

SECOND EDITION

SURGICAL CRITICAL CARE and EMERGENCY SURGERY

**CLINICAL QUESTIONS
AND ANSWERS**

EDITED BY
**FORREST "DELL" MOORE
PETER RHEE
GERARD J. FULDA**



WILEY Blackwell

**Surgical Critical Care and Emergency Surgery:
Clinical Questions and Answers**

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Clinical Questions and Answers

Second Edition

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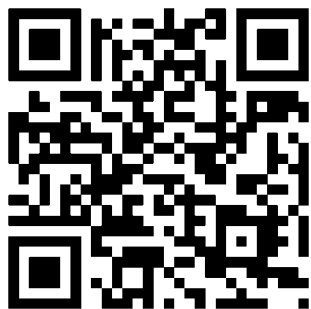
About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/moore/surgical_criticalcare_and_emergency_surgery

The website features:

- MCQs



Part One

Surgical Critical Care

1

Respiratory and Cardiovascular Physiology

Marcin Jankowski, DO and Frederick Giberson, MD

- 1 All of the following are mechanisms by which vasodilators improve cardiac function in acute decompensated left heart failure except:
- A Increase stroke volume
 - B Decrease ventricular filling pressure
 - C Increase ventricular preload
 - D Decrease end-diastolic volume
 - E Decrease ventricular afterload

Most patients with acute heart failure present with increased left-ventricular filling pressure, high systemic vascular resistance, high or normal blood pressure, and low cardiac output. These physiologic changes increase myocardial oxygen demand and decrease the pressure gradient for myocardial perfusion resulting in ischemia. Therapy with vasodilators in the acute setting can often improve hemodynamics and symptoms.

Nitroglycerine is a powerful venodilator with mild vasodilatory effects. It relieves pulmonary congestion through direct venodilation, reducing left and right ventricular filling pressures, systemic vascular resistance, wall stress, and myocardial oxygen consumption. Cardiac output usually increases due to decreased LV wall stress, decreased afterload, and improvement in myocardial ischemia. The development of “tachyphylaxis” or tolerance within 16–24 hours of starting the infusion is a potential drawback of nitroglycerine.

Nitroprusside is an equal arteriolar and venous tone reducer, lowering both systemic and vascular resistance and left and right filling pressures. Its effects on reducing afterload increase stroke volume in heart failure. Potential complications of nitroprusside include cyanide toxicity and the risk of “coronary steal syndrome.”

In patients with acute heart failure, therapeutic reduction of left-ventricular filling pressure with any of the above agents correlates with improved outcome.

Increased ventricular preload would increase the filling pressure, causing further increases in wall stress and myocardial oxygen consumption, leading to ischemia.

Answer: C

Marino, P. (2014) *The ICU Book*, 4th edn, Lippincott Williams & Wilkins, Philadelphia, PA, chapter 13.

Mehra, M.R. (2015) Heart failure: management, in *Harrison's Principles of Internal Medicine*, 19th edn (eds D. Kasper, A. Fauci, S. Hauser, *et al.*), McGraw-Hill, New York.

- 2 Which factor is most influential in optimizing the rate of volume resuscitation through venous access catheters?
- A Laminar flow
 - B Length
 - C Viscosity
 - D Radius
 - E Pressure gradient

The forces that determine flow are derived from observations on ideal hydraulic circuits that are rigid and the flow is steady and laminar. The Hagen-Poiseuille equation states that flow is determined by the fourth power of the inner radius of the tube ($Q = \Delta p \pi r^4 / 8 \mu L$), where P is pressure, μ is viscosity, L is length, and r is radius. This means that a two-fold increase in the radius of a catheter will result in a sixteen-fold increase in flow. As the equation states, the remaining components of resistance, such as pressure difference along the length of the tube and fluid viscosity, are inversely related and exert a much smaller influence on flow. Therefore, cannulation of large central veins with long catheters are much less effective than cannulation of peripheral veins with a short catheter. This illustrates that it is the size of the catheter and not the vein that determines the rate of volume infusion (see Figure 1.1).

Answer: D

Marino, P. (2014) *The ICU Book*, 4th edn, Lippincott Williams & Wilkins, Philadelphia, PA, chapter 12.

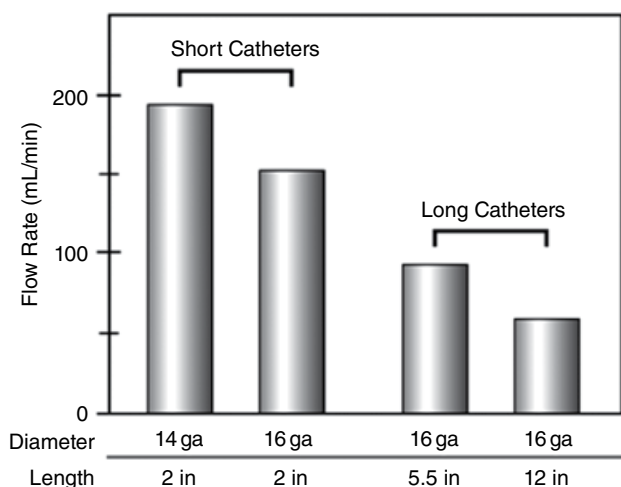


Figure 1.1 The influence of catheter dimensions on the gravity-driven infusion of water.

- 3 Choose the correct physiologic process represented by each of the cardiac pressure-volume loops in Figure 1.2.
- A 1) Increased preload, increased stroke volume,
2) Increased afterload, decreased stroke volume
- B 1) Decreased preload, increased stroke volume,
2) Decreased afterload, increased stroke volume
- C 1) Increased preload, decreased stroke volume,
2) Decreased afterload, increased stroke volume
- D 1) Decreased preload, decreased stroke volume,
2) Increased afterload, decreased stroke volume
- E 1) Decreased preload, increased stroke volume,
2) Increased afterload, decreased stroke volume

One of the most important factors in determining stroke volume is the extent of cardiac filling during diastole or the end-diastolic volume. This concept is known as the Frank–Starling law of the heart. This law states that, with all other factors equal, the stroke volume will increase as

the end-diastolic volume increases. In Figure 1.2A, the ventricular preload or end-diastolic volume (LV volume) is increased, which ultimately increases stroke volume defined by the area under the curve. Notice the LV pressure is not affected. Increased afterload, at constant preload, will have a negative impact on stroke volume. In Figure 1.2B, the ventricular afterload (LV pressure) is increased, which results in a decreased stroke volume, again defined by the area under the curve.

Answer: A

Mohrman, D. and Heller, L. (2014) *Cardiovascular Physiology*, 8th edn, McGraw-Hill, New York, chapter 3.

- 4 A 68-year-old patient is admitted to the SICU following a prolonged exploratory laparotomy and extensive lysis of adhesions for a small bowel obstruction. The patient is currently tachycardic and hypotensive. Identify the most effective way of promoting end-organ perfusion in this patient.
- A Increase arterial pressure (total peripheral resistance) with vasoactive agents
- B Decrease sympathetic drive with heavy sedation
- C Increase end-diastolic volume with controlled volume resuscitation
- D Increase contractility with a positive inotropic agent
- E Increase end-systolic volume

This patient is presumed to be in hypovolemic shock as a result of a prolonged operative procedure with inadequate perioperative fluid resuscitation. The insensible losses of an open abdomen for several hours in addition to significant fluid shifts due to the small bowel obstruction can significantly lower intravascular volume. The low urine output is another clue that this patient would benefit from controlled volume resuscitation.

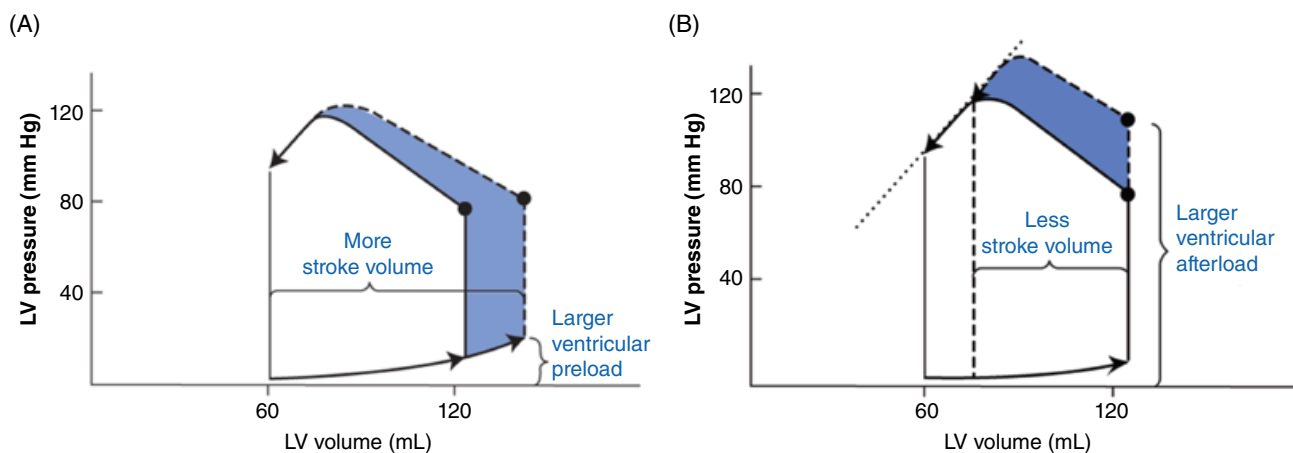


Figure 1.2

Starting a vasopressor such as norepinephrine would increase the blood pressure but the effects of increased afterload on the heart and the peripheral vasoconstriction leading to ischemia would be detrimental in this patient. Lowering the sympathetic drive with increased sedation will lead to severe hypotension and worsening shock. Increasing contractility with an inotrope in a hypovolemic patient would add great stress to the heart and still provide inadequate perfusion as a result of low preload. An increase in end-systolic volume would indicate a decreased stroke volume and lower cardiac output and would not promote end-organ perfusion.

$$\text{CO} = \text{HR} \times \text{SV}$$

$$\text{SV} = \text{EDV} - \text{ESV}$$

According to the principle of continuity, the stroke output of the heart is the main determinant of circulatory blood flow. The forces that directly affect the flow are preload, afterload and contractility. According to the Frank–Starling principle, in the normal heart diastolic volume is the principal force that governs the strength of ventricular contraction. This promotes adequate cardiac output and good end-organ perfusion.

Answer: C

Levick, J.R. (2013) *An Introduction to Cardiovascular Physiology*, Butterworth and Co. London.

- 5 Which physiologic process is least likely to increase myocardial oxygen consumption?
- A Increasing inotropic support
 - B A 100% increase in heart rate
 - C Increasing afterload
 - D 100% increase in end-diastolic volume
 - E Increasing blood pressure

Myocardial oxygen consumption (MVO_2) is primarily determined by myocyte contraction. Therefore, factors that increase tension generated by the myocytes, the rate of tension development and the number of cycles per unit time will ultimately increase myocardial oxygen consumption. According to the Law of LaPlace, cardiac wall tension is proportional to the product of intraventricular pressure and the ventricular radius.

Since the MVO_2 is closely related to wall tension, any changes that generate greater intraventricular pressure from increased afterload or inotropic stimulation will result in increased oxygen consumption. Increasing inotropy will result in increased MVO_2 due to the increased rate of tension and the increased magnitude of the tension. Doubling the heart rate will approximately double the MVO_2 due to twice the number of tension cycles per minute. Increased afterload will increase

MVO_2 due to increased wall tension. Increased preload or end-diastolic volume does not affect MVO_2 to the same extent. This is because preload is often expressed as ventricular end-diastolic volume and is not directly based on the radius. If we assume the ventricle is a sphere, then:

$$V = \frac{4}{3} \pi \cdot r^3$$

Therefore

$$r \propto \sqrt[3]{V}$$

Substituting this relationship into the Law of LaPlace

$$T \propto P \cdot \sqrt[3]{V}$$

This relationship illustrates that a 100% increase in ventricular volume will result in only a 26% increase in wall tension. In contrast, a 100% increase in ventricular pressure will result in a 100% increase in wall tension. For this reason, wall tension, and therefore MVO_2 , is far less sensitive to changes in ventricular volume than pressure.

Answer: D

Klabunde, R.E. (2011) *Cardiovascular Physiology Concepts*, 2nd edn. Lippincott, Williams & Wilkins, Philadelphia, PA.
 Rhoades, R. and Bell, D.R. (2012) *Medical Physiology: Principles for Clinical Medicine*, 4th edn, Lippincott, Williams & Wilkins, Philadelphia, PA.

- 6 A 73-year-old obese man with a past medical history significant for diabetes, hypertension, and peripheral vascular disease undergoes an elective right hemicolectomy. While in the PACU, the patient becomes acutely hypotensive and lethargic requiring immediate intubation. What effects do you expect positive pressure ventilation to have on your patient's cardiac function?
- A Increased pleural pressure, increased transmural pressure, increased ventricular afterload
 - B Decreased pleural pressure, increased transmural pressure, increased ventricular afterload
 - C Decreased pleural pressure, decreased transmural pressure, decreased ventricular afterload
 - D Increased pleural pressure, decreased transmural pressure, decreased ventricular afterload
 - E Increased pleural pressure, increased transmural pressure, decreased ventricular afterload

This patient has a significant medical history that puts him at high risk of an acute coronary event. Hypotension and decreased mental status clearly indicate the need for immediate intubation. The effects of positive pressure ventilation will have direct effects on this patient's

cardiovascular function. Ventricular afterload is a transmural force so it is directly affected by the pleural pressure on the outer surface of the heart. Positive pleural pressures will enhance ventricular emptying by promoting the inward movement of the ventricular wall during systole. In addition, the increased pleural pressure will decrease transmural pressure and decrease ventricular afterload. In this case, the positive pressure ventilation provides cardiac support by “unloading” the left ventricle resulting in increased stroke volume, cardiac output and ultimately better end-organ perfusion.

Answer: D

Cairo, J.M. (2016) Extrapulmonary effects of mechanical ventilation, in *Pilbeam's Mechanical Ventilation. Physiological and Clinical Applications*, 6th edn, Elsevier, St. Louis, MO, pp. 304–314

- 7 Following surgical debridement for lower extremity necrotizing fasciitis, a 47-year-old man is admitted to the ICU. A Swan-Ganz catheter was inserted for refractory hypotension. The initial values are CVP = 5 mm Hg, MAP = 50 mm Hg, PCWP = 8 mm Hg, PaO₂ = 60 mm Hg, CO = 4.5 L/min, SVR = 450 dynes·sec/cm⁵, and O₂ saturation of 93%. The hemoglobin is 8 g/dL. The most effective intervention to maximize perfusion pressure and oxygen delivery would be which of the following?
- A Titrate the FiO₂ to a SaO₂ > 98%
 - B Transfuse with two units of packed red blood cells
 - C Fluid bolus with 1 L normal saline
 - D Titrate the FiO₂ to a PaO₂ > 80
 - E Start a vasopressor

To maximize the oxygen delivery (DO₂) and perfusion pressure to the vital organs, it is important to determine the factors that directly affect it. According to the formula below, oxygen delivery (DO₂) is dependent on cardiac output (Q), the hemoglobin level (Hb), and the O₂ saturation (SaO₂):

$$DO_2 = Q \times (1.34 \times Hb \times SaO_2 \times 10) + (0.003 \times PaO_2)$$

This patient is likely septic from his infectious process. In addition, the long operation likely included a significant blood loss and fluid shifts so hypovolemic/hemorrhagic shock is likely contributing to this patient's hypotension. The low CVP, low wedge pressure indicates a need for volume replacement. The fact that this patient is anemic as a result of significant blood loss means that transfusing this patient would likely benefit his oxygen-carrying capacity as well as provide volume replacement. Fluid bolus is not inappropriate; however, two units of

packed red blood cells would be more appropriate. Titrating the PaO₂ would not add any benefit because, according to the above equation, it contributes very little to the overall oxygen delivery. Starting a vasopressor in a hypovolemic patient is inappropriate at this time and should be reserved for continued hypotension after adequate fluid resuscitation. Titrating the FiO₂ to a saturation of greater than 98% would not be clinically relevant. Although the patient requires better oxygen-carrying capacity, this would be better solved with red blood cell replacement.

Answer: B

Marino, P. (2014) *The ICU Book*, 4th edn, Lippincott Williams & Wilkins, Philadelphia, PA, chapter 2.

- 8 To promote adequate alveolar ventilation, decrease shunting, and ultimately improve oxygenation, the addition of positive end-expiratory pressure (PEEP) in a severely hypoxic patient with ARDS will:
- A Limit the increase in residual volume (RV)
 - B Limit the decrease in expiratory reserve volume (ERV)
 - C Limit the increase in inspiratory reserve volume (IRV)
 - D Limit the decrease in tidal volume (TV)
 - E Increase pCO₂

Patients with ARDS have a significantly decreased lung compliance, which leads to significant alveolar collapse. This results in decreased surface area for adequate gas exchange and an increased alveolar shunt fraction resulting in hypoventilation and refractory hypoxemia. The minimum volume and pressure of gas necessary to prevent small airway collapse is the critical closing volume (CCV). When CCV exceeds functional residual capacity (FRC), alveolar collapse occurs. The two components of FRC are residual volume (RV) and expiratory reserve volume (ERV).

The role of extrinsic positive end-expiratory pressure (PEEP) in ARDS is to prevent alveolar collapse, promote further alveolar recruitment, and improve oxygenation by limiting the decrease in FRC and maintaining it above the critical closing volume. Therefore, limiting the decrease in ERV will limit the decrease in FRC and keep it above the CCV thus preventing alveolar collapse.

Limiting an increase in the residual volume would keep the FRC below the CCV and promote alveolar collapse. Positive-end expiratory pressure has no effect on inspiratory reserve volume (IRV) or tidal volume (TV) and does not increase pCO₂.

Answer: B

Rimensberger, P.C. and Bryan, A.C. (1999) Measurement of functional residual capacity in the critically ill. Relevance for the assessment of respiratory mechanics during mechanical ventilation. *Intensive Care Medicine*, 25 (5), 540–542.

Sidebotham, D., McKee, A., Gillham, M., and Levy, J. (2007) *Cardiothoracic Critical Care*, Butterworth-Heinemann, Philadelphia, PA.

9 Which of the five mechanical events of the cardiac cycle is described by an initial contraction, increasing ventricular pressure and closing of the AV valves?

- A Ventricular diastole
- B Atrial systole
- C Isovolumic ventricular contraction
- D Ventricular ejection (systole)
- E Isovolumic relaxation

The repetitive cellular electrical events resulting in mechanical motions of the heart occur with each beat and make up the cardiac cycle. The mechanical events of

the cardiac cycle correlate with ECG waves and occur in five phases described in Figure 1.3.

- 1) Ventricular diastole (mid-diastole): Throughout most of ventricular diastole, the atria and ventricles are relaxed. The AV valves are open, and the ventricles fill passively.
- 2) Atrial systole: During atrial systole a small amount of additional blood is pumped into the ventricles.
- 3) Isovolumic ventricular contraction: Initial contraction increases ventricular pressure, closing the AV valves. Blood is pressurized during isovolumic ventricular contraction.
- 4) Ventricular ejection (systole): The semilunar valves open when ventricular pressures exceed pressures in the aorta and pulmonary artery. Ventricular ejection (systole) of blood follows.
- 5) Isovolumic relaxation: The semilunar valves close when the ventricles relax and pressure in the ventricles decreases. The AV valves open when pressure in the ventricles decreases below atrial pressure.

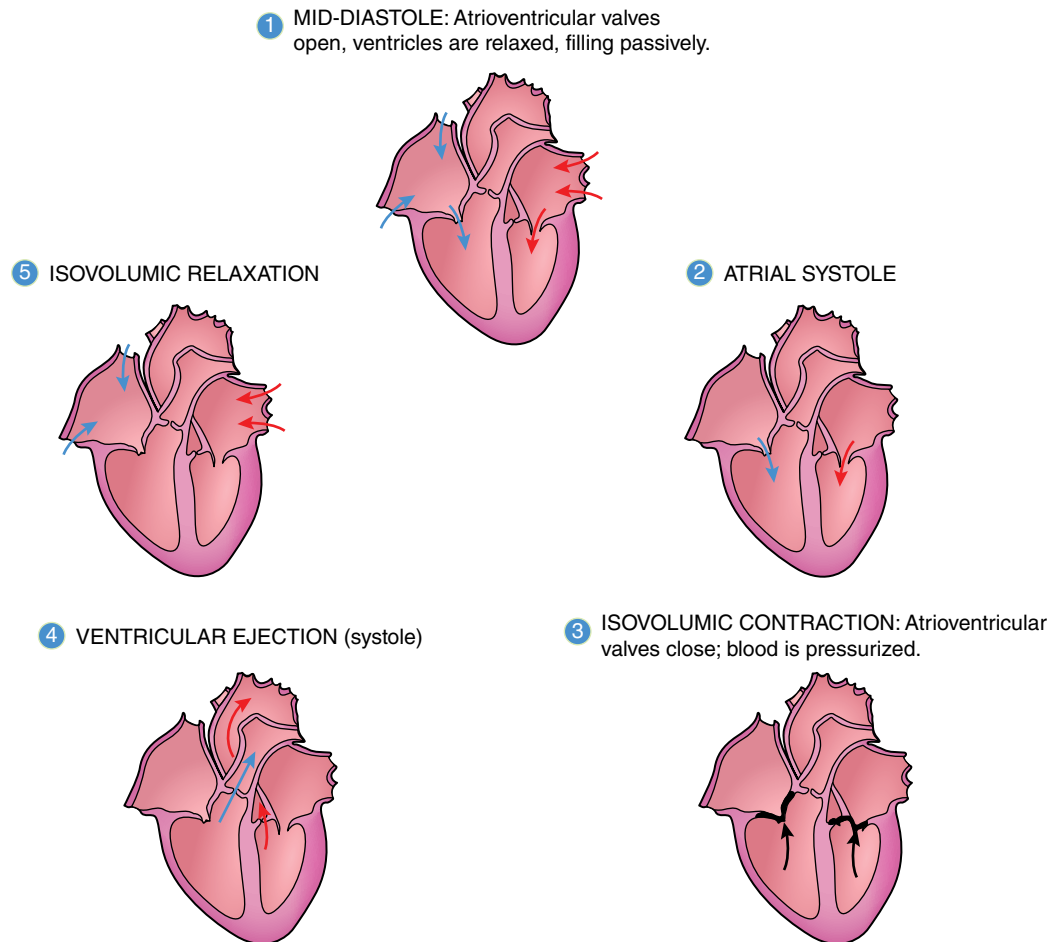


Figure 1.3 The cardiac cycle illustrated.

Atria fill with blood throughout ventricular systole, allowing rapid ventricular filling at the start of the next diastolic period.

Answer: C

- Kibble, J.D. and Halsey, C.R. (2015) Cardiovascular physiology, in *Medical Physiology: The Big Picture*, McGraw-Hill, New York, pp. 131–174.
- Barrett, K.E., Barman, S.M., Boitano, S., and Brooks, H.L. (2016) The heart as a pump, in *Ganong's Review of Medical Physiology* (K. E. Barrett, S.M. Barman, S, Boitano, and H.L. Brooks, eds), 25th edn, McGraw-Hill, New York, pp. 537–553.
- 10 *A recent post-op 78-year-old man is admitted to the STICU with an acute myocardial infarction and resulting severe hypotension. A STAT ECHO shows decompensating right-sided heart failure. CVP = 23 cmH₂O. What is the most appropriate therapeutic intervention at this time?*
- A Volume
 - B Vasodilator therapy
 - C Furosemide
 - D Inodilator therapy
 - E Mechanical cardiac support

The mainstay therapy of right-sided heart failure associated with severe hypotension as a result of an acute myocardial infarction is volume infusion. However, it is important to carefully monitor the CVP or PAWP in order to avoid worsening right heart failure resulting in left-sided heart failure as a result of interventricular interdependence. A mechanism where right-sided volume overload leads to septal deviation and compromised left ventricular filling. An elevated CVP or PAWP of > 15 should be utilized as an endpoint of volume infusion in right heart failure. At this point, inodilator therapy with dobutamine or levosimendan should be initiated. Additional volume infusion would only lead to further hemodynamic instability and potential collapse. Vasodilator therapy should only be used in normotensive heart failure due to its risk for hypotension. Diuretics should only be used in normo- or hypertensive heart failure patients. Mechanical cardiac support should only be initiated in patients who are in cardiogenic shock due to left-sided heart failure.

Acute decompensated heart failure (ADHF) can present in many different ways and require different therapeutic strategies. This patient represents the “low output” phenotype that is often associated with hypoperfusion and end-organ dysfunction. See Figure 1.4.

Answer: D

Mehra, M.R. (2015) Heart failure: management, in *Harrison's Principles of Internal Medicine*, 19th edn (D. Kasper, A. Fauci, S. Hauser, *et al.*, eds), McGraw-Hill, New York, chapter 280.

- 11 *The right atrial tracing in Figure 1.5 is consistent with:*
- A Tricuspid stenosis
 - B Normal right atrial waveform tracing
 - C Tricuspid regurgitation
 - D Constrictive pericarditis
 - E Mitral stenosis

The normal jugular venous pulse contains three positive waves (Figure 1.6). These positive deflections, labeled “a,” “c,” and “v” occur, respectively, before the carotid upstroke and just after the P wave of the ECG (a wave); simultaneous with the upstroke of the carotid pulse (c wave); and during ventricular systole until the tricuspid valve opens (v wave). The “a” wave is generated by atrial contraction, which actively fills the right ventricle in end-diastole. The “c” wave is caused either by transmission of the carotid arterial impulse through the external and internal jugular veins or by the bulging of the tricuspid valve into the right atrium in early systole. The “v” wave reflects the passive increase in pressure and volume of the right atrium as it fills in late systole and early diastole.

Normally the crests of the “a” and “v” waves are approximately equal in amplitude. The descents or troughs of the jugular venous pulse occur between the “a” and “c” wave (“x” descent), between the “c” and “v” wave (“x” descent), and between the “v” and “a” wave (“y” descent). The x and x’ descents reflect movement of the lower portion of the right atrium toward the right ventricle during the final phases of ventricular systole. The y descent represents the abrupt termination of the downstroke of the v wave during early diastole after the tricuspid valve opens and the right ventricle begins to fill passively. Normally the y descent is neither as brisk nor as deep as the x descent.

Answer: C

- Hall, J.B., Schmidt, G.A., and Wood, L.D.H. (eds) (2005) *Principles of Critical Care*, 3rd edn, McGraw-Hill, New York.
- McGee, S. (2007) *Evidence-based Physical Diagnosis*, 2nd edn, W. B. Saunders & Co., Philadelphia, PA.
- Pinsky, L.E. and Wipf, J.E. (n.d.) University of Washington Department of Medicine. *Advanced Physical Diagnosis. Learning and Teaching at the Bedside*. Edition 1, <http://depts.washington.edu/physdx/neck/index.html> (accessed November 6, 2011).

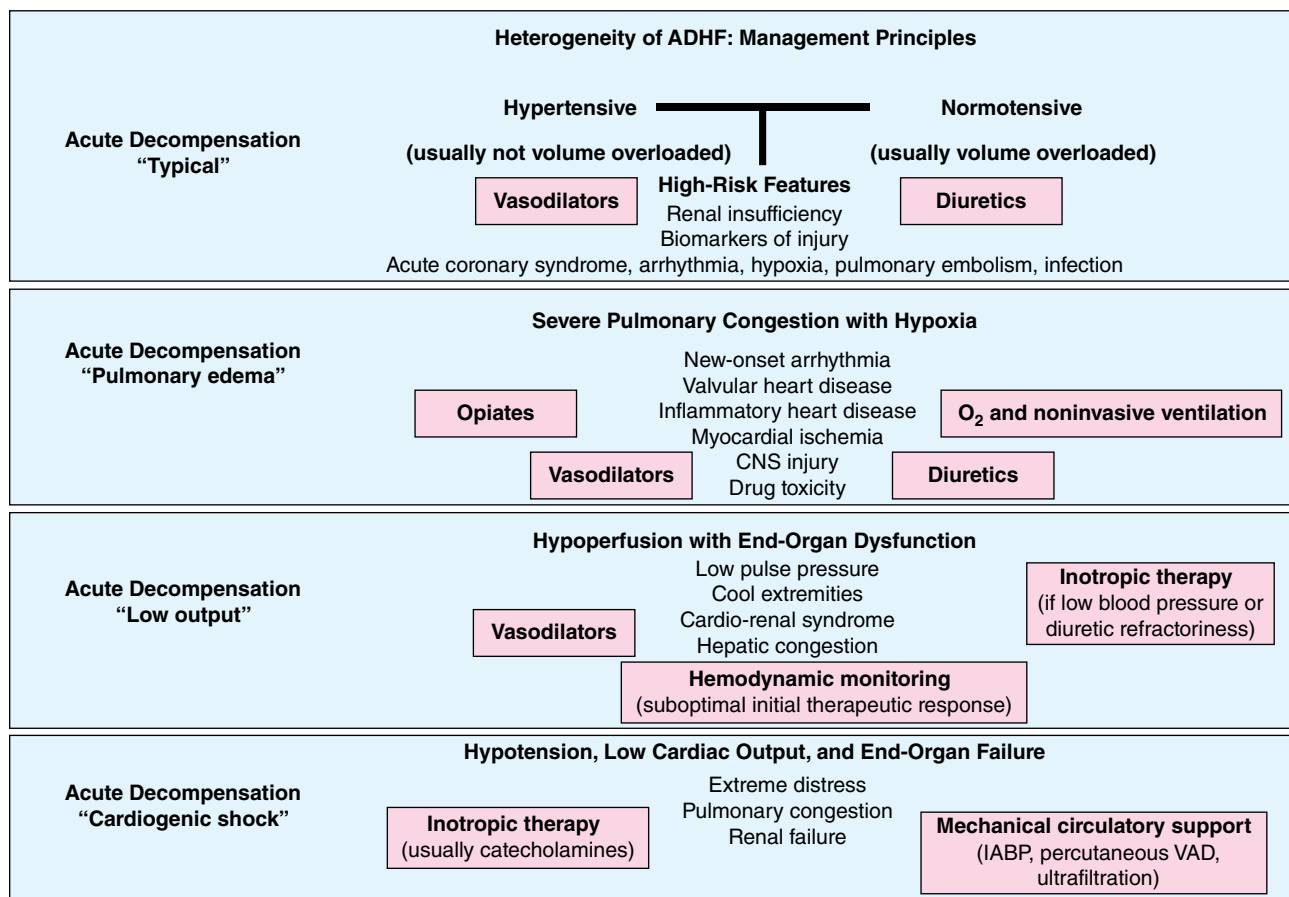


Figure 1.4

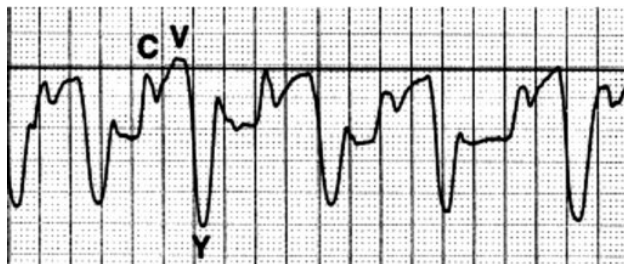


Figure 1.5

- 12 The addition of PEEP in optimizing ventilatory support in patients with ARDS does all of the following except:
- Increases functional residual capacity (FRC) above the alveolar closing pressure
 - Maximizes inspiratory alveolar recruitment
 - Limits ventilation below the lower inflection point to minimize shear-force injury
 - Improves V/Q mismatch
 - Increases the mean airway pressure

The addition of positive-end expiratory pressure (PEEP) in patients who have ARDS has been shown to be beneficial.

By maintaining a small positive pressure at the end of expiration, considerable improvement in the arterial PaO₂ can be obtained. The addition of PEEP maintains the functional residual capacity (FRC) above the critical closing volume (CCV) of the alveoli, thus preventing alveolar collapse. It also limits ventilation below the lower inflection point minimizing shear force injury to the alveoli. The prevention of alveolar collapse results in improved V/Q mismatch, decreased shunting, and improved gas exchange. The addition of PEEP in ARDS also allows for lower FiO₂ to be used in maintaining adequate oxygenation.

PEEP maximizes the expiratory alveolar recruitment; it has no effect on the inspiratory portion of ventilatory support.

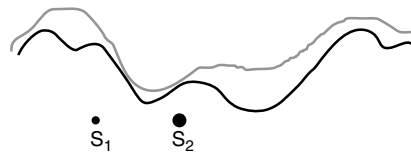
Answer: B

Gattinoni, L., Cairon, M., Cressoni, M., *et al.* (2006) Lung recruitment in patients with acute respiratory distress syndrome. *New England Journal of Medicine* **354**, 1775–1786.

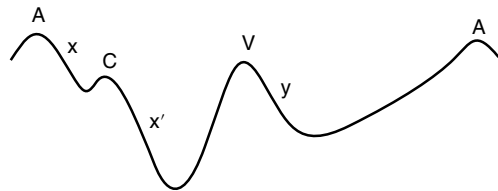
West, B. (2008) *Pulmonary Pathophysiology – The Essentials*, 8th edn, Lippincott, Williams & Wilkins, Philadelphia, PA.

(A) Tricuspid stenosis.

large a wave with impaired y descent
 normal

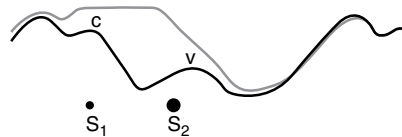


(B) Normal jugular venous tracing.



(C) Tricuspid regurgitation.

CV merger
 normal



(D) Constrictive pericarditis

striking y descent
 normal

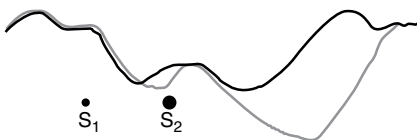


Figure 1.6

- 13 A 70-year-old man with a history of diabetes, hypertension, coronary artery disease, asthma and long-standing cigarette smoking undergoes an emergency laparotomy and Graham patch for a perforated duodenal ulcer. Following the procedure, he develops acute respiratory distress and oxygen saturation of 88%. Blood gas analysis reveals the following:
- $pH = 7.43$
 $paO_2 = 55 \text{ mm Hg}$
 $HCO_3 = 23 \text{ mmol/L}$
 $pCO_2 = 35 \text{ mm Hg}$

Based on the above results, you would calculate his A-a gradient to be (assuming atmospheric pressure at sea level, water vapor pressure = 47 mm Hg):

- A 8 mm Hg
 B 15 mm Hg

- C 30 mm Hg
 D 51 mm Hg
 E 61 mm Hg

The A-a gradient is equal to $PAO_2 - PaO_2$ (55 from ABG). The PAO_2 can be calculated using the following equation:

$$\begin{aligned}
 PaO_2 &= FiO_2 (P_B - P_{H_2O}) - (PaCO_2 / RQ) \\
 &= 0.21 (760 - 47) - (35 / 0.8) \\
 PaO_2 &= 106 \text{ mm Hg}
 \end{aligned}$$

Therefore, A-a gradient ($PaO_2 - PAO_2$) = 51 mm Hg.

Answer: D

Marino, P. (2007) *The ICU Book*, 3rd edn, Lippincott Williams & Wilkins, Philadelphia, PA, chapter 19.

- 14 What is the most likely etiology of the patient in question 13's respiratory failure and the appropriate intervention?
- A Pulmonary edema, cardiac workup
 B Neuromuscular weakness, intubation, and reversal of anesthetic
 C Pulmonary embolism, systemic anticoagulation
 D Acute asthma exacerbation, bronchodilators
 E Hypoventilation, pain control

Disorders that cause hypoxemia can be categorized into four groups: hypoventilation, low inspired oxygen, shunting, and V/Q mismatch. Although all of these can potentially present with hypoxemia, calculating the alveolar-arterial (A-a) gradient and determining whether administering 100% oxygen is of benefit, can often determine the specific type of hypoxemia and lead to quick and effective treatment.

Acute hypoventilation often presents with an elevated $PaCO_2$ and a normal A-a gradient. This is usually seen in patients with altered mental status due to excessive sedation, narcotic use, or residual anesthesia. Since this patient's $PaCO_2$ is low (35 mm Hg), it is not the cause of this patient's hypoxemia.

Low inspired oxygen presents with a low PO_2 and a normal A-a gradient. Since this patient's A-a gradient is elevated, this is unlikely the cause of the hypoxemia.

A V/Q mismatch (pulmonary embolism or acute asthma exacerbation) presents with a normal $PaCO_2$ and an elevated A-a gradient that does correct with administration of 100% oxygen. Since this patient's hypoxemia does not improve after being placed on the non-rebreather mask, it is unlikely that this is the cause.

Shunting (pulmonary edema) presents with a normal $PaCO_2$ and an elevated A-a gradient that does *not* correct

with the administration of 100% oxygen. This patient has a normal PaCO₂, an elevated A-a gradient and hypoxemia that does not correct with the administration of 100% oxygen. This patient has a pulmonary shunt.

Although an A-a gradient can vary with age and the concentration of inspired oxygen, an A-a gradient of 51 is clearly elevated. This patient has a normal PaCO₂ and an elevated A-a gradient that did not improve with 100% oxygen administration therefore a shunt is clearly present. Common causes of shunting include pulmonary edema and pneumonia.

Reviewing this patient's many risk factors for a post-operative myocardial infarction and a decreased left ventricular function makes pulmonary edema the most likely explanation.

Answer: A

Weinberger, S.E., Cockrill, B.A., and Mande, J. (2008) *Principles of Pulmonary Medicine*, 5th edn. W.B. Saunders, Philadelphia, PA.

- 15 You are taking care of a morbidly obese patient on a ventilator who is hypotensive and hypoxic. His peak airway pressures and plateau pressures have been slowly rising over the last few days. You decide to place an esophageal balloon catheter. The values are obtained:

$$P_{plat} = 45 \text{ cm H}_2\text{O}$$

$$\Delta tP = 15 \text{ cm H}_2\text{O}$$

$$\Delta P_{es} = 5 \text{ cm H}_2\text{O}$$

What is the likely cause of the increased peak airway pressures and what is your next intervention?

- A Decreased lung compliance, increase PEEP to 25 cm H₂O
- B Decreased lung compliance, high frequency oscillator ventilation
- C Decreased chest wall compliance, increase PEEP to 25 cm H₂O
- D Decreased chest wall compliance, high-frequency oscillator ventilation
- E Decreased lung compliance, bronchodilators

The high plateau pressures in this patient are concerning for worsening lung function or poor chest-wall mechanics due to obesity that don't allow for proper gas exchange. One way to differentiate the major cause of these elevated plateau pressures is to place an esophageal balloon. After placement, measuring the proper pressures on inspiration and expiration reveals that the largest contributing factor to these high pressures is the weight of the chest wall causing poor

chest-wall compliance. The small change in esophageal pressures, as compared with the larger change in transpulmonary pressures, indicates poor chest-wall compliance and good lung compliance. It is why the major factor in this patient's high inspiratory pressures is poor chest-wall compliance. The patient is hypotensive, so increasing the PEEP would likely result in further drop in blood pressure. This is why high-frequency oscillator ventilation would likely improve this patient's hypoxemia without affecting the blood pressure.

Answer: D

Talmor, D., Sarge, T., O'Donnell, C., and Ritz, R. (2006) Esophageal and transpulmonary pressures in acute respiratory failure. *Critical Care Medicine*, **34** (5), 1389–1394.

Valenza, F., Chevillard, G., Porro, G.A., and Gattinoni, L. (2007) Static and dynamic components of esophageal and central venous pressure during intra-abdominal hypertension. *Critical Care Medicine*, **35** (6), 1575–1581.

- 16 All of the following cardiovascular changes occur in pregnancy except:
- A Increased cardiac output
 - B Decreased plasma volume
 - C Increased heart rate
 - D Decreased systemic vascular resistance
 - E Increased red blood cell mass – "relative anemia"

The following cardiovascular changes occur during pregnancy:

- Decreased systemic vascular resistance
- Increased plasma volume
- Increased red blood cell volume
- Increased heart rate
- Increased ventricular distention
- Increased blood pressure
- Increased cardiac output
- Decreased peripheral vascular resistance

Answer: B

DeCherney, A.H. and Nathan, L. (2007) *Current Diagnosis and Treatment: Obstetrics and Gynecology*, 10th edn, McGraw-Hill, New York, chapter 7.

Yeomans, E.R. and Gilstrap, L.C., III. (2005) Physiologic changes in pregnancy and their impact on critical care. *Critical Care Medicine*, **33**, 256–258.

- 17 Choose the incorrect statement regarding the physiology of the intra-aortic balloon pump:

- A Shortened intraventricular contraction phase leads to increased oxygen demand
- B The tip of catheter should be between the second and third rib on a chest x-ray
- C Early inflation leads to increased afterload and decreased cardiac output
- D Early or late deflation leads to a smaller afterload reduction
- E Aortic valve insufficiency is a definite contraindication

Patients who suffer hemodynamic compromise despite medical therapies may benefit from mechanical cardiac support of an intra-aortic balloon pump (IABP). One of the benefits of this device is the decreased oxygen demand of the myocardium as a result of the shortened intraventricular contraction phase. It is of great importance to confirm the proper placement of the balloon catheter with a chest x-ray that shows the tip of the balloon catheter to be 1 to 2 cm below the aortic knob or between the second and third rib. If the balloon is placed too proximal in the aorta, occlusion of the brachiocephalic, left carotid, or left subclavian arteries may occur. If the balloon is too distal, obstruction of the celiac, superior mesenteric, and inferior mesenteric arteries may lead to mesenteric ischemia. The renal arteries may also be occluded, resulting in renal failure.

Additional complications of intra-aortic balloon-pump placement include limb ischemia, aortic dissection, neurologic complications, thrombocytopenia, bleeding, and infection.

The inflation of the balloon catheter should occur at the onset of diastole. This results in increased diastolic pressures that promote perfusion of the myocardium as well as distal organs. If inflation occurs too early it will lead to increased afterload and decreased cardiac output. Deflation should occur at the onset of systole. Early or late deflation will diminish the effects of afterload reduction. One of the definite contraindications to placement of an IABP is the presence of a hemodynamically significant aortic valve insufficiency. This would exacerbate the magnitude of the aortic regurgitation.

Answer: A

- Ferguson, J.J., Cohen, M., Freedman, R.J., *et al.* (2001) The current practice of intra-aortic balloon counterpulsation: results from the Benchmark Registry. *Journal of American Cardiology*, **38**, 1456–1462.
- Hurwitz, L.M. and Goodman, P.C. (2005) Intraaortic balloon pump location and aortic dissection. *American Journal of Roentgenology*, **184**, 1245–1246.
- Sidebotham, D., McKee, A., Gillham, M., and Levy, J. (2007) *Cardiothoracic Critical Care*, Butterworth-Heinemann, Philadelphia, PA.

- 18 Choose the **incorrect** statement regarding the West lung zones:
- A Zone 1 does not exist under normal physiologic conditions
 - B In hypovolemic states, zone 1 is converted to zone 2 and zone 3
 - C V/Q ratio is higher in zone 1 than in zone 3
 - D Artificial ventilation with excessive PEEP can increase dead space ventilation
 - E Perfusion and ventilation are better in the bases than the apices of the lungs

The three West zones of the lung divide the lung into three regions based on the relationship between alveolar pressure (PA), pulmonary arterial pressure (Pa) and pulmonary venous pressure (Pv).

Zone 1 represents alveolar dead space and is due to arterial collapse secondary to increased alveolar pressures ($PA > Pa > Pv$).

Zone 2 is approximately 3 cm above the heart and represents and represents a zone of pulsatile perfusion ($Pa > PA > Pv$).

Zone 3 represents the majority of healthy lungs where no external resistance to blood flow exists promoting continuous perfusion of ventilated lungs ($Pa > Pv > PA$).

Zone 1 does not exist under normal physiologic conditions because pulmonary arterial pressure is higher than alveolar pressure in all parts of the lung. However, when a patient is placed on mechanical ventilation (positive pressure ventilation with PEEP) the alveolar pressure (PA) becomes greater than the pulmonary arterial pressure (Pa) and pulmonary venous pressure (Pv). This represents a conversion of zone 3 to zone 1 and 2 and marks an increase in alveolar dead space. In a hypovolemic state, the pulmonary arterial and venous pressures fall below the alveolar pressures representing a similar conversion of zone 3 to zone 1 and 2. Both perfusion and ventilation are better at the bases than the apices. However, perfusion is better at the bases and ventilation is better at the apices due to gravitational forces.

Answer: B

Lumb, A. (2000) *Nunn's Applied Respiratory Physiology*, 5 edn, Butterworth-Heinemann, Oxford.

West, J., Dollery, C., and Naimark, A. (1964) Distribution of blood flow in isolated lung; relation to vascular and alveolar pressures. *Journal of Applied Physiology*, **19**, 713–724.

- 19 Choose the **correct** statement regarding clinical implications of cardiopulmonary interactions during mechanical ventilation: