\textsubscript{2} detector. On auscultation of the chest, breath sounds can be heard in bilateral lung fields during manual ventilation with no rush of air heard over the stomach. Chest compressions are continued. He is ventilated via the endotracheal tube for a full round of CPR, and the color change does not go away.

Which of the following most likely accounts for the low-level color variation on the end-tidal CO\textsubscript{2} tester?
A. Severe acidosis
B. Venous hyperoxia post-cardiac arrest
C. Hypoventilation with the bag ventilation
D. Low cardiac output

7. A 30-year-old man is placed on peripheral venoarterial extracorporeal membrane oxygenation (ECMO) using femoral artery and vein catheters for worsening cardiogenic shock due to viral myocarditis. During the following week his native cardiac function appears to be improving, although he develops ventilator-associated pneumonia and is being treated with broad-spectrum antibiotics. Arterial blood gas (ABG) analysis drawn from the right radial arterial line is pH 7.48, partial arterial carbon dioxide pressure (PaCO\textsubscript{2}) 32 mm Hg, partial arterial oxygen pressure (PaO\textsubscript{2}) 60 mm Hg. Oxygen saturation is 90% on 60% fraction of inspired oxygen (F\textsubscript{io2}) and 7.5 cm \textsubscript{H}\textsubscript{2}O positive end-expiratory pressure, tidal volume 450 mL, respiratory rate 18 breaths/min. Chest radiograph shows a right middle lobe and left lower lobe infiltrate. Post-oxygenator ABG readings are pH 7.40, PaCO\textsubscript{2} 40, PaO\textsubscript{2} 300. The nurse alerts the attending physician to the patient’s declining oxygen saturations throughout the day and the low partial arterial oxygen pressure.

Which of the following is the most appropriate next step in management?
A. Change out the oxygenator.
B. Add a second oxygenator to the circuit.
C. Consider changing to central venoarterial ECMO or increasing circuit flow.
D. Increase the ventilator F\textsubscript{io2}.
E. Convert to venovenous ECMO.
8. A 74-year-old man is being treated in the ICU for severe community-acquired pneumonia. In the evening, he has been reporting severe, sharp, substernal chest pain with radiation of his symptoms. Vital signs are: blood pressure 132/68 mm Hg, heart rate 113 beats/min, respiratory rate 22 breaths/min, oxygen saturation 94% on 2 liters, and initial troponin T level 0.03 ng/dL. On physical examination, he is moderately distressed and sitting upright. The rest of the examination is significant for flat neck veins, bibasilar rales on lung examination, and distal, harsh, heart sounds. His ECG is shown below.

Which of the following is/are the most appropriate next step(s) in management?
A. Emergent percutaneous coronary intervention
B. Chest CT angiography and heparin infusion
C. Aggressive diuresis
D. Nonsteroidal anti-inflammatory therapy and reassurance
9. An 82-year-old man is transferred to the ICU two days after a total hip replacement with chest pain and new hypotension. He is afebrile, with a heart rate of 75 beats/min, blood pressure 88/67 mm Hg, respiratory rate 22 breaths/min, and oxygen saturation 98% on room air. He is clammy and confused, but is able to state that his chest pain is 9 out of 10. The ECG obtained on ICU admission is shown below.

Which of the following medications should be administered while awaiting arrival of the cardiology team?
A. Nitroglycerin to reduce his chest pain, with a goal of 0 out of 10
B. Aspirin, 162-325 mg, immediately
C. IV diltiazem to target a heart rate of 60 beats/min
D. IV morphine, 4 mg

10. Which of the following is true about perioperative 5-lead telemetry monitoring in noncardiac surgery patients?
A. Leads II and V5 have equivalent sensitivity for myocardial ischemia.
B. Leads II, V4, and V5, when used together, have superior sensitivity to detect myocardial ischemia.
C. Lead II has superior sensitivity for myocardial ischemia compared to lead V5.
D. Lead V5 has superior sensitivity for rate and rhythm disturbances compared to lead II.
E. Lead V4 has less sensitivity than lead V2 for myocardial ischemia.
11. Which of the following agents is most appropriate to administer should the patient with the ECG shown below develop atrial fibrillation with rapid ventricular rate?

A. Procainamide  
B. Metoprolol  
C. Verapamil  
D. Adenosine  
E. Digoxin

12. A 66-year-old man with aortic stenosis arrives in the ICU after repair of an abdominal aortic aneurysm. Shortly after admission, his ECG shows a change from normal sinus rhythm to atrial fibrillation at a ventricular rate of 84 beats/min. His blood pressure shows a slight change from 110/60 mm Hg to 100/52 mm Hg. Pulmonary artery occlusion pressure (PAOP) increased from 16 to 22 mm Hg with prominent a waves noted.

Which of the following is the most appropriate course of action?
A. Increase myocardial contractility with inotropic support.  
B. Maintain PAOP below 20 mm Hg with diuretics.  
C. Attempt conversion to sinus rhythm with amiodarone.  
D. Decrease peripheral vascular resistance with vasodilator therapy.
13. An 86-year-old woman with a history of hypertension, heart failure, and atrial fibrillation has had weakness, abdominal pain, blurry vision, and vomiting for two days. Her blood pressure is 92/55 mm Hg. She takes metoprolol, digoxin, aspirin, and lisinopril. Her ECG on ICU admission is shown below.

Which of the following is the most appropriate treatment at this time?
A. Calcium chloride
B. Digoxin immune Fab
C. Activated charcoal
D. Potassium repletion
E. Dialysis

14. A 70-year-old woman with a history of nephrolithiasis and myocardial infarction is admitted to the ICU with hypotension and urosepsis. She is treated with fluid resuscitation, antibiotics, and vasopressor support. While being admitted, she develops atrial fibrillation with rapid ventricular rate, which is controlled with a diltiazem infusion. An echocardiogram shows mild systolic impairment, normal left atrial size, and normal valves. Thyroid function tests are normal. Three days later, the urosepsis has resolved and she undergoes diuresis but remains in atrial fibrillation with ventricular rate controlled on an oral medication regimen. An oral anticoagulation is being considered, given the ongoing atrial fibrillation.

Which of the following is the best risk-benefit analysis of thrombosis and bleeding?
A. Thrombosis risk: high, bleeding risk: high
B. Thrombosis risk: high, bleeding risk: low
C. Thrombosis risk: low, bleeding risk: high
D. Thrombosis risk: low, bleeding risk: low
15. A 68-year-old man is undergoing a mitral valve replacement for severe mitral regurgitation. He is unable to be weaned from cardiopulmonary bypass. An intra-aortic balloon pump (IABP) is placed and he is able to separate from the heart-lung machine. The IABP pressure tracing in the ICU is shown below. Which of the following is the necessary correction?
A. No adjustment is necessary; the IABP is augmenting correctly.
B. Set the balloon pump to trigger later; the tracing suggests early balloon inflation.
C. Set the balloon pump to trigger earlier; the tracing suggests late balloon inflation.
D. Set the balloon pump to deflate earlier; the tracing suggests late balloon deflation.

16. A 76-year-old man develops right ventricular failure on postoperative day three after implantation of a left ventricular assist device. Furosemide, 120 mg IV every 6 hours, is administered and, initially, the patient’s response is vigorous, but after four days, his response is significantly diminished. Which of the following most likely explains the patient’s decreased response to the furosemide?
A. Atrophy of cells in the distal convoluted tubule
B. Increased protein binding of furosemide
C. Competitive inhibition of furosimide transport
D. Decreased stimulation of the renin-angiotensin-aldosterone system

17. A 67-year-old man is admitted to the ICU after implantation of a continuous flow left ventricular assist device. Three days later, his device monitor displays a gradual increase in pump power from 4.3 to 6.7 watts, and the pulsatility index has decreased from 6.2 to 4.3. The speed set on the device is unchanged. Repeated laboratory values demonstrate an increased serum lactate dehydrogenase, increased serum total bilirubin, and a mixed venous oxygen saturation that has decreased from 62% to 49%. Which of the following is the most likely explanation for these findings?
A. Popliteal vein thrombosis
B. Acute mitral regurgitation
C. Impeller thrombosis
D. Right ventricular failure
18. A 70-year-old woman with chronic ischemic heart failure and ejection fraction of 15% is in the ICU for acute exacerbation of heart failure. She has no valvular abnormalities and is being evaluated for possible revascularization. She has had previous cardiac catheterizations, which demonstrated multiple proximal left-sided lesions that were not amenable to percutaneous revascularization and stenting. Past medical history also includes diabetes mellitus, chronic obstructive pulmonary disease, obesity, and hypertension. Chest radiograph demonstrates bilateral infiltrates. She is intubated and placed on mechanical ventilation. She becomes hypotensive; pulmonary artery and radial artery catheters are inserted. The cardiac index (CI) is 1.1 L/min/m² and dobutamine is started without commensurate increase in CI. She becomes hypotensive, and her heart rate increases from 85 to 110 beats/min with ST changes.

Which of the following is the most appropriate next step in management?
A. Give 2 liters of IV fluids rapidly to increase mean arterial pressure (MAP).
B. Start esmolol infusion to decrease heart rate.
C. Insert intra-aortic balloon pump to unload left ventricle and increase coronary perfusion pressure.
D. Start phenylephrine infusion to increase MAP.

19. A 43-year-old man is admitted to the ICU with nausea, vomiting, and jaundice. He says that the symptoms have been progressing over the past two months. His medical history is significant for end-stage non-ischemic cardiomyopathy, and he has a HeartMate II left ventricular assist device (LVAD). Current vital signs are: blood pressure 121/83 mm Hg, heart rate 98 beats/min, respiratory rate 14 breaths/min. His hemoglobin is 8.3 g/dL; four months ago it was 12.1 g/dL. His current lactate dehydrogenase is 2,123 U/L. His LVAD setting is 9,000 RPM, pulsatility index 1.3, and pump power 8.4 watts with occasional increases to 13 watts. Transthoracic echocardiography shows a well-filled left ventricular cavity and a ventricular septum with a slight rightward bow. His INR is 2.8.

Which of the following is the best treatment for this patient?
A. Continue observation.
B. Increase the RPM speed.
C. Increase anticoagulation therapy with a goal of raising INR to 4.
D. Initiate pump exchange.

20. Despite the decreased use of pulmonary artery catheters (PAC) in the ICU because of controversy regarding their benefit, in which of the following clinical situations is a PAC most useful?
A. Acute respiratory distress syndrome
B. Septic shock
C. Severe pulmonary hypertension
D. Traumatic brain injury
21. A 72-year-old woman with a history of atrial fibrillation is intubated for respiratory distress secondary to septic shock. She receives a 30 mL/kg IV fluid bolus and her lactate decreases from 5.0 to 3.0 mmol/L in two hours. She has a right subclavian central line and a right radial arterial line. Her central venous pressure (CVP) is 12 mm Hg. Her vital signs, relevant laboratory results, and ventilator settings are as follows: sodium 135 mEq/L, potassium 3.7 mEq/L, chloride 100 mEq/L, carbon dioxide 18 mmol/L, blood urea nitrogen 22 mg/dL, creatinine 1.5 mg/dL. Ventilator tidal volume: 7 mL/kg (patient is dysynchronous with ventilator), respiratory rate 12 breaths/min (set), patient is breathing 15 breaths/min, positive end-expiratory pressure 5 cm H₂O, fraction of inspired oxygen 65%.

Which of the following statements is most correct regarding intravascular volume assessment in this patient?
A. Pulse pressure variation can accurately predict her fluid responsiveness.
B. An increase in CVP (from 12 to 15 mm Hg) will predict her fluid responsiveness.
C. A positive passive leg raise will predict her fluid responsiveness.
D. A low pulmonary artery occlusion pressure will best predict her fluid responsiveness.

22. In which of the following patients would use of a conventional pulse contour monitoring device measuring stroke volume variability and cardiac output provide the most reliable data?
A. Cirrhotic patient with severe sepsis who is tachypneic
B. Septic intubated patient with severe acute respiratory distress syndrome receiving lung protective strategy with tidal volume of 5 mL/kg
C. Patient under general anesthesia in sinus rhythm undergoing major intra-abdominal surgery with prolonged cavity exposure and expected to lose a moderate volume of blood
D. Patient status post large acute anterior myocardial infarction on pressors with an intra-aortic balloon pump
E. Patient with a large pulmonary embolus who is tachycardic with multifocal atrial tachycardia

23. A patient in septic shock with acute respiratory failure who is receiving mechanical ventilation in pressure control mode has a pulmonary artery catheter in place to assist in management of fluids and pressor therapy.

Convention regarding the recording of pulmonary arterial pressure is that measurements be taken at which of the following times?
A. At peak of inhalation and just before the QRS
B. At end of exhalation and during the QRS
C. At peak of inhalation and just after the QRS
D. At end of exhalation and just after the QRS
24. A 24-year-old man is involved in a motor vehicle collision. He is following commands. Head and cervical spine CT is negative, chest radiograph is normal, focused assessment with sonography in trauma (FAST) and extended FAST examinations are normal. Hemoglobin is 9.1 g/dL, base deficit is -8 mmol/L, and lactic acid level is 4.5 mmol/L. He is transferred to the ICU, where his mean arterial pressure is 51 mm Hg. On focused echocardiography, his inferior vena cava is 1.4 cm with greater than 50% collapsibility and left ventricular walls are touching on parasternal long-axis view.

Which of the following is the most likely cause of shock?
A. Heart failure
B. Infection
C. Hypovolemia
D. Spinal cord injury

25. Which of the following is correct regarding the hemodynamic measurements afforded by a central venous catheter?
A. Central venous pressure (CVP) is not affected by cardiac function.
B. Isolated right ventricular dysfunction will not affect CVP.
C. Central venous oxygen saturation parallels true mixed venous oxygen saturation.
D. There is little value in CVP waveform analysis.

26. A 25-year-old woman is admitted to the ICU after a motor vehicle collision. On ICU day four, she has diffuse body rash and shock. A retained tampon is found on tertiary survey. After receiving broad-spectrum antibiotics and three liters of crystalloid IV fluids, she remains hypotensive. She is started on vasopressors for refractory shock and intubated for respiratory distress.

Regarding the use of pulse pressure variation to help determine if she is volume responsive, which of the following is correct?
A. It is accurate if the patient is taking spontaneous breaths while on mechanical ventilation.
B. It is accurate if the patient is in atrial fibrillation.
C. It is accurate in patients with left ventricular and right ventricular dysfunction or valvular disorders.
D. A pulse pressure variation greater than 13% accurately predicts that the patient will be volume responsive.
27. Two days ago, a man underwent cardiac bypass surgery with revascularization of three vessels and placement of a bioprosthetic aortic valve. His postoperative course was complicated by cardiogenic shock requiring inotropic support. He required transfusion of blood products postoperatively. He was extubated and transitioned to supplemental oxygen via nasal cannula. His inotropic support was weaned off. He experiences an acute hypoxic event and is in acute respiratory distress and is then intubated. In the peri-intubation period, he has a cardiac arrest. As part of the procedure, a continuous capnogram is placed in line with the endotracheal tube.

Regarding end-tidal carbon dioxide (ETCO₂), which of the following statements is true?
A. An esophageal intubation is always manifested with a flat ETCO₂.
B. A decrease in ETCO₂ can be caused by fever, hypoventilation, partial airway obstruction, and the use of sodium bicarbonate.
C. An increase in ETCO₂ can be caused by hypothermia and hypovolemia.
D. An ETCO₂ value of greater than 20 mm Hg is a reflection of poor-quality chest compressions.
E. A sudden increase in ETCO₂ during chest compressions can be a sign of return of spontaneous circulation.

28. The arterial line tracing shown below is from a patient on positive pressure mechanical ventilation. Which letter best corresponds to the inspiratory phase of a positive pressure breath

A. A
B. B
C. C
D. Cannot tell from the information provided

29. Which of the following best characterizes the relationship of cardiac output and stroke volume during positive pressure ventilation while pulse pressure variation is observed on an arterial line tracing?
A. Right ventricular (RV) stroke volume increases and is highest during inspiration.
B. RV stroke volume decreases during expiration.
C. Left ventricular stroke volume is highest during expiration.
D. Systolic blood pressure decreases during the inspiratory phase.
E. RV stroke volume and cardiac output increase during the expiratory phase.
30. Which of the following will most likely interfere with obtaining an accurate assessment of pulse pressure variation for predicting fluid responsiveness?
A. Ventilation with tidal volume set at 10 mL/kg of ideal body weight
B. Ventilation with tidal volume set at 6 mL/kg of ideal body weight
C. Muscle relaxation with vecuronium
D. Continuous renal replacement therapy running at –50 mL/hr

31. An 83-year-old, 41-kg (90.4-lb) woman with aortic stenosis (aortic valve area 0.9 cm²) and syncopal episodes presents for an aortic valve replacement. Her preoperative echocardiogram demonstrates an ejection fraction of 60%, moderate aortic stenosis, moderate aortic insufficiency, and mild pulmonic insufficiency with mild pulmonary hypertension and evidence of diastolic relaxation abnormality. She also is found to have a 70% stenosis of her right coronary artery (RCA). She is admitted to the ICU after an uneventful coronary artery bypass graft to her and an aortic valve replacement. Over the course of the evening, her central venous pressure (CVP) increases from 10 to 15 mm Hg and phenylephrine is started because her blood pressure begins to decrease and is unresponsive to the administration of two liters crystalloid. Pulmonary arterial pressures increase from 20/15 mm Hg to 40/20 mm Hg, and her urine output begins to decrease, with an elevated lactate level. Arterial blood gas analysis reveals pH 7.21, partial arterial oxygen pressure 167 mm Hg (30% fraction of inspired oxygen), partial arterial carbon dioxide pressure 33 mm Hg. Chest tube output remains serosanguinous 20 mL/hour. Cardiac enzymes are within normal limits. CVP tracing is shown below.

Which of the following is the most likely cause of the patient’s clinical picture?
A. Cardiac tamponade
B. Pulmonary embolus
C. Tricuspid regurgitation
D. Kinking of RCA graft causing right ventricular ischemia
32. A 78-year-old woman admitted to the ICU for chest trauma develops a supraventricular arrhythmia with rapid ventricular response. Her blood pressure is 80/50 mm Hg, so the decision is made to perform synchronized cardioversion with 200 joules. Before synchronized cardioversion, she becomes unresponsive and pulseless as the rhythm changes to that shown in the figure below. The shock button on the defibrillator is pressed but it fails to deliver a shock.

Which of the following is the most likely reason for this failure?
A. The defibrillator battery failed.
B. The device is unable to sync.
C. Ventricular fibrillation cannot be shocked in sync mode.
D. A lead has lost contact, causing the irregular rhythm.

33. A patient in the ICU being treated with ciprofloxacin for colitis suddenly develops the rhythm and arterial line tracing shown below.

Which of the following is the most important next step in resuscitation for this patient?
A. IV magnesium, 2 g
B. IV vasopressin, 40-unit bolus
C. Overdrive pacing
D. Chest compressions at a rate of 100 per minute
34. A 65-year-old man collapsed on the street and a bystander performed cardiopulmonary resuscitation (CPR). Emergency medical personnel arrive nine minutes later and find the patient in asystole. CPR is continued and return of spontaneous circulation is obtained 20 minutes post arrest. The initial ECG suggests an anterior ST elevation myocardial infarction. The patient is transferred to a local hospital and undergoes emergency coronary angiography, which reveals a proximal left anterior descending artery occlusion, treated with percutaneous stenting. At 90 minutes post arrest, he is admitted to the ICU, where therapeutic hypothermia is immediately initiated. Three days later he is warmed, and neurologic examination is performed.

Which of the following is the most reliable indicator of a poor neurologic outcome?

A. Presence of myoclonus
B. Absence of pupillary reflex
C. Absence of corneal reflex
D. Absence of motor movements

35. A 27-year-old man falls off a fishing boat and is rescued by prehospital providers after being submerged for 25 minutes in 3°C (37.4°F) water. He is pulseless and, after defibrillation with an automated external defibrillator, there is a transient return of spontaneous circulation (ROSC) and cardiopulmonary resuscitation is continued. At hospital admission, his core temperature is 18.2°C (64.8°F), pupils are fixed and dilated, and ventricular fibrillation is identified. Multiple defibrillation attempts, along with the administration of advanced cardiac life support protocol drugs are unsuccessful at establishing ROSC. Laboratory results are: arterial blood gases, pH 6.71, partial arterial carbon dioxide pressure 75, partial arterial oxygen pressure 70, base excess -22.1 mmol/L; sodium 142 mEq/L, potassium 5.9 mEq/L, chloride 108 mEq/L, bicarbonate 10 mEq/L, lactate 18 mmol/L.

Which of the following is the most appropriate next step in management?

A. Administer procainamide before making a defibrillation attempt.
B. Initiate rewarming with forced air, lavage of warm saline in the stomach, and infusion of warm saline IV.
C. Initiate venoarterial extracorporeal membrane oxygenation support.
D. Obtain brain CT to identify irreversible changes secondary to hypoxia.
36. A 53-year-old woman collapses on the street, witnessed by bystanders, who initiate cardiopulmonary resuscitation and call emergency medical services (EMS). When EMS arrive, they find her to be in ventricular fibrillation and resuscitate her with chest compressions, epinephrine, and defibrillation. On arrival in the emergency department, she is intubated for airway protection, given her depressed mental status, and remains hemodynamically stable. Her ECG shows diffuse ST depressions.

Which of the following interventions is most likely to improve her neurologic outcome?
A. Amiodarone, 150 mg bolus, followed by 1 mg/min for 8 hours, then 0.5 mg/min for 16 hours
B. Active temperature control to a target temperature of 33-36°C (91.4-96.8°F) for at least 24 hours
C. Lung-protective ventilation strategy (including tidal volume 4-8 mL/kg of ideal body weight)
D. Immediate cardiac catheterization for revascularization

37. A 58-year-old man with a history of chronic low back pain, hyperlipidemia, and moderate chronic obstructive pulmonary disease (COPD) is evaluated in the emergency department for progressively worsening dyspnea. He is admitted to the hospital for COPD exacerbation. He is treated with albuterol nebulizer, prednisone, and noninvasive ventilation. His symptoms improve and he is discharged on hospital day five. One week later, he collapses at the airport while traveling. He is unresponsive and pulseless.

Which of the following is the most appropriate immediate course of action?
A. Administer IM naloxone, 0.4 mg.
B. Check airway patency.
C. Put automated external defibrillator pads in place.
D. Start chest compressions.

38. A 45-year-old homeless man is admitted to the psychiatric ward for acute psychosis. His vital signs have been stable and he is eating well. On hospital day 3, he complains of shortness of breath. His blood pressure is 80/35 mm Hg, heart rate 165 beats/min respiratory rate 20 breaths/min and oxygen saturation 94% on 4 L/min nasal cannula. ECG reveals supraventricular tachycardia.

Which of the following is the most appropriate intervention at this time?
A. Metoprolol, 5 mg IV
B. Naloxone, 0.4 mg IV
C. Synchronized cardioversion
D. Verapamil, 2.5 mg IV
39. A 71-year-old, 87-kg (192 lbs) man was admitted to the hospital nine days ago for worsening dyspnea and orthopnea. He is now transferred from the ward to the ICU for acute-onset fever of 38.8°C (101.8°F), tachycardia, and hypotension. He has a history of severe mitral stenosis. A transthoracic echocardiogram obtained on admission demonstrates a mean transvalvular gradient of 17 mm Hg and a valve area of 0.88 cm².

Which of the following is the most appropriate initial management of this patient?
A. Bumetanide, 4 mg IV bolus
B. Esmolol, 100 mg IV bolus followed by IV esmolol infusion
C. Epinephrine, 0.1 µg/kg/min IV infusion
D. Ringer lactated solution, 250 mL IV bolus

40. A 68-year-old woman with a long history of smoking and hypertension presents with moderate back pain. A CT angiogram identifies an enlarged thoracic aorta (5.6 cm in diameter), with a pedunculated outpouching near the takeoff of the left subclavian artery. An esmolol infusion is started overnight for blood pressure and heart rate control. The next morning, she undergoes an endovascular repair of the aneurysm and is then admitted to the cardiovascular ICU. Several hours later, the patient has decreased lower extremity movement on neurologic examination.

Which of the following is the most appropriate course of action?
A. Clamp the spinal drain.
B. Increase the blood pressure.
C. Transfuse red blood cells.
D. Obtain an emergent CT myelogram.

41. A 35-year-old, previously healthy man is evaluated in the emergency department for worsening dyspnea. He says that his shortness of breath started during the past week and is getting worse. He denies chest pain or leg swelling but reports subjective fevers and chills. He is admitted to the medical floor for presumed pneumonia and started on antibiotics based on his low-grade fever, tachycardia, oxygen requirement, and chest radiograph showing diffuse interstitial disease and mild pulmonary edema. During the next 12 hours his symptoms worsen, and he is transferred to the ICU for agitation, worsening dyspnea, and hypotension unresponsive to IV fluids. Vitals signs are: temperature 39°C (102.2°F), heart rate 128 beats/min, blood pressure 85/60 mm Hg, respiratory rate 24 breaths/min, oxygen saturation 93% on nonrebreather mask. Physical examination is notable for faint diastolic murmur, bilateral lung crackles, and track marks on the left forearm. Laboratory analysis is notable for white blood cell count of 23,000/µL, creatinine 1.8 mmol/L, lactate 4 mmol/L, and troponin of 1.04 ng/ml. ECG shows sinus tachycardia. Blood cultures are positive for methicillin-resistant Staphylococcus aureus.
Which of the following is the best next step in management?
A. Broaden antibiotics and give 2 liters IV normal saline for hypotension and lactic acidosis.
B. Broaden antibiotics, obtain immediate echocardiography, consult cardiac surgery, and institute appropriate medical management.
C. Call cardiology for catheterization laboratory activation and placement of intra-aortic balloon pump.
D. Broaden antibiotics, obtain immediate echocardiography, institute appropriate medical management, and consult cardiac surgery when blood cultures clear.

42. An 83-year-old man with severe aortic stenosis, chronic obstructive pulmonary disease, and coronary artery disease, who is status post coronary artery bypass graft, hypertension, and hyperlipidemia, is admitted to the ICU hemodynamically stable and comfortable after a transcatheter aortic valve replacement via a transfemoral approach using a balloon expandable valve. Sixty minutes after ICU admission, his vital signs have acutely changed: temperature is 37.9°C (100.2°F), blood pressure 82/50 mm Hg, heart rate 145 beats/min, oxygen saturation 90% on nonrebreather face mask. He is awake and denies chest pain. Physical examination demonstrates clear lungs bilaterally, regular tachycardia, low-pitched early diastolic murmur, and a soft S1 sound. His jugular venous pulsation is visible 5 cm above the sternal notch and refills slowly after occlusion. His extremities are cold, and he has thready pulses at the radial, dorsalis pedis, and posterior tibial arteries.

Which of the following is the most likely etiology for his change in physiology?
A. Pericardial tamponade
B. Acute mitral insufficiency
C. Acute aortic insufficiency
D. Aortic dissection

43. A 60-year-old man was admitted to the oncology floor for a urinary tract infection associated with mild confusion. His medical history is significant for metastatic non-small cell lung cancer, status post chemotherapy and radiation therapy, hypertension, and a deep venous thrombosis, for which he takes enoxaparin. Throughout the night, he is noted to have more shortness of breath and new-onset hypotension, and is urgently transferred to the ICU, where his vital signs are: temperature 38°C (100.4°F), heart rate 140 beats/min, respiratory rate 35 breaths/min, blood pressure 70/50 mm Hg, oxygen saturation 99% on a nonrebreather mask. Physical examination is notable for equal and reactive pupils, distended neck veins with supple neck, clear lungs, distant heart sounds, soft abdomen, cool extremities without edema, and confusion. Chest radiograph shows clear lungs and cardiomegaly. ECG shows sinus tachycardia with low voltage. Laboratory results are pending.
Which of the following is the most appropriate next step in management?

A. Intubate, then administer 2 L IV crystalloid fluid bolus and broad-spectrum antibiotics, and obtain pan-cultures.
B. Initiate crystalloid resuscitation while performing bedside cardiac ultrasound.
C. Initiate bilevel positive airway pressure; IV morphine, 2 mg, for dyspnea; IV furosemide, 40 mg; and consult cardiology for new-onset congestive heart failure.
D. Initiate massive blood transfusion protocol and give protamine for enoxaparin reversal.
E. Give IV tissue plasminogen activator for massive pulmonary embolism.

44. A 30-year-old man with a history of seizures, for which he takes phenytoin, is evaluated in the emergency department for shortness of breath and fatigue. He says that his symptoms have progressed during the past few weeks despite an antibiotic prescribed by his primary care provider. Vitals signs on arrival are: temperature 38.5°C (101.3°F), heart rate 110 beats/min, respiratory rate 26 breaths/min, blood pressure 100/70 mm Hg, oxygen saturation 93% on 8-L/min nasal cannula. Physical examination is notable for normal mentation, supple neck, elevated jugular venous distension, tachycardia with an S3 gallop, crackles in the lung bases, soft abdomen, warm extremities with 1+ bilateral lower extremity pitting edema, and a faint but diffuse macular rash on his trunk and legs. ECG shows nonspecific T wave inversions. Chest radiograph shows mild pulmonary edema. Laboratory assessment is notable for a troponin 3.0 ng/ml, B-type natriuretic peptide 3,000 pg/ml, creatinine 1.70 mg/dL, sodium 130 mEq/L, potassium 4.4 mEq/L, white blood cell count 13,000/µL, hemoglobin 12 g/dL, platelets 265,000/µL, eosinophils 1,100/µL, aspartate aminotransferase 280 U/L, alanine aminotransferase 250 U/L, alkaline phosphatase 60 U/L, bilirubin 1.0 mg/dL. Echocardiography shows biventricular systolic dysfunction with ejection fraction of 20%.

Which of the following diagnostic tests is most important in determining treatment and prognosis?

A. Skin punch biopsy
B. Right upper quadrant ultrasound
C. Right heart catheterization
D. Endomyocardial biopsy
E. Blood cultures
1. **Rationale**

   All patients with stress-induced cardiomyopathy who develop shock need urgent echocardiography to evaluate for left ventricular outflow tract obstruction, occurring in 13% to 18% of patients. Shock in patients with left ventricular outflow tract obstruction should not be treated with inotropic agents, which can worsen the obstruction. Beta-blockers may resolve the obstruction and improve hemodynamics. Alpha-agonists may also improve hemodynamics by increasing afterload and reducing the gradient. However, the use of alpha-agonists requires close observation for vasoconstrictive effects that may be dangerous in patients at risk for coronary vasospasm. Patients who do not respond to initial medical therapy may respond to an intra-aortic balloon pump despite the small risk that afterload reduction will exacerbate the obstruction.

**References:**


2. **Rationale**

   Prolongation of the QT interval can be congenital or acquired by drug administration. This may be the result of the drug itself or the inhibition of metabolic pathways that will increase the effective dose of the drug. A QTc greater than 500 ms is a prolonged QT that may result in torsades de pointes (TdP) and possibly sudden death. Repletion of magnesium is useful for the prevention and treatment of TdP, and removal of any agent that may prolong the QTc is optimal. Amiodarone would be harmful because it could actually lower the heart rate and increase the QT interval. Bicarbonate has no role in QTc prolongation. Methadone could lead to prolonged QT syndrome and should be discontinued.

**Answer: C**

**Answer: A**
Rationale Answer: B

This patient has evidence of cardiogenic shock secondary to Takotsubo cardiomyopathy from intense emotional stress. This is a rapidly reversible heart failure syndrome that often mimics acute coronary syndrome and is essentially a diagnosis of exclusion. Patients often have ST elevation on ECG and elevated troponin levels and either no coronary artery disease or very minimal amounts on catheterization. Classic findings on echocardiography are apical ballooning due to hypokinesis of the apex and a hyperdynamic basal segment. The pathophysiology of the disease is thought to be excessive catecholamine stress on the heart.

Treatment for patients with Takotsubo cardiomyopathy is generally supportive but a minority of patients will develop hemodynamic compromise requiring further action. Although no guidelines exist, it is generally recommended to avoid giving additional catecholamines, such as dobutamine or norepinephrine, since this may worsen the condition and stress on the heart. If left ventricular outflow tract (LVOT) obstruction is present, tachycardia should be avoided, since it will reduce diastolic filling time. Increased inotropic stimulation can also worsen LVOT obstruction. Phenylephrine is the vasopressor of choice, combined with beta blockade in this scenario. An intra-aortic balloon pump is an option for patients in cardiogenic shock without LVOT obstruction, as well as aggressive diuresis.

References:
4. **Rationale**

Patients with advanced heart failure have improved survival rates and quality of life when treated with implantable left ventricular assist devices (LVADs) compared to medical therapy. LVADs are now placed as a bridge to cardiac transplantation and as end-destination therapy. With the higher prevalence of these devices in the community, it is important for the intensivist to have an understanding of their basics. The inflow cannula (blood to device) is inserted into the apex of the left ventricle. The outflow cannula (blood to patient) is inserted into the ascending or descending aorta. The pump speed with the programmable variable is titrated in the operating room and ICU. In the immediate postoperative period, complications include bleeding, hypovolemia, arrhythmia, thrombosis, in-flow cannula “suck-down” events, device malposition, and right-sided heart failure. Later complications include infection, thrombosis, and drive line/machine issues. Troubleshooting these early complications in the ICU requires a thorough review of the available hemodynamic parameters, review of the right-side heart physiology, and close evaluation of echocardiographic findings. In this case, the bedside echocardiogram clearly shows right ventricular compression and physiologic parameters consistent with tamponade.

**References:**

5. **Rationale**

Cerebral hyperperfusion syndrome following a carotid endarterectomy classically occurs two to three hours postoperatively. Signs and symptoms can include ipsilateral headache, eye pain, somnolence, agitation, confusion, and seizures. It is thought to be a result of impaired autoregulation distal to the stenosis, resulting in excessive blood flow after removal of the stenosis. Systemic hypertension can exacerbate the resulting cerebral edema, so pharmacologic reduction in cerebral perfusion pressure with drugs such as labetalol is appropriate. The syndrome usually resolves within 24 hours after surgery. Treating symptoms with additional narcotics would be beneficial only if the blood pressure elevation is the result of surgical pain, which does not appear to be the case. Radiographic and Doppler imaging in the absence of lateral neurologic deficits are not warranted.
Rationale  
Answer: D

Qualitative capnometry is a simple and rapid confirmatory test for appropriate endotracheal tube placement. The device is attached in-line between the endotracheal tube and the delivery system. The chemical indicator in the device will change color depending on the CO$_2$ concentration detected. The higher the CO$_2$ level, the more color change will be seen from purple to yellow.

In patients with profoundly low cardiac output, end-tidal CO$_2$ levels may be significantly lowered due to diminished lung perfusion. Ventilation is directly correlated to pulmonary perfusion. A profoundly low cardiac output could cause the clinical scenario in this case. Possible additional factors are ineffective chest compressions, massive pulmonary emboli, tension pneumothorax, or some other cause of inadequate cardiac output during cardiopulmonary resuscitation. Often there will be a significant increase in end-tidal CO$_2$ levels once return of spontaneous circulation has been achieved. Severe acidosis would not be associated with a lower level of end-tidal CO$_2$.

Venous hyperoxia has been documented in post-cardiac arrest patients after the return of spontaneous circulation. It is presumed to be secondary to bioenergetics failure and defects in oxygen utilization after a prolonged pulseless time. This does not have any impact on end-tidal CO$_2$ detection.

It would be extremely unlikely that low-level color change is due to hypoventilation after endotracheal intubation. It would be even more unlikely that the low-level color change persisted after the delivery of multiple breaths.

References:
7. **Rationale**

A potential complication of peripheral venoarterial extracorporeal membrane oxygenation (ECMO) is differential hypoxia, also known as north-south syndrome. If there is adequate native cardiac function with concomitant respiratory failure, the mixing point of retrograde blood flow from the femoral artery can be more distal, leading to upper body hypoxemia. This can be monitored by comparing arterial blood gas analysis results from the patient’s right radial artery to those from the left, as well as comparing to post-oxygenator blood gas values. Solutions to this problem can be to change the central cannulation, increase ECMO flows, or consider a change to venovenous ECMO if cardiac function is adequate.

Based on the post-oxygenator blood gas values, the oxygenator is working effectively and does not need to be changed, nor does an additional one need to be added. Increasing the fraction of inspired oxygen, which is already at 60%, would be suboptimal for a patient with acute respiratory distress syndrome, and could lead to oxygen free radical toxicity.

**References:**


8. **Rationale**

The patient presents with acute pericarditis probably related to his pneumonia. The prototypical features of acute pericarditis are acute chest pain with radiation to the trapezius ridge, that is improved with sitting forward, pericardial friction rub, and an ECG tracing with diffuse ST elevation with concave upstrokes and reciprocal ST depression in leads VR and V1. Transthoracic echocardiography should be obtained to evaluate for the presence of pericardial effusions that can lead to pericardial tamponade. In 80% to 90% of acute pericarditis cases, the etiology is idiopathic. Up to 90% of acute idiopathic pericarditis cases are self-limiting. Recommended initial treatment is nonsteroidal anti-inflammatory drugs. Combination therapy with colchicine can also be used. A recent study showed that combination therapy has been shown to reduce the rate of persistent and recurrent pericarditis.

**References:**

9. **Rationale**  
**Answer: B**

This patient has an inferior ST elevation myocardial infarction (STEMI) with cardiogenic shock. Even though he recently had surgery, aspirin is indicated. The 2015 American Heart Association (AHA) guidelines recommend doses of 162-325 mg. Nitroglycerin is appropriate in STEMI to relieve chest pain; however, both hypotension with a systolic blood pressure less than 90 mm Hg and a suspected right ventricular infarct, such as in this case, are contraindications to nitroglycerin. Similarly, morphine can be considered for chest pain but is not the most important next step. Diltiazem is contraindicated in STEMI with evidence of cardiogenic shock. Currently, the administration of supplemental oxygen to normoxic patients with acute coronary syndrome is not well supported by data. The 2015 AHA guidelines state that withholding oxygen in normoxic patients can be considered. Therefore, with the patient’s oxygen saturation of 98%, providing 100% oxygen is unlikely to be the most important next step.

**Reference:**

10. **Rationale**  
**Answer: B**

Several studies have demonstrated the superior sensitivity of precordial leads compared to limb leads for the detection of perioperative myocardial ischemia. London et al demonstrated that precordial leads V4 and V5 had the highest sensitivity individually. Combining leads II, V4, and V5 had a sensitivity of 96%, which increased to 100% with the addition of V2 and V3. This stresses the importance of appropriate lead placement as well as review of all leads when there is clinical concern.

**References:**
11. Rationale

This patient has Wolff-Parkinson-White (WPW) syndrome, as evidenced by the short PR interval and presence of a delta wave on the ECG. WPW is one of the pre-excitation syndromes, which cause early depolarization of the ventricle through an abnormal connection between atria and ventricles called the accessory pathway. WPW syndrome is very rare, existing in only 0.1-3 per 1000, and is usually asymptomatic. Some patients, however, may experience syncope and palpitations. They are at greater risk for atrial fibrillation, which can lead to ventricular fibrillation. Patients are also at risk for circus reentrant tachycardias leading to paroxysmal supraventricular tachycardia or ventricular tachycardia and also for sudden cardiac death. Treatment options for people with arrhythmias and WPW syndrome include non-pharmacologic therapies (ie, catheter ablation of the accessory pathway) and pharmacologic therapy (to slow ventricular heart rates or to prevent arrhythmias).

In patients with WPW, a sinus beat originates from the sinoatrial (SA) node and goes through the accessory pathway and also through the atrioventricular (AV) node. Since the ECG is a summation of vectors seen by each lead, the result is a shorter PR interval and wider QRS than normal; we also see the characteristic delta wave. ST-T wave changes are also possible. In general, the AV node has a longer refractory time than the accessory pathway; therefore, an impulse transmitted through the accessory pathway will activate the ventricles earlier than if it had traveled through the AV node. Impulses can be transmitted in a retrograde or anterograde fashion through the accessory pathway, thus allowing for the development of circus reentrant tachycardias.

Sympathetic stimulation (anxiety, hypovolemia, hypoxemia and acidosis, light planes of anesthesia) as well as drugs (pancuronium, meperidine, ketamine, ephedrine, digoxin, verapamil) and electrolyte abnormalities (potassium and calcium) can induce tachyarrhythmias by increasing conduction through the accessory pathway.

When atrial contraction becomes chaotic, as in atrial fibrillation (atria can produce 300 to 500 contractions per minute), the AV node will produce a narrow complex beat (irregularly irregular narrow-complex tachycardia) and the accessory pathway will produce a wide complex beat (irregularly irregular wide-complex tachycardia). P waves will not be present. WPW atrial fibrillation is the only arrhythmia in which there is irregularly irregular wide- and narrow-complex tachycardia.

The combination of atrial fibrillation and WPW can be fatal because of rapid conduction of the atrial activity through the accessory pathway, resulting in rapid ventricular rates and leading to ventricular fibrillation. AV nodal blocking agents (metoprolol, diltiazem, digoxin, sotalol) should be avoided in this setting because they paradoxically increase ventricular rates since more atrial activity will pass through the fast-conducting accessory pathway and fewer through the AV node itself. Procainamide has been the traditional recommended therapy for unstable WPW. It is a class Ia antiarrhythmic, which increases the refractory period and decreases conduction through the accessory pathway.

Procainamide is typically infused intravenously at 20 to 50 mg/min, given while monitoring blood pressure closely every 5 to 10 minutes until the arrhythmia terminates, hypotension ensues, the QRS is prolonged by more than 50%, or a total of 17 mg/kg (1.2 g for a 70-kg (154.3-lb) patient) has been given. Even if it does not result in tachycardia termination, IV procainamide will usually slow the tachy-
cardia rate and improve the hemodynamic state. Of course, synchronized cardioversion should be performed in patients who are hemodynamically unstable.

References:

12. Rationale

In the management of aortic stenosis, maintenance of sinus rhythm is beneficial, to allow appropriate diastolic filling needed to maintain adequate cardiac output. Cannon a waves are created when blood pushes against stenotic mitral or tricuspid valves and when there is a loss of atrioventricular (AV) synchrony. The a wave produced by the simultaneous contraction of the atria and ventricles occurs later in the cardiac cycle at the timing of the normal v wave. The combination of the a and v waves creates the cannon wave. Cannon waves will also appear if there are premature ventricular contractions, or reentrant ventricular tachycardia.
13. **Rationale**

This patient has a toxic level of digoxin, which commonly occurs in older patients who develop acute kidney injury. Digoxin is associated with a number of cardiac dysrrhythmias, including supraventricular tachycardia, sinus bradycardia, atroventricular blocks, and ventricular tachycardia. Calcium chloride is theoretically contraindicated in digoxin overdose because it may potentiate digoxin’s mechanism of action. Digoxin immune Fab is the best treatment for digoxin overdose. In acute ingestions, activated charcoal could be considered, but her symptoms have been present for more than a day. Potassium repletion is not indicated in digoxin toxicity. At this time, given her blood pressure, there is no role for epinephrine. Dialysis has, at best, minimal effects on digoxin clearance.

**References:**


14. **Rationale**

Atrial fibrillation (AF) that occurs during critical illness presents challenges for management. Recent evidence suggests that patients in whom AF develops during acute illness (eg, sepsis) have high long-term risks for AF-associated complications, such as stroke, heart failure, and death. Therefore, all patients with AF lasting longer than 48 hours should be evaluated for anticoagulation. Decisions about appropriate thromboprophylaxis require individual assessment of stroke risk and bleeding risk on such therapy. Validated risk scoring tools have been developed for clinical use to assess the risks of bleeding and thrombosis.

The CHADS2 score is a simple and popular tool to assess thrombosis risk in patients with AF. However, recent data suggests that this score fails to account for risk in several common subpopulations, including patients with peripheral vascular disease, women, and those for whom variations in thrombotic risk occur with age. To account for these limitations, the CHA2DS2-VASc score was developed and is currently recommended for use in AF risk assessment. In general, for patients who have had prior stroke, transient ischemic attack, or CHA2DS2-VASc score of 2 or higher, oral anticoagulants are recom-
Self-Assessment in Adult Multiprofessional Critical Care

The intra-aortic balloon pump (IABP) is a mechanical circulatory device that has been used to support patients in cardiogenic shock from myocardial infarction, intractable ventricular arrhythmias, and acute decompensated heart failure. The timing of inflation and deflation of the IABP is crucial in facilitating hemodynamic support by increasing coronary perfusion and reducing afterload. Electrocardiography and systemic aortic pressure waveforms can be used to trigger the IABP. In a patient with sinus rhythm, the IABP is set to inflate mid-T wave and deflate at the peak of the R wave on ECG, which corresponds to the start of diastole and systole, respectively. In the aortic pressure waveform trigger mode, the balloon inflates with the dicrotic notch, which corresponds to aortic valve closure, and deflates before the systolic arterial upstroke that represents aortic valve opening.

The pressure waveform depicted shows diastolic augmentation and an absence of a dicrotic notch. This indicates that the balloon is inflating early. Early balloon inflation impairs systolic function by increasing afterload and myocardial oxygen demand.
References:

16. Rationale

Resistance to loop diuretics is a common complication of chronic therapy in congestive heart failure. Furosemide effectively blunts the reabsorption of sodium and water in the thick ascending limb of the loop of Henle, but this delivers a greater luminal sodium concentration to the distal convoluted tubule (DCT). Cells in the DCT hypertrophy in response to this increased sodium concentration, and absorb more sodium and water, which decreases the effectiveness of furosemide.

Loop diuretics act at targets on the luminal side of the thick ascending limb of the loop of Henle. To get to its target, furosemide must be filtered at the glomerulus and then secreted into the tubular lumen by the organic ion transporter. Blood urea nitrogen and other organic acids compete for binding sites on that transporter and make furosemide secretion less efficient.

The reduction in plasma volume caused by effective diuresis results in activation of the renal-angiotensin-aldosterone system and the sympathetic nervous system. This reduces glomerular filtration rate, leads to decreased tubular flow, and increased reabsorption of sodium and water.

Furosemide binding to plasma proteins increases the effectiveness of the drug because plasma binding is required for secretion into the tubular lumen. Hypoalbuminemia is common in heart failure and enhances conversion of furosemide to its inactive form. Increased plasma protein binding increases, not decreases, the effectiveness of furosemide as a loop diuretic, which is why co-administration of furosemide with albumin has been demonstrated to improve the response to diuretics in patients with cirrhosis and nephrotic syndrome.

References:
17. **Rationale**  

This patient’s device has an increase in power required to drive the impeller at an unchanged speed and a reduced pulsatility index. The pulsatility index is an ongoing measure of the contribution of the patient’s native left ventricular (LV) contraction to flow through the device. Impeller thrombosis obstructs optimal impeller function and increases the power required to achieve the set speed. In addition, the degree to which native LV contraction can contribute to increases in flow through the impeller is blunted by the presence of a thrombus in the blood flow path. This leads to a decrease in the pulsatility index. Increased serum lactate dehydrogenase (LDH) and total bilirubin are consistent with red cell hemolysis, which is a hallmark of pump thrombosis. Because the impeller is less effective at generating flow, total cardiac output can decrease, which might lead to a decrease in mixed venous oxygen saturation (SmvO$_2$). Popliteal vein thrombosis might increase LDH and total bilirubin, but it should not change the power requirement or decrease the patient’s mixed venous oxygen saturation. Right ventricular failure might decrease SmvO$_2$ and increase total bilirubin, but would not decrease pump power or increase LDH. Similarly, acute mitral regurgitation might reduce the SmvO$_2$ and increase total bilirubin, but would not cause a change in pump power or increase LDH. The option most consistent with these findings is impeller thrombosis.

**Reference:**  

18. **Rationale**  

This patient has a rapidly failing heart and requires immediate stabilization until she can either undergo definitive operative management or otherwise be hemodynamically stabilized. Other possible interventions include the placement of alternative cardiac assist devices. All other proposed interventions might be expected to lead to immediate cardiovascular collapse. In particular, initiating a phenylephrine infusion, which might be a feasible intervention after the insertion of a cardiac assist device, would, as a primary intervention in this acute setting, increase afterload and further decrease cardiac index. The patient is already in fulminate congestive heart failure and giving more fluids would exacerbate her already tenuous hemodynamic condition. Low-dose esmolol might be cautiously used in other similar situations but here would undoubtedly contribute to the patient’s hypotension.

**References:**  
19. **Rationale**

This patient has an elevated lactate dehydrogenase, with low pulsatility index, and high pump power, which indicates pump thrombosis. With the thrombosis in his device, there is increased resistance in the LVAD, which reduces its ability to offload the heart. The left ventricle must now expel most of the blood through the aortic valve instead of through the device, which explains the normal blood pressure. Thrombolytic therapy has been attempted to remove clot burden in the LVAD with nominal success but is associated with higher risk of bleeding complications. In the context of pump thrombosis, increasing the LVAD speed will hemolzye blood more. The patient’s INR is appropriate; increasing the goal therapy will increase risk of bleeding. The best course of action is to exchange the device.

**References:**

20. **Rationale**

The first description of a pulmonary arterial catheter (PAC) inserted without the use of fluoroscopy appeared in 1970. It has multiple ports to allow for measurement of intracardiac pressures and thermodilution cardiac output, infusion of medications, and atrial or ventricular pacing. The catheter can be placed at bedside in the ICU without fluoroscopy through a large-bore central venous introducer. The PAC’s position can be identified using the characteristic waveforms as the catheter traverses through the superior vena cava, right atrium, and right ventricle into the pulmonary artery. The figure below shows the waveforms seen as the PAC traverses cardiac structures.

The correlation between the right atrial, ventricular, pulmonary arterial, or pulmonary arterial occlusion pressure (also called pulmonary capillary wedge pressure) and left ventricular pressure is poor and, as such, these measurements can at best be considered surrogates. Fluctuations in intrathoracic and pericardial pressures negatively impact the utility of wedge pressure measurement as well, since left ventricular filling pressure is truly a measurement of the transmural pressure across the ventricular wall. PACs still have an important role in the management of pulmonary hypertension, including diagnosis and monitoring response to therapy.

The Berlin criteria for acute respiratory distress syndrome no longer include PAC findings due to poor interobserver reliability in the diagnosis of noncardiogenic extravascular fluid. The use of PACs in the routine management of septic shock and traumatic brain injury is not supported by clinical trials.
References:

21. **Rationale**

Increased evidence has shown that static markers, such as central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP), of preload responsiveness are inaccurate. A growing body of literature supports the use of dynamic markers as better indicators. These include pulse pressure variation (PPV), stroke volume variation, and passive leg raise. The passive leg-raising technique involves lowering a patient’s thorax into a horizontal position and raising the legs 45 degrees in the air for 30 to 90 seconds. Passive leg raise is equivalent to a 150- to 300-mL IV bolus. Venous blood from the legs flows into the thorax, leading to an increase in preload and a subsequent increase in stroke volume,
if the patient is volume responsive. This method of assessing volume responsiveness is accurate in patients who are spontaneously breathing, mechanically ventilated, and who have cardiac arrhythmia, provided they are monitored with a cardiac output monitoring device to assess stroke volume. Devices that have been used are pulse contour continuous cardiac output, Vigileo/FloTrac, esophageal Doppler, and echocardiography.

PPV would be inaccurate in this patient because she is spontaneously breathing over the ventilator and her tidal volume is less than 8 mL/kg of IBW. PPV relies on heart and lung interactions, which are not constant in this patient. CVP due to the changes in venous tone, intrathoracic pressures, and heart compliance leads to a poor relationship between CVP and right ventricular end-diastolic volume in the critically ill patient. Numerous studies have demonstrated a poor relationship between CVP and the change in CVP in demonstrating fluid responsiveness. Similar results have been shown for PAOP.

References:

22. Rationale

Conventional monitors of stroke volume variation and cardiac output measurement are highly dependent on the aortic pulse contour for their measurements. Arrhythmias that alter beat-to-beat cardiac loading conditions will affect the reliability of measurements, as will use of an intra-aortic balloon pump by altering the diastolic waveform. Conditions associated with low vascular resistance also are problematic for these devices. Cyclic variations in respiratory waveform, as would be expected in a spontaneously breathing patient or in a patient with acute pulmonary disease manifesting with severe pulmonary noncompliance, also affect the reliability of the measurements. The monitors are generally recommended when patients are on a respirator with tidal volumes of at least 8 mL/kg. Hence the intraoperative patient without an arrhythmia receiving controlled ventilation while under anesthesia presents the most ideal conditions for this type of monitoring device.
23. Rationale

For both ventilated and nonventilated patients, hemodynamic parameters are measured when the chest comes to rest at the end of the exhalatory phase of the respiratory cycle. The point in the cardiac cycle where central venous pressure and pulmonary artery occlusion pressure (PAOP) readings are taken is the end of diastole, which coincides with the point at which the C wave occurs. The C wave is often not visible; however, in the case of the PAOP, the timing of the C wave coincides with the end of the QRS.

References:

24. Rationale

In recent years ultrasonography, particularly echocardiography, has become much more popular among intensivist for its portability, noninvasiveness, and ability to be repeated as many times as necessary. Disadvantages include operator dependency and the need for significant training, since under- or over-estimation of findings can lead to erroneous therapy.

In this patient, the echocardiographic findings speak against heart failure in that he has preserved contractility. Infection is unlikely, given the clinical scenario. His hemodynamic picture and echocardiographic findings suggest hypovolemia. Possible sources of bleeding, given the normal focused assessment with sonography in trauma examination and chest radiograph, include retroperitoneal hematoma or blood loss from long bone fracture.

References:

25. Rationale

Central venous pressure (CVP) is an accurate reflection of right atrial pressure, which is the back pressure for venous return. Right atrial pressure is typically kept low, so venous return is unimpeded. As such, cardiac dysfunction is typically reflected by an elevation in CVP. CVP is a reflection of right-sided filling pressures; therefore, processes that affect right-sided cardiac function can elevate this hemodynamic parameter. These include pulmonary hypertension, right ventricular dysfunction, and tricuspid regurgitation. The absolute values of central venous oxygen saturation are not identical to true mixed venous oxygen saturation and are typically higher during shock states. However, as hemodynamics change, the trend in these two parameters follow the same pattern. Analysis of CVP waveforms can provide useful insight into hemodynamics. For example, a large v wave indicates tricuspid regurgitation, and a large y descent indicates restrictive cardiac dysfunction. However, there is currently significant debate as to the utility of CVP monitoring, especially with respect to fluid status.

References:

26. Rationale

Determining volume responsiveness is a critical step in resuscitation care. Using dynamic markers of preload responsiveness is increasing, given the physiologic superiority of this approach and the poor sensitivity and specificity of static markers, such as central venous pressure. Dynamic measurements such as pulse pressure variation, stroke volume variation, and ultrasound assessment, provide more accurate determinations of the patient’s place on the Frank-Starling curve. Pulse pressure variation is often cited in the literature as one of the most precise measurements for determining volume status, but it is important to note that particular conditions apply. Positive pressure ventilation induces cyclic changes in preload and afterload on the right and left ventricle that are more pronounced when the patient is on the steep portion of the Frank-Starling curve. A pulse pressure variation of greater than 13% has been verified in the literature to indicate that the patient will likely be volume responsive. However, in order for this to be accurate, the patient must be in sinus rhythm, must not be taking spontaneous breaths, ideally should not be ventilated with very low tidal volumes, and should not have significant left ventricular or right ventricular dysfunction. This case also demonstrates the importance of performing a tertiary survey on all trauma patients.
References:

27. Rationale

Quantitative capnography should be considered for all mechanically ventilated patients because it provides a direct measurement of pulmonary ventilation. A normal ETCO₂ value in a healthy adult should be 35 to 45 mm Hg. Capnography indirectly provides measurements on metabolism and cardiac output. Physiologic states that decrease whole body metabolism, hypothermia, and poor circulating volume will be reflected numerically as a decrease in ETCO₂. In contrast, physiologic states that increase whole body metabolism, such as fever, will be reflected as an increase in ETCO₂. Physiologic states that decrease cardiac output, such as a pulmonary embolism, will be reflected as a decrease in ETCO₂. Pharmacologic interventions, such as giving a load of sodium bicarbonate, will be reflected almost immediately with an increase in ETCO₂ as the lungs compensate for the increased carbon dioxide load. Quantitative capnography can provide confirmatory information with endotracheal intubation. An esophageal intubation may initially have an ETCO₂ waveform due to intragastric CO₂ that rapidly disappears. Two practical uses of quantitative capnography during cardiopulmonary resuscitation are: 1) evaluating the effectiveness of chest compressions, and 2) identifying return of spontaneous circulation (ROSC). High-quality chest compressions are reflected by ETCO₂ values between 10 and 20 mm Hg in the intubated patient. ROSC is reflected as a spontaneous significant increase in ETCO₂ (toward the physiologic normal range of 35 mm Hg) as the heart improves cardiac output by regaining a rhythm and circulates CO₂ into the lungs.

References:

28. Rationale

The variation of the pulse pressure on an arterial line tracing during positive pressure mechanical ventilation reflects the effects of increasing and decreasing intrathoracic pressure in an alternating fashion, on stroke volume. This is sometimes best thought of by looking at the effects on the right and left heart separately.

Focusing on the right side of the heart, during inspiration on a positive pressure breath, there is an increase in intrathoracic pressure as air is forced into the lungs and alveoli from the ventilator. The
increased lung volume and pressure in the lungs and alveoli increases pulmonary vascular resistance, which increases right ventricular (RV) afterload. At the same time, the increase in intrathoracic pressure impedes venous return into the chest through the vena cava, which causes a decrease in preload. The combined effect of a decrease in preload and increase in afterload cause a decrease in stroke volume and cardiac output from the right heart into the pulmonary vasculature during inspiration.

During the positive pressure breath, the vena cava begins to back up (venous return is impeded) until the expiratory phase begins and the patient exhales. During exhalation, the intrathoracic pressure drops and the there is a rush of blood into the right side of the heart, and RV preload increases. At the same time, with air now leaving the lung, the decrease in pulmonary vascular resistance and RV afterload favors an increase in stroke volume from the right ventricle. The net result is an increase in stroke volume and cardiac output during the expiratory phase of mechanical ventilation for the right side of the heart.

On the left side of the heart we see the opposite effects, in part due to a time lag between the cardiac output during the preceding few beats, which is now arriving to the left. The primary mechanism affecting stroke volume in the left heart during inspiration is an increase in venous return, as the blood that was in the pulmonary vascular system is being pushed out as the lungs expand. In addition, although the cardiac output from the right side of the heart is diminished during the inspiratory phase, the blood returning to the left atrium from the lungs is the blood that was pumped from the right ventricle to the pulmonary vasculature during the expiratory phase immediately beforehand, which had a high stroke volume and high cardiac output. These two effects lead to an increase in venous return for the left ventricle and an increase in stroke volume throughout the inspiratory phase. This manifests as a progressive increase in the systolic pressure on the arterial waveform, as well as an increase in the pulse pressure throughout the inspiratory phase of a positive pressure mechanical breath (option A).

Again, the opposite occurs on the left side of the heart during the expiratory phase. There is less venous return to the left ventricle because the cardiac output from the right was reduced during the preceding few beats. This results in a decrease in stroke volume, and decrease in systolic pressure on the arterial waveform (options B and C).

These alterations in increasing and decreasing stroke volume with changes in intrathoracic pressure are exacerbated when a patient is on the ascending limb of the Frank-Starling curve. When end-diastolic volume is already adequate and the patient is on the plateau of the curve, the effects of positive pressure ventilation are diminished.

The arterial line and ventilator tracings show the pulse pressure variations during mechanical ventilation. The systolic blood pressure and pulse pressure are maximal during inspiration and decline in expiration.
References:

29. Rationale
   Answer: E

Focusing on the right side of the heart, during inspiration on a positive pressure breath, there is an increase in intrathoracic pressure as air is forced into the lungs and alveoli from the ventilator. The increased lung volume and pressure in the lungs and alveoli increases pulmonary vascular resistance, which increases right ventricular (RV) afterload. At the same time, the increase in intrathoracic pressure impedes venous return into the chest through the vena cava, which causes a decrease in preload. The combined effect of a decrease in preload and increase in afterload results in a decrease in stroke volume and cardiac output from the right heart into the pulmonary vasculature during inspiration.

During the positive pressure breath, the vena cava begins to back up (venous return is impeded) until the expiratory phase begins and the patient exhales. During exhalation, the intrathoracic pressure drops and there is a rush of blood into the right side of the heart, and right ventricular preload increases. At the same time, with the air now leaving the lung, the decrease in pulmonary vascular resistance and RV afterload favors an increase in stroke volume from the right ventricle. The net result is an increase in stroke volume and cardiac output during the expiratory phase of mechanical ventilation for the right side of the heart.

The primary mechanism affecting stroke volume in the left heart during inspiration is an increase in venous return, as the blood that was in the pulmonary vascular system is being pushed out as the lungs expand. This leads to an increase in venous return for the left ventricle and an increase in stroke volume throughout the inspiratory phase. This manifests as a progressive increase in the systolic pressure on the arterial waveform, as well as an increase in the pulse pressure throughout the inspiratory phase of a positive pressure mechanical breath.

Reference:

30. Rationale
   Answer: B

The changes in pulse pressure variation (PPV) on an arterial line tracing are a manifestation of the change in intrathoracic pressure. Increasing the tidal volume will result in a greater change in intrathoracic pressure and have a greater effect on right ventricular (RV) venous return. Likewise, decreasing tidal volumes will mute this effect and diminish the variability. For optimal detection of PPV, tidal volumes of 8 to 10 mL/kg or greater have been recommended.
Muscle relaxation with vecuronium (option C) will not interfere with accurate PPV measurements. Ventilator dyssynchrony will interrupt the rhythmic change in positive pressure in the chest and may interfere with accurately interpreting PPV. Muscle relaxation would therefore assist in obtaining accurate readings.

Continuous renal replacement therapy running at net –50 mL/hr (option D) would not have an effect on the accuracy of PPV. It may make PPV more pronounced over time because volume is taken off but it will not change the ability to accurately assess PPV.

Atrial fibrillation, premature ventricular contractions, premature atrial contractions, third-degree heart block, supraventricular tachycardia, ventricular tachycardia, and ventricular fibrillation will all interfere with the regular rhythmic heart rate needed to reliably determine PPV. Other variables that may decrease the effects of positive pressure ventilation on PPV include high respiratory rates, decreased lung compliance, decreased chest wall compliance, and RV failure.

Reference:

31. Rationale

This patient has clearly developed right heart failure as evidenced by her rising CVP, elevated pulmonary artery pressures, declining blood pressure, and renal failure. In cardiac tamponade, atrial and ventricular pressures equalize; therefore, when the tricuspid valve opens, there is not a prominent y descent. The x descent appears steep with an attenuated y descent.

A pulmonary embolus (PE) would cause right ventricular (RV) strain and elevated CVP. A PE large enough to cause RV failure would also cause hypoxemia and tachypnea, leading to respiratory alkalosis.

This patient demonstrates RV failure leading to elevated CVP, low mean arterial pressure due to severe tricuspid regurgitation, and volume overload. In a patient this size, the two liters of fluid given are likely to cause acute dilation of the right ventricle, stretching of the tricuspid annulus and acute tricuspid regurgitation that may lead to hepatic congestion, liver failure, and renal failure. Medical management includes inotropes and elevated heart rate to decrease diastolic filling time. The CVP waveform depicts an exaggerated v wave indicative of elevated RA pressure from backflow from the right ventricle into the RA through the patent tricuspid valve during ventricular systole when blood is supposed to be ejecting through the pulmonic valve.

RV ischemia due to graft dysfunction is not likely to cause severe tricuspid regurgitation without any other right ventricular wall motion abnormalities. A list of various CVP waveforms and their associated pathology is found below:
### References:


32. **Rationale**

When using a non-automated defibrillator, clinicians should monitor for rhythm changes before delivery of synchronized cardioversion or defibrillation. A battery failure is a rare event, and is prevented further by regular system checks and maintenance to guarantee that the device is fully charged. Sudden arrhythmias are always dynamic scenarios in which a loose lead could be responsible for the observed rhythm.

Clearly this patient had a supraventricular arrhythmia with hemodynamic instability, so the decision was made to cardiovert, and the defibrillator was placed in synch mode. However, the rhythm evolved into ventricular fibrillation, which will not be shocked when the sync button remains on.

**References:**

33. **Rationale**

The rhythm shown is a polymorphic ventricular tachycardia, also known as torsades de pointes (TdP), which may be associated with medications that prolong the QT interval, such as ciprofloxacin. While magnesium is an appropriate treatment for this condition, this patient has clearly lost cardiac output, as indicated by the flattening of the arterial line tracing. To maintain cerebral perfusion, cardiopulmonary resuscitation should be started immediately while magnesium is being obtained. Vasopressin is no longer indicated in cardiac arrest per the 2015 American Heart Association advanced cardiac life support algorithms. Isoproterenol may be used, rarely, in some cases of bradycardia-associated TdP, but this is not the first intervention indicated. Synchronized cardioversion is not appropriate because the patient is clearly not stable, given this tracing.

**Reference:**
34. Rationale

The prognosis of patients who are admitted in an unconscious state after successful cardiopulmonary resuscitation has significantly improved during the past decade, in large part because of aggressive post-resuscitation care, including the use of therapeutic targeted temperature management (TTM). In the early phase of post-resuscitative care, assessment of neurologic outcome remains a challenge.

In patients not treated with TTM, a lack of motor response or extensor response to pain and the absence of brainstem reflexes (including pupillary, corneal, and oculocephalic reactivity) at 72 hours post cardiac arrest are regarded as reliable signs of poor neurologic outcome. However, hypothermia reduces clearance of the sedative and analgesic agents commonly used during TTM. In addition, some patients may have altered renal and hepatic function after cardiac arrest, which may further delay drug clearance. The combined effects of TTM and controlled sedation alters portions of the neurologic examination, making it insufficiently accurate to predict prognosis in the early phase of unconsciousness post cardiac arrest. A recent meta-analysis of studies in patients treated with TTM post cardiac arrest found that a score of 1 to 2 on the motor portion of the Glasgow Coma Scale at 72 hours had a high average false-positive rate of 21%. Brainstem responses had better accuracy, but absent corneal reflexes yielded an average false-positive rate of 2%, while bilaterally absent pupillary reflexes had the lowest false-positive rate (0.4%) to predict poor neurologic outcome. The presence of myoclonus, (distinct from status myoclonus) also has a high false-positive rate (5%) and should not be used to predict poor neurologic outcomes. A multimodal approach, including full neurologic examination, electrophysiologic testing (EEG and somatosensory evoked potentials), neuroimaging, and chemical biomarkers, provides the most reliable information for prognostication of unconscious cardiac arrest survivors.

References:
35. **Rationale**

After submersion in water, victims initially hold their breath, which is then typically followed by laryngospasm, hypoxia and hypercarbia that then cause relaxation of the larynx and allow the aspiration of water. With decreases in body temperature, normal sinus rhythm transforms into sinus bradycardia, which is followed by atrial fibrillation, ventricular fibrillation, and asystole. Ongoing severe acidosis and hypoxemia contribute to instability of myocardial membranes and prevent restoration of normal sinus rhythm.

In this case, venoarterial extracorporeal membrane oxygenation (V-A ECMO) will help to rewarm, provide hemodynamic support in the settings of malignant ventricular arrhythmia and severe acidosis, and oxygenate. In the settings of non-hypothermia-induced out-of-hospital and in-hospital cardiac arrest V-A ECMO is associated with survival rates of 10% to 30%. Rewarming with forced air, IV fluids, warm humidified gases, and gastric and bladder lavage are only effective in hypothermic patients with stable circulation.

Ongoing attempts at defibrillation are unlikely to succeed until the patient is adequately rewarmed (greater than 30°C [86°F]). At 18°C (64.4°F), the brain can tolerate periods of circulatory arrest ten times longer than at 37°C. Dilated pupils can be caused by a variety of insults and must not be regarded as a sign of death. Good neurologic recovery has been reported after cardiac arrest and a core temperature of 13.7°C (56.66°F) after immersion in cold water with prolonged CPR. Therefore, patients should not be considered dead until rewarming efforts have failed.

**References:**
36. Rationale

Amiodarone may be used to treat or prevent ventricular dysrhythmias associated with cardiac arrest, but it has not been shown to improve neurologically intact survival after cardiac arrest.

Targeted temperature management has been shown to provide a significant neurologically intact survival benefit over no temperature control in two randomized trials. A subsequent trial did not demonstrate a difference between active management at 33°C (91.4°F) versus 36°C (96.8°F), and it remains unclear whether some patients benefit from different specific targets.

While lung-protective strategy is generally considered good practice, and post-cardiac arrest patients are at increased risk for lung injury, there has not been an established benefit regarding neurologically intact survival.

While immediate cardiac catheterization is supported by a literature base for patients with primary ventricular dysrhythmias, the established benefit for neurologically intact survival is not as well established as that for targeted temperature management.

References:

37. Rationale

The American Heart Association guidelines for cardiac life support recommend the immediate initiation of chest compressions, rather than airway management, in out-of-hospital cardiac arrest. While airway management and use of an automated external defibrillator are important for resuscitation, chest compressions are the initial step in the algorithm. Early initiation of chest compressions has been shown to improve neurologic outcomes in out-of-hospital cardiac arrest. Nalaxone administration may be beneficial in a patient who is suspected of being unresponsive due to an opioid overdose. If opioid overdose is expected in a pulseless patient, the initial step is still chest compressions.
References:

38. Rationale

Answer: C

Synchronized cardioversion is appropriate at this time since he is symptomatic, is hypotensive, and has an organized rhythm. Metoprolol and verapamil administration will worsen his hypotension. There is no suggestion of a narcotic overdose that would be treated with naloxone.

References:

39. Rationale

Answer: D

This patient has severe mitral stenosis complicated by acute fever, tachycardia, and hypotension. With a valve area of $0.88 \text{ cm}^2$, coupled with his known transvalvular gradient, the necessary time for adequate ventricular filling in diastole is longer than if the mitral stenosis were not present. Consequently, tachycardia and loss of atrial contraction can be poorly tolerated. At the time of ICU transfer the cause of his hypotension is not known. Diuretics and beta-adrenergic antagonists may be appropriate for decompensated severe mitral stenosis and evidence of pulmonary congestion, but in the setting of hypotension of unknown etiology, these drugs are not warranted. IV epinephrine is not the best first choice when the cause of hypotension and the patient’s underlying hemodynamic state is unknown. The best initial management of this patient’s hypotension is a judicious fluid bolus while additional imaging and monitoring is obtained to determine more definitive therapy.

References:
Blood supply to the thoracic spinal cord is largely dependent on a single anterior spinal artery and two posterolateral spinal arteries traveling cephalad to caudal. These arteries are largely dependent on blood supply from the anterior and posterior radicular arteries and the segmental medullary arteries. These originate from the segmental (intercostal) arteries, which arise from the thoracic aorta (see figure shown below). The largest of the segmental medullary arteries is the artery of Adamkiewicz, originating from the lower thoracic aorta (T8-L1) in most people.

Interruption of blood flow through the intercostal arteries can occur as a result of endovascular graft deployment in the aorta. Patients at higher risk for spinal cord ischemia postoperatively include those with: 1) decrease/disruption of collateral circulation due to prior abdominal aortic aneurysm repair, severe atherosclerosis of the thoracic aorta resulting in decreased collateral circulation, or more extensive graft coverage of thoracic aorta, 2) hypotension resulting in decreased spinal cord perfusion pressure, or 3) increased spinal fluid pressure or central venous pressure.

Spinal cord perfusion pressure is calculated as mean arterial pressure minus cerebrospinal fluid pressure (or central venous pressure). Spinal drains are often placed before thoracic endograft procedures to reduce the resistance to blood flow, thereby encouraging spinal cord perfusion.

Maintaining spinal cord perfusion is especially critical in the early postoperative period, when the spinal cord may be recovering from intraoperative ischemic insult. Decreased neurologic function after aortic repair is a neurologic emergency; taking action to improve spinal cord perfusion is essential to minimize long-term sequelae. The spinal drain can be opened or repositioned to increase drainage of cerebrospinal fluid to improve spinal cord perfusion. Increasing blood pressure will also increase spinal cord perfusion.
References:

41. Rationale

Acute aortic insufficiency is a medical emergency requiring prompt surgical intervention. However, the diagnosis is often challenging to make, leading to delays in management. As is the case here, presentations mimicking pneumonia or nonvalvular heart failure may complicate the picture, delaying definitive therapy. Given the diastolic murmur, evidence of mixed cardiogenic and septic shock, needle track marks, and blood cultures positive for methicillin-resistant *Staphylococcus aureus* (MRSA), the most likely diagnosis is infective endocarditis causing acute aortic regurgitation.

Treatment should be directed at broadening antibiotic coverage to include MRSA, obtaining an immediate echocardiography to confirm the diagnosis, and involving cardiac surgery for definitive management. Instituting appropriate medical therapy, including vasopressors, inotropes, and intubation, is
necessary, but is not a substitute for surgical therapy in such a critically ill state. Providing additional IV fluid boluses is unlikely to improve hypotension, and may worsen oxygenation given evidence of cardiogenic shock. Placing an intra-aortic balloon pump is contraindicated here due to aortic insufficiency and worsening of the regurgitation when the balloon inflates during diastole. Delaying surgical repair for days until blood cultures return negative is not optimal management in this setting.

References:

42. Rationale

Acute aortic insufficiency, either perivalvular or central, is a known complication of transcatheter aortic valve replacement (TAVR), with an incidence reported as high as 70%. Acute aortic regurgitation results in sudden volume overload to the left ventricle (LV) during systole. As a result of this, LV end-systolic volume is higher than normal and the pressure gradient between the left atrium and the LV decreases earlier in subsequent diastole. This results in early mitral valve closure, which is audible as a soft S1 sound. Early systolic and even diastolic mitral regurgitation have been observed due to changes in ventriculo-arterial gradient in the LV. This leads to a reduction in LV stroke volume and a reflex tachycardia. As a result, patients often have thready pulses and cold extremities.

Pericardial tamponade has been observed after TAVR, but is rare. In addition, the patient’s jugular venous pulsation is reported to be 5 cm above the sternal angle with slow refill, which is inconsistent with tamponade. Sepsis is associated with tachycardia and hypotension, but usually not with cold extremities and a normal temperature. Acute third-degree heart block has been reported after TAVR, but is not usually associated with a rapid heart rate. Complete loss of atrioventricular conduction can cause decreased cardiac output and hypotension, but usually the cause of diminished cardiac output is bradycardia, not decreased stroke volume. In addition a diastolic murmur is usually not observed. Ascending aortic dissection is a rare but known complication, certainly rarer than aortic insufficiency. However, this should be accompanied by coronary ischemia. A typical murmur associated with acute mitral regurgitation is a high-pitched, blowing holosystolic murmur.

References:
43. **Rationale**

This patient has a malignant pericardial effusion that has led to tamponade physiology and obstructive shock. The most prudent course of action when first examining him would be to perform a rapid ultrasound assessment for shock and hypotension (RUSH) examination and provide an IV fluid bolus once the effusion is seen. At that point, a decision would need to be made to drain the effusion at the bedside or in the catheterization laboratory, depending on his clinical status. Bedside ultrasound for acute dyspnea and shock is a critical tool for intensivists. This will help rapidly narrow the differential diagnosis and improve decision making.

Intubating the patient may eventually be necessary but, given his mental status, adequate oxygenation, and profound hypotension, this may not be the first action to take. Septic shock certainly remains in the differential diagnosis, and providing antibiotics and obtaining cultures may still be advisable even with tamponade findings on ultrasound. Based on history and examination, it is less likely that this presentation is acute heart failure or pulmonary embolism, and bedside cardiac ultrasound will also help to prove this. A significant retroperitoneal bleed may be the cause of his hypotension, but history and examination are relatively inconsistent with this, and protamine is not a pure reversal agent for enoxaparin, but it may help.

**References:**


44. **Rationale**

Although a rare and challenging diagnosis to make, this patient has hypersensitivity eosinophilic myocarditis and is in fulminant heart failure. The offending agent in this case is phenytoin, which the patient has taken for seizures for many years. Although not always present, peripheral eosinophilia can be a clue, in addition to the faint rash noted on the trunk. Due to the high mortality associated with this disease, early diagnosis is key, and an endomyocardial biopsy is the test of choice. Once the diagnosis is confirmed, in addition to withdrawal of the offending drug, immunosuppressive therapy can commence, often with dramatic results.

A right upper quadrant ultrasound would be prudent, given the elevated liver function tests, but this is most likely due to congestive hepatopathy, and would not yield a diagnosis. Similarly, a skin punch biopsy and blood cultures would probably be performed on this patient to look for autoimmune and infectious causes, but an endomyocardial biopsy would still be needed. A chest CT would be a low-yield test, and may even cause harm, given his acute kidney injury.

**References:**
