38. Severinghaus JS, Stafford M, Bradley FA: Transcutaneous Po_2 electrode design, calibration, and temperature gradient problems, *Acta Anaesthesiol Scand* 68(Suppl):118–122, 1978.
39. Reed RL, Maier RV, Del Londicho M, et al.: Correlation of hemodynamic variables with transcutaneous Po_2 measurements in critically ill patients, *J Trauma* 25:1045–1053, 1985.
40. Lubbers DW: Theoretical basis of transcutaneous blood gas measurements, *Crit Care Med* 9:721–733, 1981.
41. Restrepo RD, Hirst KR, Wittnebel L, Wettstein R: AARC clinical practice guideline: transcutaneous monitoring of carbon dioxide and oxygen 2012, *Respir Care* 57:1955–1962, 2012.
42. Wahr JA, Tremper KK: Noninvasive oxygen monitoring techniques, *Crit Care Clin* 11:199–217, 1995.
43. AARC Clinical Practice Guidelines: metabolic measurement using indirect calorimetry during mechanical ventilation—2004 revision and update, *Respir Care* 39:1170–1175, 2004.
44. Ferrannini E: The theoretical basis of indirect calorimetry: a review, *Metabolism* 37:287–301, 1987.
45. Head CA, Grossman GD, Jordan JC, et al.: A valve system for the accurate measurement of energy expenditure in mechanically ventilated patients, *Respir Care* 30:969–973, 1985.
46. Browning JA, Linberg SE, Turney SZ, et al.: The effects of a fluctuating F_io_2 on metabolic measurements in mechanically ventilated patients, *Crit Care Med* 10:82–85, 1982.
47. Ultman JS, Bursztein S: Analysis of error in determination of respiratory gas exchange at varying Fio_2, *J Appl Physiol* 50:210–216, 1981.
48. Consolazio CF, Johnson RE, Pecora IJ: *Physiological measurements of metabolic function in man*, New York, NY, 1963, McGraw-Hill.
49. Aschoff P, Pohl J: Rhythm variations in energy metabolism, *Fed Proc* 29:1541–1542, 1970.
50. Weissman C, Kemper M: Metabolic measurements in the critically ill, *Crit Care Clin* 11:169–197, 1995.
51. Vermeij C, Feenstra BWA, Van Lanchot JB, et al.: Day to day variability of energy expenditure in critically ill surgical patients, *Crit Care Med* 17:623–626, 1989.
52. Askanazi J, Carpentier YA, Elwyn D, et al.: Influence of total parenteral nutrition on fuel utilization in injury and sepsis, *Ann Surg* 191:40–46, 1980.
53. Askanazi J, Rosenbaum SH, Hyman AL, et al.: Respiratory changes induced by large glucose loads of total parenteral nutrition, *JAMA* 243:1444–1447, 1980.
54. Marini JJ: Lung mechanics determination at the bedside: instrumentation and clinical application, *Respir Care* 35:669–696, 1990.
55. Kacmarek RM, Hess DR: Airway pressure, flow, and volume waveforms and lung mechanics during mechanical ventilation. In Kacmarek RM, Hess DR, Stoller JK, editors: *Monitoring in respiratory care*, St. Louis, MO, 1993, Mosby, pp 497–544.
56. Nilsestuen JO, Hargett KD: Using ventilator graphics to identify patient-ventilator asynchrony, *Respir Care* 50:202–234, 2005.
57. East TD: What makes noninvasive monitoring tick? A review of basic engineering principles, *Respir Care* 35:500–519, 1990.
58. Hess DR: Respiratory mechanics in mechanically ventilated patients, *Respir Care* 59(11):1773–1794, 2014.
59. Sanborn WG: Monitoring respiratory mechanics during mechanical ventilation: where do the signals come from? *Respir Care* 50(1):28–52, 2005.
60. Levitzky MG: *Pulmonary physiology*, ed 9, New York, NY, 2018, McGraw-Hill.
61. Branson RD, Davis K: Work of breathing by five ventilators used for long-term support: the effects of PEEP and simulated patient demand, *Respir Care* 40:1270–1278, 1995.
62. Marini JJ: The role of the inspiratory circuit in the work of breathing during mechanical ventilation, *Respir Care* 32:419–430, 1987.
63. Hess DR, Kacmarek RM: *Essentials of mechanical ventilation*, ed 4, New York, NY, 2019, McGraw-Hill.
64. Branson RD: Enhanced capabilities of current ICU ventilators: do they really benefit patients? *Respir Care* 36:362–376, 1991.
65. MacIntyre NR: Weaning from mechanical ventilatory support: volume-assisting intermittent breaths versus pressure-assisting every breath, *Respir Care* 33:121–125, 1988.
66. Marini JJ, Capps J, Culver B: The inspiratory work of breathing during assisted mechanical ventilation, *Chest* 87:612–618, 1985.
67. Henning RJ, Shubin H, Weil MH: The measurement of the work of breathing for the clinical assessment of ventilator dependence, *Crit Care Med* 5:264–268, 1977.
68. Benditt JO: Esophageal and gastric pressure measurements, *Respir Care* 50:68–75, 2005.
69. Bellemare F, Grassino A: Effect of pressure and timing of contraction on human diaphragm fatigue, *J Appl Physiol* 53:1190–1195, 1982.
70. Field S, Sanci S, Grassino A: Respiratory muscle oxygen consumption estimated by the diaphragm pressure-time index, *J Appl Physiol* 57:44–51, 1984.
71. Sassoon CSH, Light RW, Lodia R, et al.: Pressure-time product during continuous positive airway pressure, pressure support ventilation, and T-piece during weaning from mechanical ventilation, *Am Rev Respir Dis* 143:469–475, 1991.

CHAPTER 11

Hemodynamic Monitoring

OUTLINE

KEY TERMS

- Afterload
- Balloon-tipped, flow-directed catheter
- Cardiac index
- Cardiac work
- Central venous lines
- Contractility
- Diastole
- dP/dT
- Ejection fraction
- Fick principle
- French (Fr) size
- Incisura
- Physiological shunt
- Preload
- Pulmonary artery catheter
- Pulmonary vascular resistance
- Pulse pressure
- Retrograde
- Stroke index
- Stroke work
- Swan-Ganz catheter
- Systemic vascular resistance
- Tachycardia

LEARNING OBJECTIVES

On completion of this chapter, the reader will be able to do the following:

1. Discuss how changes in heart rate, preload, contractility, and afterload can alter cardiac function and cardiac output.
2. Identify indicators of left ventricular preload, contractility, and afterload.
3. Name the major components of a hemodynamic monitoring system.
4. Explain the proper technique for insertion and maintenance of a systemic arterial line, and list the most common complications that can occur with this type of monitoring system.
5. Describe the procedures for insertion and placement of a central venous line, and list the potential complications associated with these devices.
6. Interpret the waveforms generated during the insertion of a pulmonary artery catheter.
7. Calculate arterial and venous oxygen content, cardiac output, cardiac index, stroke index, cardiac cycle time, left ventricular stroke work index, right ventricular stroke work index, and pulmonary and systemic vascular resistance.
8. List normal values for measured and derived hemodynamic variables.
9. Describe the most common complications associated with pulmonary artery catheterization and discuss strategies that can be used to minimize these complications.
10. Compare the effects of spontaneous and mechanical ventilation breathing on hemodynamic values.
11. Define the following terms: *incisura, pulse pressure, stroke index, stroke work, systemic vascular resistance, pulmonary vascular resistance, ejection fraction.*
12. Explain how measurements of pulmonary artery occlusion pressure can be used to evaluate left ventricular function.
13. Differentiate between cardiogenic and noncardiogenic pulmonary edema using hemodynamic parameters.
14. From a patient case, describe how hemodynamic monitoring can be used in the diagnosis and treatment of selected cases of critically ill patients.

The primary indication for hemodynamic monitoring is the management of critically ill patients who demonstrate evidence of compromised cardiovascular function. Hemodynamic monitoring can therefore be used for the diagnosis and treatment of life-threatening conditions, such as shock, heart failure, pulmonary hypertension, complicated

myocardial infarction, acute respiratory distress syndrome (ARDS), chest trauma, severe burn injury, severe dehydration, and after cardiac surgery.

Invasive hemodynamic monitoring requires the insertion of arterial and intracardiac catheters. Measurements typically include systemic arterial pressure, central venous pressure, pulmonary artery (PA) pressures, arterial and mixed venous blood gases, and cardiac output. Once obtained, these measurements can be used to calculate a series of derived variables, including oxygen (O_2) delivery (DO_2), **cardiac index** (CI), stroke index (SI), vascular resistance, and cardiac work, which in turn can be used to better define abnormalities in cardiopulmonary function and ultimately guide therapeutic interventions.

The efficacy and potential risks of hemodynamic monitoring have been the subject of considerable debate. Although the frequency of using invasive techniques has significantly decreased with the introduction of noninvasive technology (e.g., pulse oximetry, cardiac ultrasound), most agree that the benefits outweigh the risks in critically ill patients who require continuous invasive hemodynamic monitoring. A consensus statement released in 2000 by the National Heart, Lung, and Blood Institute and the U.S. Food and Drug Administration suggested that more randomized clinical studies should be performed to better determine which patients would benefit most from hemodynamic monitoring.[1] Furthermore, it was agreed that all clinicians involved in hemodynamic monitoring should have a working knowledge of cardiovascular physiology and the technical problems most often encountered with this type of monitoring. This chapter presents information that will help clinicians avoid many of the technical problems associated with hemodynamic monitoring. It also provides a concise description of the measurements and derived variables that are most often used and explains how they can be applied to patient treatment.

REVIEW OF CARDIOVASCULAR PRINCIPLES

It is important to understand the sequence of mechanical events occurring during the cardiac cycle to appreciate fully the various factors that influence hemodynamic measurements. Fig. 11.1 is a Wiggers diagram that illustrates the pressure and volume events that occur in the left atrium, left ventricle, and aorta during a single heartbeat. A more detailed description of these events can be found in the references listed at the end of this chapter.[2,3]

Factors Influencing Cardiac Output

The outputs of the right and left ventricle are ultimately influenced by four main factors: heart rate, preload, contractility, and afterload. An individual's heart rate, which is simply the number of times the heart beats per minute, can vary considerably, depending on the patient's age and body habitus, core temperature, level of activity, and even psychological state. Indeed, heart rates can range from 50 to 200 beats per minute in a normal healthy adult.

The **preload**, which is typically defined as the filling pressure of the ventricle at the end of ventricular **diastole**, is estimated by measuring the end-diastolic pressures (EDPs). The amount of blood present in the ventricle at the end of ventricular diastole depends on the level of venous return and compliance of the ventricle. Ultimately, preload reflects the length of the ventricular muscle fibers and thus the ability of these fibers to generate the necessary tension in the next ventricular contraction. This is a basic principle of cardiovascular physiology (sometimes called the Frank-Starling mechanism or length-tension relationship). This principle states in basic terms that the heart pumps what it receives. This relationship holds until one reaches relatively high ventricular volumes, when the muscle fibers are overstretched and unable to generate the necessary tension to elicit a contraction that will adequately eject the required stroke volume (SV). The end result at these higher ventricular volumes is ventricular dilation and failure.

The right ventricular end-diastolic pressure (RVEDP) is typically used as an indicator of the right ventricular preload, and the left ventricular end-diastolic pressure (LVEDP) is used to estimate the left ventricular preload. Because both of these intracardiac pressures are difficult to measure in the critical care setting, clinicians rely on measurements of right atrial (RAP) or central venous pressure (CVP) and PA occlusion pressure (PAOP; which for practical purposes is equivalent to pulmonary capillary wedge pressure [PCWP]) to estimate the RVEDP and LVEDP, respectively. It is important to understand that CVP and PAOP can accurately reflect the RVEDP and LVEDP only when the former measurements are made at the end of ventricular diastole and if there is no evidence of valve dysfunction because they are registering **retrograde*** pressures in the right and left atria.

Contractility, which is related to the force that the ventricle generates during each cardiac cycle, can be estimated using the **ejection fraction**, which is calculated as the ratio of the SV to the ventricular end-diastolic volume. For example, a 154-lb (70-kg) adult man typically has an SV of about 70 mL and an end-diastolic volume of about 140 mL. His ejection fraction would be 0.5 (70 mL/140 mL). Alternatively, contractility can be estimated by calculating the slope of ventricular pressure time interval during the initial period of contraction. This change in pressure relative to time (**dP/dT**) is thought to be a practical way of estimating the force of ventricular muscle contraction.

Afterload is usually defined as the impedance that the left and right ventricles must overcome to eject blood into the great vessels. Clinically, this impedance is better expressed as systemic and pulmonary vascular resistances. The **systemic vascular resistance** (SVR) is used to describe the afterload that the left ventricle must overcome to eject blood into the systemic circulation. The **pulmonary vascular resistance** (PVR) reflects the afterload that the right ventricle must overcome to eject blood into the pulmonary circulation. Increases in afterload are generally associated with reductions in cardiac output, whereas decreases in afterload are associated with increases in cardiac output. For example, systemic hypertension and pulmonary hypertension lead to increases in SVR and PVR, respectively. In both cases, the cardiac output will be reduced. Administering a systemic vasodilator (e.g., nitroprusside) or a pulmonary vasodilator (e.g., tolazoline) will reduce the SVR and PVR, respectively, and result in an increase in cardiac output.

Retrograde means moving in the opposite direction. In this case, the catheter is measuring pressures downstream from the catheter tip. Measurements made at the end of diastole will occur when the atrioventricular valves are open. Any type of valve dysfunction that alters the path between the atria and the ventricles will therefore lead to erroneous measurements.

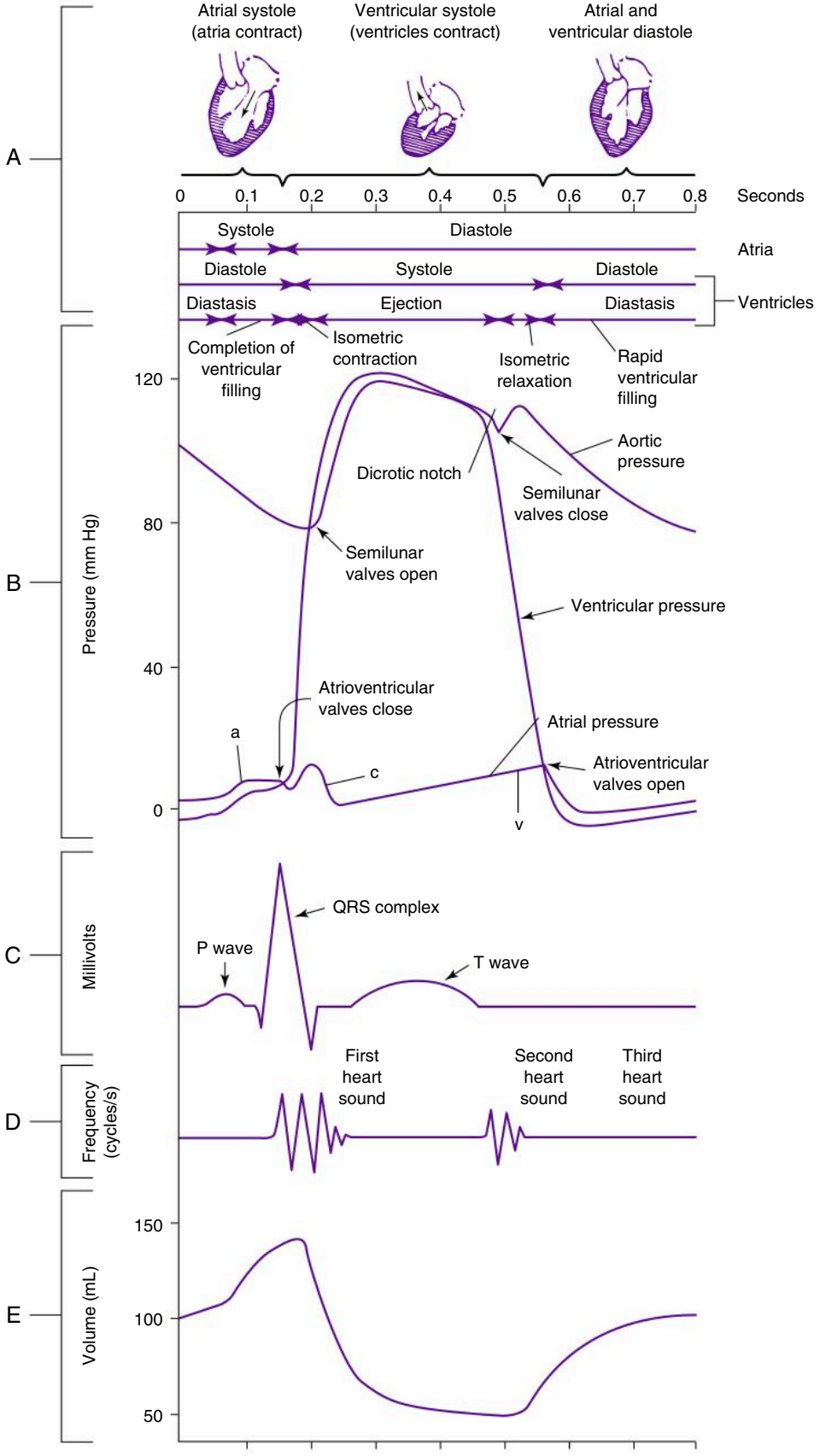


Fig. 11.1 Wiggers diagram illustrating the pressure, volume, and flow changes that occur in the systemic circulation during one cardiac cycle. (A) Timing of cardiac events; (B) simultaneous pressures created in the aorta, left ventricle, and left atrium during the cardiac cycle; (C) electrical activity during the cardiac cycle; (D) heart sounds corresponding to the cardiac cycle; (E) ventricular blood volume during the cardiac cycle. (See text for additional information.) (From Moffett DF, Moffett SB, Schauf CL: *Human physiology: foundations and frontiers,* ed 2, St. Louis, MO, 1993, Mosby.)

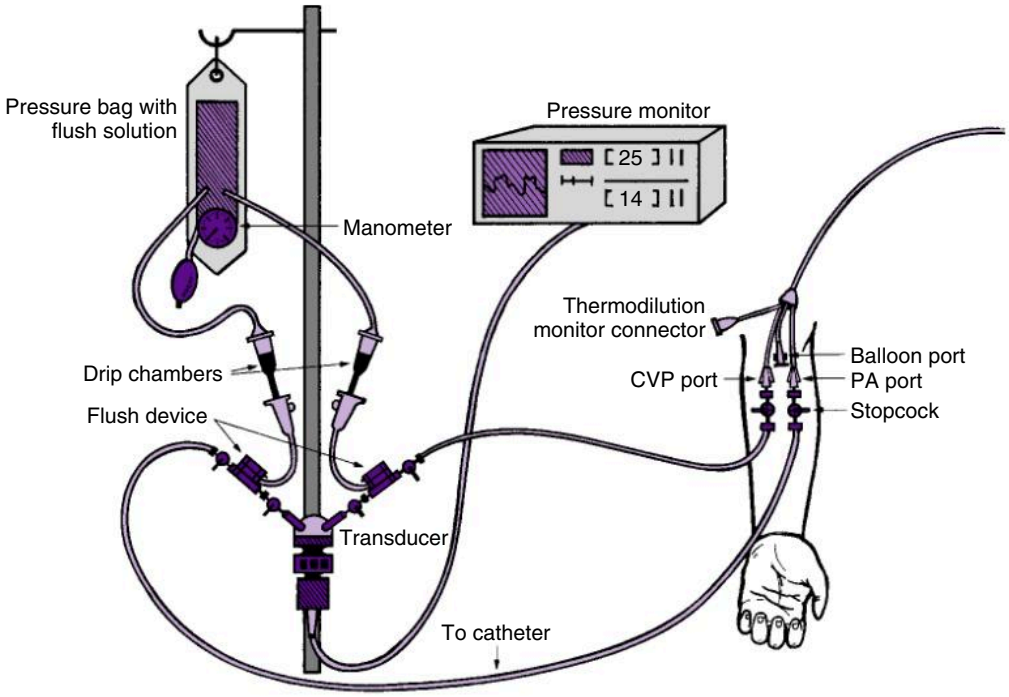


Fig. 11.2 Invasive vascular monitoring system showing the catheter connected from the vessel to a transducer and pressurized intravenous bag containing heparinized saline. The transducer is connected to an amplifier and recording device and monitor. (The transducer pictured is an older model used here for easier visualization.)

OBTAINING HEMODYNAMIC MEASUREMENTS

Clinicians who perform hemodynamic monitoring must understand the physical and technical factors that can influence intravascular measurements and thus determine the likelihood of obtaining meaningful data. The following sections first describe how hemodynamic measurements are obtained and then discuss how various pathophysiological events can alter pressure, volume, and flow tracings.

Hemodynamic Monitoring Systems

Hemodynamic monitoring systems consist of equipment that detects small physiological signal (vascular pressure) changes and converts them to electrical impulses, which can then be amplified and recorded on a computer monitor or strip chart recorder. As shown in the invasive vascular monitoring system in Fig. 11.2, a catheter is inserted into a peripheral artery, a central vein, or PA. The catheter transmits pressure changes within the vessel or cardiac chamber through a hollow plastic tubing filled with a heparinized solution of saline to a transducer, which converts these fluid pressure changes to a digital signal for display.

A strain gauge pressure transducer (Fig. 11.3) uses an electrical circuit known as a *Wheatstone bridge*.[3] The operating principle for this type of device is fairly straightforward. Fluid enters the dome portion of the transducer by way of the fluid-filled plastic line (see Figs. 11.2 and 11.3). A diaphragm separates the fluid-filled compartment from the electronic portion of the transducer. Pressure transmitted to the fluid-filled dome portion of the transducer causes movement of the diaphragm, which alters the length of the wires of the Wheatstone bridge. Changes in the length of the wires result in changes in resistance to the current flow through the Wheatstone bridge (i.e., an increase in the length of the wires results in an increased electrical resistance; decrease in length results in a decrease in resistance to electron flow). Thus the

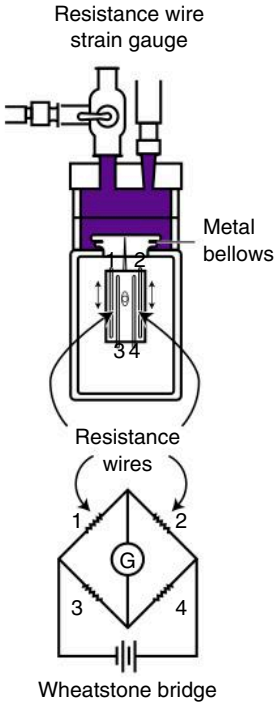


Fig. 11.3 Strain gauge pressure transducer incorporating a Wheatstone bridge. (From Cairo JM: *Mosby's respiratory care equipment,* ed 11, St. Louis, MO, 2022, Elsevier.)

pressure changes are directly proportional to the change in current flow through the circuit and inversely proportional to the resistance offered by the circuit.

Fluid Pressures

Fluid can exert pressure while it is in motion (hydrodynamic pressure) or while at a standstill (hydrostatic pressure). These pressures play integral roles when using hemodynamic monitoring systems. Two factors affect fluid-filled systems:

1. Dynamic pressure element
2. Static pressure head

The *dynamic pressure element* is pressure applied to fluid inside a system by fluid outside a system. It has to do with catheter position in relation to the flow of the blood within the vessel. For example, for an arterial line placed in a radial artery to accurately measure pressures that are the direct result of the work of the left heart, it must be placed with the end of the catheter facing the source of blood flow ("looking" upstream toward the left ventricle). A PA catheter, which measures pressures within a vessel (e.g., pulmonary capillaries) or left heart chamber, must be placed so that the tip of the catheter is facing downstream of the flow of blood (i.e., right ventricle) to estimate blood pressures filling the left side of the heart.

Static pressure head refers to the pressure placed on the transducer as related to the tip of the catheter. To measure pressure accurately, the transducer must be at the same height or level as the catheter tip. If the catheter tip is higher than the transducer, the monitor will read higher than the actual pressure as a result of the fluid "pushing" downstream (because of gravity) from the catheter tip to the transducer. If the catheter tip is placed below the level of the transducer, the fluid will flow away from the transducer toward the catheter tip and produce a reading lower than the actual pressure. The degree of error is about 1.86 mm Hg for each centimeter of distance from the reference point (e.g., if the transducer is 5 cm above the reference point, the corrected measurement should be 9.3 mm Hg [$5 \times 1.86 = 9.3$]). This adjustment can be avoided by positioning the transducer at the level of the midthoracic line of the patient—the *epistatic* line—to measure CVP accurately. This is about 5 cm behind the sternal angle (the angle of Louis). Once the epistatic line has been determined, it is important to zero-balance the transducer to this reference point. Zero-balancing the transducer references the transducer to atmospheric pressure, which is usually read as 0 mm Hg.

Systemic Artery Catheterization

Direct measurement of the systemic arterial pressure requires the insertion of a catheter into a peripheral artery, such as the radial, brachial, or femoral arteries. Note that the radial artery is the most commonly used site because of easy accessibility and collateral circulation to the hand from the ulnar artery. Either a percutaneous technique or a surgical cutdown technique can be used to insert the catheter. The percutaneous approach is most often used in the critical care setting; the surgical cutdown procedure is used when the percutaneous technique is unsuccessful.[4,5] Box 11.1 summarizes the recommended technique for inserting and maintaining a systemic arterial line (Key Point 11.1).

After the catheter has been positioned in the artery, a surgical dressing is applied and the catheter is secured with tape or sutured to the skin. Maintenance of the arterial line requires the use of a continuous pressurized flush mechanism to irrigate the catheter with a heparinized solution at a low flow (e.g., 2–3 mL/h).[5] The irrigating solution of 0.9% sodium chloride (NaCl) containing 1 to 2 units of heparin per milliliter of normal saline (i.e., 100–200 units of heparin/dL). Commercially available arterial line sets also have a rapid flush mechanism for quickly clearing the catheter. Prolonged or frequent flushing of the arterial line should be avoided because this can lead to the inadvertent administration of large amounts of flush volume to the patient. This latter point is a particularly important issue for neonatal and pediatric patients who weigh less than 44 lb (20 kg).

Daily inspection of the insertion site for signs of infection, ischemia, and bleeding is essential to avoid serious complications. The following factors can increase the risk for infections in patients with arterial lines:

- Insertion of the arterial line by surgical cutdown
- Prolonged cannulation (>4 days)
- Altered host defense

Distal ischemia should be suspected when pallor distal to the insertion site occurs, particularly if it is accompanied by pain and paresthesia in the affected limb. The most common cause of decreased perfusion is thrombus formation, which occludes the catheter tip. Other risk factors for acute distal ischemia include hypotension, severe peripheral vascular disease, and the use of vasopressor drugs.[4,5]

Fever in any patient with an intravascular line should alert the clinician to infectious complications. The catheter is removed if there is evidence of local infection or the presence of distal ischemia. Difficulty withdrawing blood or persistence of damped

BOX 11.1 Insertion and Maintenance of Arterial Catheters

- Aseptically assemble, flush, and test tubing and catheter.
- Perform a modified Allen test to ensure ulnar artery refill time of 5 to 10 seconds.
- Prepare and drape the area of insertion (sterile technique is necessary).
- If necessary, infiltrate the skin around the insertion site with a local anesthetic (e.g., lidocaine).
- Percutaneously insert the catheter at approximately 30-degree angle. (If pulse is weak or otherwise inaccessible, surgical cutdown may be necessary.)
- Advance the catheter into the artery while holding the needle secure.
- Remove the needle and secure the catheter.
- Attach tubing for drip solution and observe monitor for proper waveform.
- Frequently monitor:
  - Insertion site for signs of infection
  - Extremity distal to insertion site for adequate circulation
- Catheter should be removed if:
  - There is clot formation as evidenced by difficulty with blood sampling or a persistently damped waveform
  - Extremity distal to insertion site becomes ischemic
  - Insertion site becomes infected

Key Point 11.1 The Modified Allen test must be performed before a radial artery catheter is inserted.

tracings should also alert the clinician to possible complications, such as the presence of air bubbles in the line or occlusion of the catheterized artery.

Bleeding can occur if the line becomes disconnected or is improperly handled. Hemorrhage is a distinct possibility if the line is left open; therefore the clinician should stabilize the catheterized site by taping the patient's arm to a board and placing it above the bed linen for easy observation. Another common problem encountered with the insertion of arterial lines is hematoma formation, which can develop if bleeding occurs under the skin during or immediately after the catheterization procedure. Hematoma typically occurs more often when the catheter is placed through a large-gauge needle.

Central Venous Lines

Catheters placed in the vena cava or right atria are generally called **central venous lines**. Although these lines are most often used to administer fluids, drugs, and nutritional solutions, they can also be used to monitor right heart pressures. During ventricular systole or atrial diastole, when the tricuspid valve is closed, the pressure measured in the right atrium or vena cava reflects the RAP. At the end of ventricular diastole and atrial systole, when the tricuspid valve is open, the pressure measured in the right atrium reflects the right ventricular pressure. Thus the CVP measured at the end of ventricular diastole can be used to monitor intravenous (IV) fluid administration and estimate the filling pressure or preload of the right ventricle (i.e., RVEDP).

CVP catheters are usually inserted percutaneously into a large central vein, such as the internal jugular, or peripherally through the medial basilic or lateral cephalic vein.[6] Pressure measurements are usually performed during exhalation and when the patient is supine. The transducer is zeroed at the level of the right atrium. A normal value for CVP is 2 to 6 mm Hg.

The most common problems encountered with insertion of CVP catheters are pneumothorax, hemothorax, and vessel damage. Other potential complications include infection, thrombosis, and bleeding. Placement of the catheter usually is confirmed using radiography.

Pulmonary Artery Catheterization

Bedside catheterization of the right heart and PA became possible when Swan and colleagues[7] introduced a **balloon-tipped, flow-directed catheter** in the early 1970s.[8] The balloon-tipped, flow-directed catheter (also referred to as the **Swan-Ganz catheter** or **pulmonary artery catheter**) (Fig. 11.4) is a multiple-lumen catheter constructed of radiopaque polyvinylchloride. The standard adult catheter is 110 cm in length and is available in 7- and 8-**French (Fr) sizes**. Remember that the French size divided by π, or 3.14, equals the external diameter of the catheter in millimeters. Pediatric catheters are 60 cm in length and available in 4- and 5-Fr sizes. Both adult and pediatric catheters are marked at 10-cm increments. As with systemic arterial catheters, a pressurized flush solution must be run through the catheter at a rate of 1 to 5 mL/h (except when making pressure measurements) to prevent clot formation within the catheter's lumen.

Dual-lumen catheters have one lumen that connects to a balloon located near the tip of the catheter and a second lumen that runs the length of the catheter and terminates at a port at the distal end of the catheter. Triple-lumen catheters have an additional proximal port that terminates approximately 30 cm from the tip of the catheter, or at the level of the right atrium. This third lumen can be used to measure right atrial pressures or for administrating intravenous medications.

Thermodilution catheters incorporate a thermistor connector, which contains electrical wires that connect to a thermistor located approximately 1.5 inches (3 cm) from the tip of the catheter. When measuring cardiac output using the thermodilution technique, a bolus of saline or 5% dextrose (cold or room temperature) is injected through the catheter's third (proximal) lumen, which is positioned at the level of the right atrium. The cardiac output is calculated by integrating the change in temperature that is sensed by the thermistor near the tip of the catheter as the injected saline (or dextrose) solution mixes with the patient's pulmonary blood flow.

Pacing catheters with bands for atrial, ventricular, and atrioventricular (AV) cardiac pacing are also available. The bands,

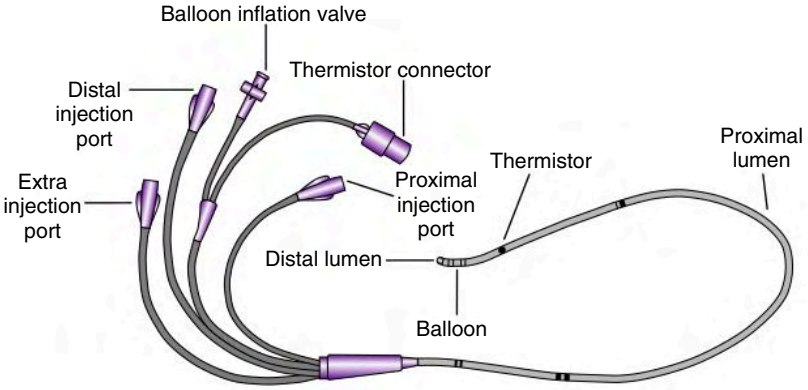


Fig. 11.4 Balloon-tipped, flow-directed right-sided heart catheter showing many of the features that are typically available on pulmonary artery (PA) catheters. Distal lumen opens into the PA. Fiberoptic filaments used for mixed venous oxygen saturation ($S\bar{v}O_2$) monitoring and the balloon are also located at the tip. The thermistor bead is located 1.5 inches from the tip and is connected by a wire through the catheter to the connector for the thermodilution cardiac output computer. The proximal lumen located 30 cm back from the tip opens into the right atrium. Catheters are available for ventricular, atrial, or atrioventricular sequential pacing using either pacemaker bands positioned on the catheter or pacing leads, which are passed through the lumen. Ventricular bands or lumens are located 20 cm from the tip and atrial located 30 cm from the tip.

which are located approximately 20 cm from the tip of the catheter, have external leads that connect to a pacemaker.

As with CVP catheters, PA catheters can be inserted using a percutaneous approach or via surgical cutdown technique. The subclavian, internal jugular, external jugular, femoral, or antecubital veins are usually used for percutaneous insertion; surgical cutdown is often necessary when the subclavian or antecubital veins are used. The clinician must consider a number of potential complications when choosing an insertion site, including pneumothorax, arterial lacerations, venous thrombosis or phlebitis, and air embolism (Table 11.1).

Positioning the catheter can be accomplished by fluoroscopy or by monitoring the pressure tracings generated as the catheter is slowly advanced into the right side of the heart and PA. Fluoroscopy is typically reserved for the catheterization laboratory, whereas continuous pressure monitoring with electrocardiography is routinely used in the critical care setting.

TABLE 11.1 Insertion Sites of the Pulmonary Artery Catheter and Associated Problems

Site	Associated Problems
Internal jugular	Pneumothorax, hemothorax
Subclavian	Severe thrombocytopenia (difficult-to-control bleeding), pneumothorax (more frequently than with internal jugular), hemothorax
Antecubital	Phlebitis; catheter tip may migrate with movement of the arm; difficult site for catheter advancement
Femoral	Phlebitis; catheter tip may migrate with movement of the leg

Fig. 11.5 shows a series of tracings obtained during PA catheter placement.[8,9] The catheter is introduced into the peripheral vein and slowly advanced until it enters the intrathoracic vessels (i.e., superior or inferior vena cava). When the catheter enters the intrathoracic vessels, venous pulse waveforms with characteristic respiratory fluctuations are recorded. If these respiratory fluctuations are absent, the results obtained will be erroneous. Loss of these respiratory fluctuations may indicate that the stopcock is closed between the catheter and pressure transducer. It can also occur if the catheter is kinked or a blood clot or air is present in the tubing. Once the catheter enters the intrathoracic vessels, the balloon is inflated with air so that it can be flow-directed by the blood through the right atrium and right ventricle into the PA. (NOTE: It is important to fully inflate the balloon to avoid endocardial or PA damage or induce ventricular arrhythmias.) Pediatric (4–5 Fr) catheters have a balloon volume of 0.8 mL; the balloon volume for adult (7–8 Fr) catheters is 1.5 mL. The catheter is slowly advanced until it wedges in a small PA.

As shown in Fig. 11.5, the wedged position can be easily identified because the PA occlusion pressure (PAOP) is characteristically lower than or equal to the PA diastolic pressure. An overdamped tracing (i.e., loss of a distinctive PAOP waveform) usually indicates a mechanical problem, such as the presence of an air bubble in the catheter tubing or protrusion of the balloon over the catheter tip. The mean PAOP may exceed the PA diastolic pressure in patients with mitral stenosis or mitral regurgitation.[10] The PAOP can be identified by another means; when the catheter occludes a small PA, a continuous column of blood equilibrates between the left atrium and distal port of the catheter and the

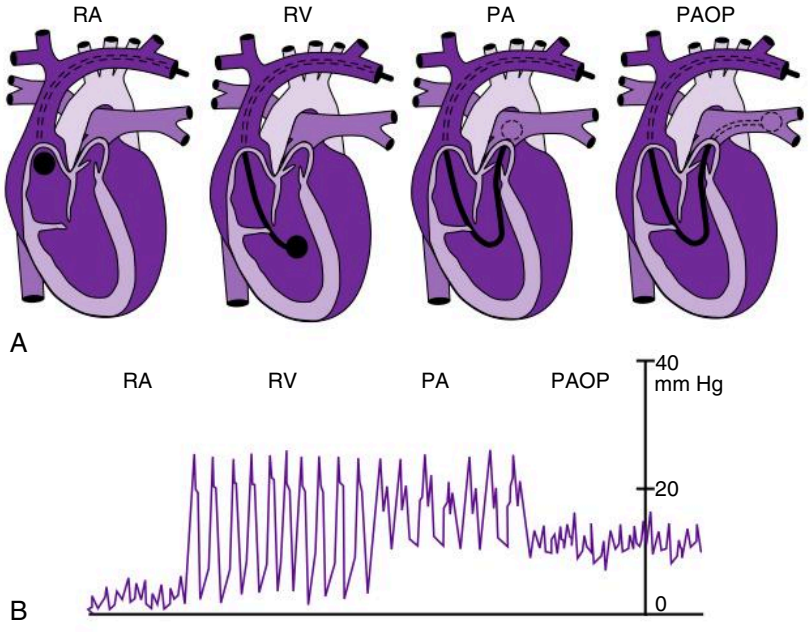


Fig. 11.5 The waveforms seen during the advancement of the catheter from the right atrium to the wedged position. (A) Position of the pulmonary artery catheter in the heart. (B) Corresponding waveforms seen on a pressure-time tracing as the catheter is advanced from the right atrium to the wedged position. *PA,* Pulmonary artery; *PAOP,* pulmonary artery occlusion pressure; *RA,* right atrial pressure; *RV,* right ventricle. (From Piraino T: Monitoring the patient in the intensive care unit. In: Kacmarek RM, Stoller JS, Heuer AJ, editors: *Egan's fundamentals of respiratory care,* ed 12, St. Louis, MO, 2021, Elsevier.)

pressure tracing will register a left atrial waveform (i.e., a, c, and v waves).

The catheter must be wedged in a zone 3 position in the lung to reflect the pulmonary venous pressure accurately.[11] As illustrated in Fig. 11.6, if the catheter is positioned in zone 1 or zone 2, the alveolar pressure will exceed the pulmonary venous pressure and cause the vessels distal to the balloon to collapse (Table 11.2). The intravascular pressure reading will reflect the intraalveolar pressure rather than the pressure transmitted from the left atrium. This problem is accentuated by the application of positive end-expiratory pressure (PEEP) during mechanical ventilation (i.e., the alveolar pressure rises) or by hemorrhage when the pulmonary venous pressure is reduced. We discuss this problem in more detail later in this chapter (Key Point 11.2).

***Key Point* 11.2** The PA catheter must be wedged in a zone 3 position in the lung to reflect the pulmonary venous pressure accurately.

Table 11.3 lists the most common complications associated with right heart catheterization using a balloon-tipped, flow-directed catheter. Ventricular arrhythmias are fairly common during catheter insertion and are often self-limiting. Electrolyte disturbances, hypoxemia, and acidosis increase the risk for developing ventricular ectopy. Careful monitoring of the patient's

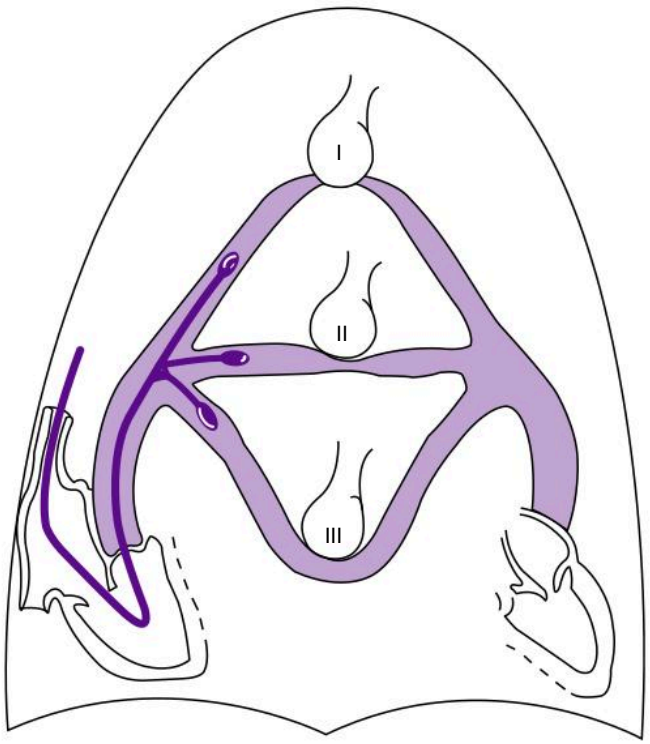


Fig. 11.6 The position of the pulmonary artery (PA) catheter tip in relation to West's zone of the lung. For PA occlusion pressure to be a valid estimate of left heart pressures, a continuous column of blood needs to be present between the catheter tip and left atrium. In zones I and II, the pulmonary vessels may be partially or completely compressed by adjacent pulmonary alveolar pressures. (See text for additional information.)

TABLE 11.2 West's Zones of the Lung

Lung Zone	Pressure Relationships	Explanation
Zone 1	$P_{alv} > P_a > P_v$	Essentially functions as alveolar dead space—ventilation in excess of perfusion
Zone 2	$P_a > P_{alv} > P_v$	Blood flow is a result of the pressure difference between pulmonary artery and alveolar pressures
Zone 3	$P_a > P_v > P_{alv}$	Blood flow is a result of arteriovenous pressure differences

P_a, arterial pressure; P_{alv}, alveolar pressure; $P\bar{v}$, venous pressure.

TABLE 11.3 Complications Associated With Pulmonary Artery Catheterization

Complications	Causes
Cardiac Arrhythmias	
Premature ventricular contraction (PVC) Premature atrial contraction (PAC)	Heart valve or endocardium irritation by the catheter
Ventricular tachycardia	
Ventricular fibrillation	
Atrial flutter	
Atrial fibrillation	
Insertion Procedure or Insertion Site	
Infection	Nonsterile technique or irritation of the wound
Pneumothorax	Air entering pleural space during insertion
Air embolism	Air entering vessel during insertion
Access vessel thrombosis	Irritation of vessel by catheter or nonsterile insertion technique or phlebitis
Pulmonary Circulation	
Pulmonary artery rupture or perforation	Overinflation of catheter balloon
Pulmonary infarction	Overinflation of catheter balloon, prolonged wedging, clots formed in or near the catheter, or catheter advancement into a smaller artery
Pulmonary Artery Catheter	
Balloon rupture—air embolism	Loss of catheter balloon elasticity or overinflation
Catheter knotting	Excessive catheter movement
Damped waveform	Air in line, clot in the system, kinks in line, catheter tip against vessel wall, overwedging, or blood on the transducer
Catheter whip or fling	High cardiac output or abnormal vessel diameter

BOX 11.2 Risk Factors for Catheter-Associated Pulmonary Artery Rupture

- Age >60 years
- Pulmonary hypertension
- Improper balloon inflation
- Improper catheter positioning
- Cardiopulmonary bypass surgery
- Anticoagulation therapy

electrocardiogram during catheter placement can alert the clinician of the development of arrhythmias and reduce chances of provoking a potentially lethal complication such as ventricular tachycardia and fibrillation.

Box 11.2 lists the risk factors most often associated with PA infarction and rupture.[12,13] Pulmonary artery infarction and rupture can be minimized by preventing thrombus development, which is accomplished by instilling a continuous flushed solution containing heparin. Pulmonary infarction and rupture can also be reduced by ensuring that the catheter balloon is deflated after wedge pressure measurements are made. Furthermore, it is important that the balloon is inflated for only 15 to 30 seconds when measuring the PAOP, particularly in patients with pulmonary hypertension. Balloon rupture is most often associated with prolonged duration of catheterization because the balloon will typically lose its elasticity with exposure to blood.

INTERPRETATION OF HEMODYNAMIC PROFILES

As mentioned previously, accurate interpretation of hemodynamic data requires a working knowledge of cardiovascular physiology. The hemodynamic profile ultimately focuses on factors that influence cardiac output: that is, heart rate, preload, contractility, and afterload.

The information in this section provides an overview of basic measurements and derived variables used in a standard hemodynamic profile. A list of excellent references related to the use of hemodynamic monitoring in critical care can be found at the end of this chapter for more detailed information about this area of clinical physiology.

Heart Rate

The resting heart rate of a healthy adult is typically 60 to 100 beats/min. The heart rates for neonates and infants are considerably higher. Although the resting heart rates for toddlers and adolescents are higher than for adults, the difference is minimal by the end of the first decade of life.[14] Table 11.4 compares the mean and normal range for heart rate at various stages of life.[14]

Bradycardia (heart rates <60 beats/min) is associated with increases in parasympathetic tone or decreases in sympathetic tone. **Tachycardia** (heart rates >100 beats/min) is associated with increases in sympathetic tone or decreases in parasympathetic tone.

The typical adult can maintain an adequate cardiac output at heart rates of 40 to 50 beats/min as long as SV increases proportionally. (Well-trained athletes are good examples of this concept.) Cardiac output will increase with heart rates up to about 200 to 220 beats/min, assuming that the patient responds normally to sympathoadrenal stimulation.[15] Heart rates above 220 beats/min cause a decrease in cardiac output because diastolic filling time is reduced (i.e., decreased ventricular filling from reduced venous return).

TABLE 11.4 Normal Blood Pressures and Heart Rates in Children

Age	Blood Pressure Average for Males (Girls 5% Lower)	HEART RATE Average	HEART RATE Range
Neonate	75/50	140	100–190
1–6 mo	80/50	145	110–190
6–12 mo	90/65	140	110–180
1–2 y	95/65	125	100–160
2–6 y	100/60	100	65–130
6–12 y	110/60	80	55–110
12–16 y	110/65	75	55–100

Data from Rubenstein JS, Hageman JR: Monitoring of critically ill infants and children, *Crit Care Clin* 4:621–639, 1988.

Systemic Arterial Pressure

Fig. 11.7 illustrates a typical systemic arterial pressure tracing. Normal systemic arterial pressure (systolic/diastolic pressures) for adults range from 90 to 140 mm Hg/60 to 90 mm Hg with a normal mean arterial pressure (MAP) of 70 to 100 mm Hg.* Fig. 11.8 shows the effect of age on systolic and diastolic pressure in adult subjects.[16] It is generally accepted that systemic hypertension exists when the systolic arterial pressure is greater than 140 mm Hg and the diastolic pressure is greater than 90 mm Hg. Systemic hypotension is associated with systolic pressures less than 100 mm Hg and diastolic pressures less than 60 mm Hg. It is important to recognize that systemic arterial pressures in children, particularly very young children, are significantly different from those in adults (see Table 11.4).[14]

Although it may not be apparent, the systemic arterial pressure waveform will change in shape and magnitude, depending on the site of the measurement and the age of the patient. For example, systolic pressure increases as the site of measurement moves away from the heart. This effect is more obvious in young patients than in older patients. Diastolic pressure, on the other hand, is affected by vascular tone. An increase in diastolic pressure is associated with vasoconstriction, whereas a decrease in diastolic pressure is associated with vasodilation.

Changes in vascular tone can also cause the **incisura** to shift on the downslope of the arterial pressure tracing.[16,17] Vasodilation will cause it to shift closer to the baseline. The dicrotic notch also becomes less distinct as the site of measurement is moved farther from the heart. Indeed, it may not be present in arterial pressure tracing obtained from the femoral artery.

Pulse pressure, which is the difference between the systolic and diastolic pressures, is influenced primarily by the stroke volume and the arterial compliance. A wide pulse pressure is associated with an increased SV and a decreased arterial compliance; a

*The MAP can be calculated in two ways. MAP = diastolic pressure + 1/3 (pulse pressure). Alternatively, MAP can be estimated using the formula (systolic pressure + 2[diastolic pressure])/3.

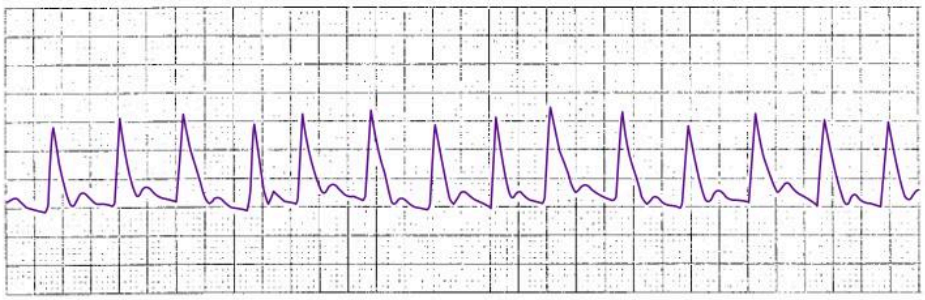


Fig. 11.7 A typical arterial pressure tracing.

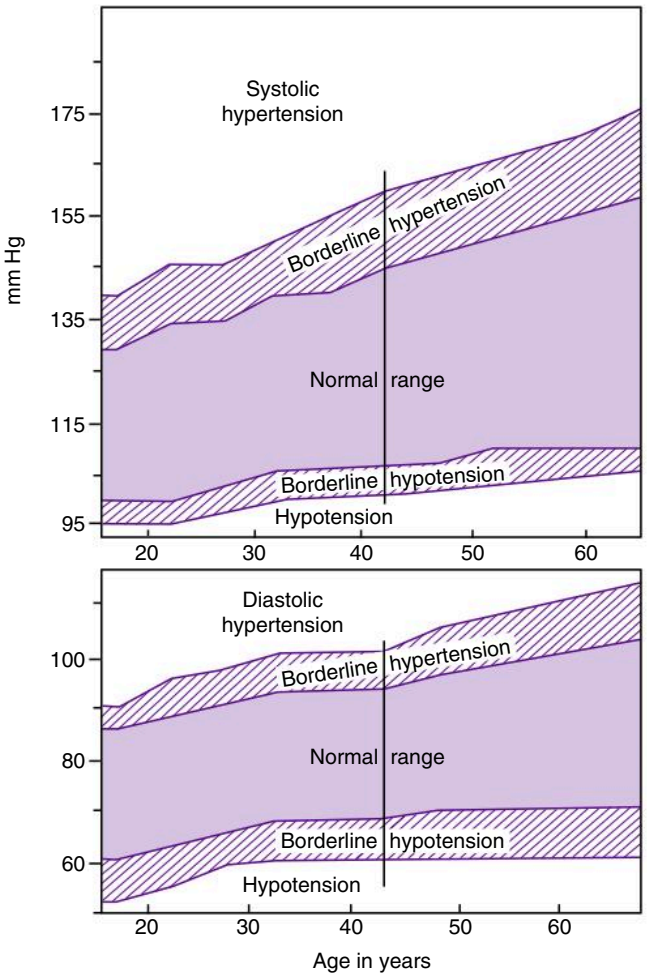


Fig. 11.8 Effect of age on arterial blood pressure measurements. (From Rushmer RF: *Cardiovascular dynamics,* ed 4, Philadelphia, PA, 1976, WB Saunders.)

narrow pulse pressure is associated with a decreased SV and an increased arterial compliance.

Right Atrial and Pulmonary Artery Pressures

Proper positioning of the balloon flotation catheter allows for continuous monitoring of RAP and PA pressure and intermittent measurements of the PAOP. The RAP can be continuously monitored through the proximal lumen of a PA catheter or through a CVP line. Pulmonary artery pressures can be monitored continuously through the distal lumen of a PA catheter. Left atrial and ventricular pressures can be measured intermittently during PAOP determinations. PAOP determinations represent retrograde pressure measurements that are obtained by inflating the balloon of the PA catheter until it occludes a small PA and wedges to block blood flow past the catheter tip. As mentioned previously, PAOP measured at the end of ventricular diastole reflects LVEDP because the mitral valve is open and the pressure in the left ventricle is transmitted backward into the pulmonary circulation to the catheter tip.

Direct measurements of right ventricular pressures are usually obtained only during insertion of the catheter. Identification of the right ventricular pressure waveform during continuous monitoring indicates that the catheter has slipped into the right ventricle. It should be repositioned by reinflating the balloon and allowing the blood flow to carry the catheter back into the PA. The balloon should be deflated after the catheter is repositioned in the PA. As mentioned, the balloon should be inflated for short periods when measuring PAOP.

Atrial Pressures

The RAP and left atrial pressure (LAP) are reported as mean values rather than as systolic and diastolic values. The RAP (more specifically CVP) normally ranges from 2 to 6 mm Hg; the LAP, as estimated from PAOP, ranges from 5 to 12 mm Hg. CVP and PAOP measurements are commonly used to determine overall fluid balance. A low CVP or PAOP suggests hypovolemia, whereas elevations of either of these pressures indicate hypervolemia or ventricular failure (Key Point 11.3).

The PAOP also plays an important role in assessment of pulmonary hydrostatic pressure in the formation of pulmonary edema. PAOP can help distinguish cardiogenic pulmonary edema (increased pulmonary capillary hydrostatic pressure) from noncardiogenic pulmonary edema (normal pulmonary capillary hydrostatic pressure) as occurs in ARDS. For example, the finding of bilateral infiltrates on chest radiographs coupled with a PAOP

Key Point 11.3 It is important to remember that CVP and PAOP reflect right and left ventricular pressures and volumes, respectively, only when the measurements are made at the end of ventricular diastole. Also, tricuspid and mitral valve disease (e.g., stenosis or regurgitation) can alter the retrograde transmission of pressure from the right ventricle to the right atrium (CVP) or from the left ventricle to the left atrium and ultimately the pulmonary circulation (PAOP).

BOX 11.3 Causes of Abnormal Right Atrial and Pulmonary Artery Occlusion Pressure Values and Patterns

Abnormal Values

Elevated Right Atrial Pressure (RAP)
- Volume overload
- Right ventricular (RV) failure
- Tricuspid stenosis or regurgitation
- Cardiac tamponade
- Constrictive pericarditis
- Chronic left ventricular (LV) failure

Elevated Pulmonary Artery Occlusion Pressure (PAOP)
- Volume overload
- Left ventricle failure
- Mitral stenosis or regurgitation
- Cardiac tamponade
- Constrictive pericarditis
- High positive end-expiratory pressure

Low RAP or PAOP
- Hypovolemia

Abnormal Patterns

Large a Waves
- Tricuspid/mitral stenosis
- Decreased ventricular compliance
- Compliance
- Loss of atrioventricular synchrony
- Third-degree block
- Any other electrical dissociation

Absent a Waves
- Atrial fibrillation
- Atrial flutter
- Junctional rhythms
- Paced rhythms
- Ventricular rhythms

Modified from Daily EK: Hemodynamic waveform analysis, *J Cardiovasc Nurs* 15:6–22, 2001.

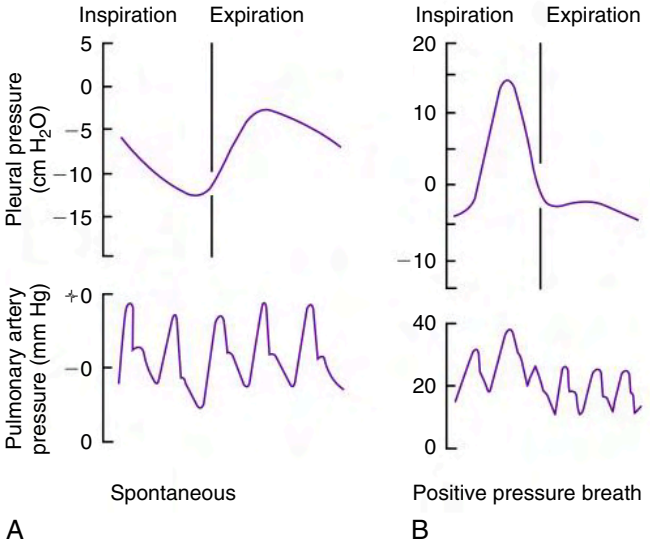


Fig. 11.9 Pulmonary artery (PA) response to ventilation. (A) PA response during ventilation. The PA pressure falls during inspiration and rises during expiration. (B) PA pressure response during mechanical ventilation. Notice that the PA pressure rises during inspiration and falls during expiration.

greater than 25 mm Hg suggests the presence of cardiogenic pulmonary edema resulting from left-sided heart failure.[18,19] This is in contrast to a finding of bilateral infiltrates on chest radiography with a normal PAOP, which would indicate the presence of noncardiogenic pulmonary edema resulting from damage to the alveolar-capillary membrane and suggests the presence of ARDS.

Box 11.3 lists several other conditions that can adversely affect RAP and PAOP values and waveforms.[17,18] Some conditions affect the magnitude of the RAP and LAP, whereas other pathophysiological events alter the contour of these atrial waveforms.

Pulmonary Artery Pressure

The PA pressure waveform resembles the systemic arterial waveform discussed previously. However, the PA systolic and diastolic pressures are considerably lower than the systemic pressures (e.g., the PA systolic pressure for a healthy adult is 15–35 mm Hg and the PA diastolic pressure is 5–15 mm Hg). As does the systemic arterial pressure tracing, the PA pressure tracing shows a rapid rise to peak pressure during systole followed by a gradual tapering to the dicrotic notch (which in this case represents closure of the pulmonic valve) and eventual descent to the end-diastolic level.

The baseline of the PA pressure tracing shows characteristic respiratory fluctuations arising from changes in the intrathoracic pressure (Fig. 11.9).[17] With spontaneous breathing, the intrapleural pressure decreases during inspiration, causing the PA wave pattern to descend. Conversely, with spontaneous expiration, intrapleural pressure increases and the wave rises. With positive pressure breathing, the curve rises as intrapleural pressures become positive and falls during the expiratory phase.[19] The intrapleural pressure is the same for spontaneous (negative pressure) and positive pressure breathing at the end of expiration as long as PEEP is not used. For this reason, PA pressure, by convention, is measured at end expiration.

The mode of mechanical ventilation used can significantly affect measured hemodynamic parameters.[19,20] It has been shown that the lower mean inspiratory pressures present with IMV and pressure support ventilation (PSV) minimize the hemodynamic effects of positive intrathoracic pressure and help maintain right heart preload and cardiac output.[20] Pressure-controlled ventilation has about the same effect on hemodynamic values as does volume-controlled ventilation. However, pressure control inverse ratio ventilation (PC-IRV) decreases CI and thus DO_2.[20-23]

Use of PEEP, either applied or inadvertent (e.g., auto-PEEP), at levels greater than 15 cm H_2O, can produce erroneously elevated pressure readings. The pressures in the thoracic circulation will rise when using PEEP therapy because of compression of the vessels by the increased lung volumes (increased functional residual capacity [FRC]). PAOP, which reflects preload of the left side of the heart, is a valuable parameter to monitor when performing an optimum PEEP study. If PAOP increases significantly during the study, it could indicate overinflation of the alveoli. Actual blood flow through the vessels might not be affected because the transmural pressure (the pressure difference between inside and outside a vessel) may not have actually decreased.

It is recommended that a patient not be taken off the ventilator, nor should PEEP be discontinued to measure PAOP, if it is desirable to assess cardiac filling during mechanical ventilation.[20] If the patient requires high levels of ventilatory support or PEEP or both, discontinuing this support for the time it would take to

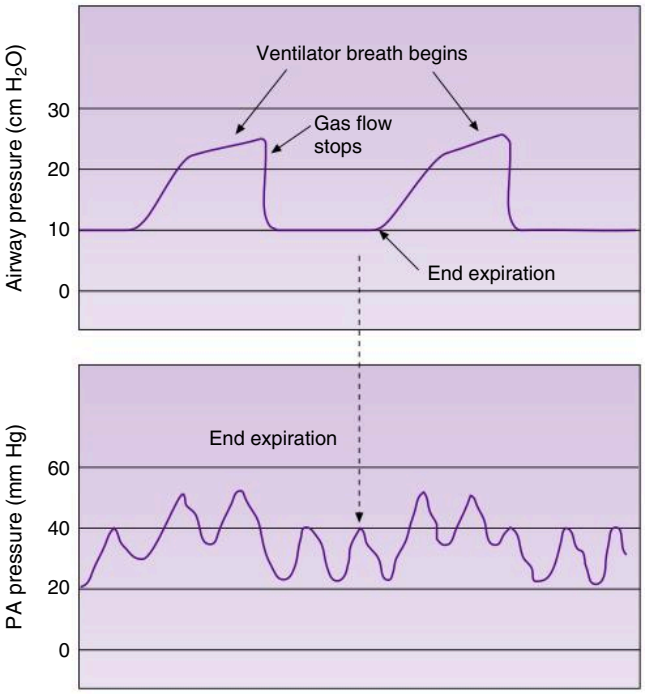


Fig. 11.10 Tracings illustrating both airway pressures and hemodynamic waveforms used to identify end expiration. (Redrawn from Ahrens TS, Taylor LA: *Hemodynamic waveform analysis,* Philadelphia, PA, 1992, WB Saunders. Used with permission.)

measure PAOP accurately could produce hypoxemia and hypoventilation, from which the patient would recover slowly.

Some practitioners calculate an airway pressure transmission ratio (APTR) and correct the PAOP obtained during ventilation by this ratio. APTR is calculated by measuring the airway pressure change during a breath and the respiratory variation in PAOP. This procedure is performed with the catheter in the wedge position and evaluated over several breaths. Change in pressure (ΔP) equals the plateau pressure (P_{plat}) minus the end-expiratory pressure (EEP). The respiratory-induced change in PAOP is the maximum mean PAOP minus the minimum mean PAOP (during a ventilator breath).[20] The transmission ratio is the PAOP divided by the change in airway pressure. The resulting "true" PAOP is

$$PAOP = EEP \times (1 - PT_{PAOP} \times PAOP)$$

where PT_{PAOP} is the PAOP pressure transmission ratio.[20]

It may not be appropriate to correct PAOP relative to the PEEP level because the practitioner does not know what effect lung zones and lung compliance actually have on PAOP. It may be prudent to trend the patient data rather than try to obtain an absolute value. It is interesting to note that right atrial pressure (i.e., CVP) may be a more accurate indicator of left ventricular end-diastolic volume than estimating APTR when a total PEEP of 10 cm H_2O or greater is present.[20]

Fig. 11.10 shows both airway pressures and PA pressure during positive pressure breathing. Fig. 11.11 shows the effect of varying levels of PEEP on PA pressure measurements.

Fig. 11.11 Effects of varying levels of positive end-expiratory pressure (PEEP) on hemodynamic measurements. As PEEP is raised from 5 to 10 to 15 cm H_2O (bottom panel), you can see the corresponding rise in pulmonary artery pressure (middle panel) and fall in systemic arterial pressure (top panel). (Courtesy Jon Nilsestuen, PhD, RRT, University of Texas at Galveston.)

Case Study 11.1

Evaluation of Pressure Tracing

The following vascular pressure-time waveform is recorded on a patient on mechanical ventilation. Answer the following questions:

1. In what vessel or heart chamber is this catheter located?
2. What is the most accurate estimate of systolic and diastolic pressure?

Pathological conditions and pharmacological interventions can significantly affect PA pressure. Pulmonary hypertension, pulmonary embolus, and congestive heart failure are associated with increased PVR, which in turn leads to increased PA systolic pressure. In contrast, inhaled nitric oxide, which selectively dilates the pulmonary vasculature, decreases PVR and PA systolic pressure (Case Study 11.1).

Cardiac Output

Cardiac output ($\dot{Q}$) is the volume of blood that is pumped by the heart per minute, and it is usually expressed in liters per minute (L/min) or milliliters per minute (mL/min). Cardiac output normally ranges from 4 to 8 L/min. It can be calculated by multiplying the heart rate by the SV. The SV is the volume of blood pumped by the heart per beat; it can be expressed in liters per beat (L/beat) or milliliters per beat (mL/beat). In many cases, the cardiac output and the SV may be reported relative to the person's body surface area (BSA), which can be easily obtained using a Dubois chart such as the one found in Fig. 6.1. This indexing technique allows the clinician to compare an individual's cardiac output or stroke output with that of normal healthy individuals of the same weight and height (BSA is calculated using these two anthropometric values).

CI is calculated by dividing the cardiac output by the BSA, or

$$\text{CI} = \frac{\text{Cardiac Output}}{\text{BSA}} = \frac{\dot{Q}}{\text{BSA}}$$

Similarly, **stroke index** (SI) is calculated by dividing SV by BSA, or

$$\text{SI} = \frac{\text{SV}}{\text{BSA}}$$

A normal CI for an adult is about 2.5 to 4.0 L/min/m^2. The SI normally ranges from 35 to 55 mL/beat/m^2 (Case Study 11.2).

Decreases in either heart rate or SV can cause reductions in cardiac output. Decreases in the effective ventricular rate are usually associated with the following:

- A decrease in sympathetic tone as occurs with the use of β-adrenergic blockade or
- An increase in parasympathetic tone, and
- The presence of various types of bradyarrhythmias

Decreases in SV are associated with reduced preload or contractility of the heart or with an abnormally high afterload. Note that tachyarrhythmias associated with high heart rates can lead to decreases in ventricular filling, which can ultimately result in reductions in cardiac output.

In contrast, increases in cardiac output are associated with increases in heart rate or SV. Increases in heart rate associated with either an increase in sympathetic tone or a decrease in parasympathetic tone will lead to an increased cardiac output. Increases in SV are associated with increases in preload and contractility and with reductions in afterload.

Fick Principle and Cardiac Output Measurements

Most experts agree that the gold standard for determining cardiac output involves direct measurements of O_2 consumption and arterial and mixed venous O_2 contents. Once these measurements are made, cardiac output can be calculated using the **Fick principle,**

$$\dot{Q} = \dot{V}O_2 / [(C_aO_2 - C\bar{v}O_2) \times 10]$$

where $\dot{Q}$ is cardiac output, $\dot{V}O_2$ is O_2 consumption, C_aO_2 is the O_2 content of arterial blood, and $C\bar{v}O_2$ is the O_2 content of mixed venous blood.

Oxygen consumption ($\dot{V}O_2$) can be derived from measurements of the fractional concentration of inspired and expired O_2 and minute ventilation. If these measurements are not available, many clinicians use an O_2 consumption of 3.5 mL/kg/min as an estimate of the person's $\dot{V}O_2$. For example, a 154-lb (70-kg) individual would have a $\dot{V}O_2$ of about 250 mL/min in the calculation of cardiac output. As you might expect, this practice may lead to erroneous results, particularly in critically ill patients.

Calculation of O_2 content requires the measurement of O_2 partial pressures and saturations for O_2 of arterial and mixed venous blood. Arterial samples can be obtained from a peripheral artery; mixed venous blood samples can only be obtained during right heart catheterization by withdrawing blood from the PA (i.e., the distal port of the PA catheter). The O_2 saturation of arterial blood (S_aO_2) is normally about 98%, and the arterial O_2 content (C_aO_2) of a normal healthy individual is approximately 20 vol% (200 mL/L of whole blood). The mixed venous O_2 saturation ($S\bar{v}O_2$) is normally about 75%, and the mixed venous O_2 content ($C\bar{v}O_2$) is about 15 vol% (150 mL/L of whole blood). Critical Care Concept 11.1 provides a sample calculation of cardiac output using the Fick principle.

Case Study 11.2

Cardiac Index and Stroke Index

A patient has a BSA = 1.7 m^2, a heart rate of 110 beats/min, and $\dot{Q}$ of 3 L/min. Calculate CI and SI. How do this patient's values compare with normal values?

CRITICAL CARE CONCEPT 11.1

Fick Principle

A 70-kg man receiving volume-targeted mechanical ventilation at an F_IO_2 of 0.35 has an O_2 consumption of 300 mL/min, a C_aO_2 of 18 vol%, and a $C\bar{v}O_2$ of 13 vol%. What is his cardiac output ($\dot{Q}$)?

See Appendix A for the answer.

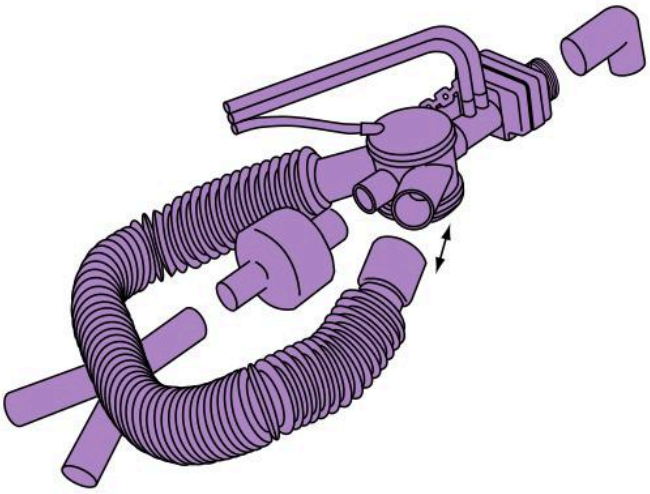


Fig. 11.12 Rebreathing circuit used by the Philips NM3 Monitor to estimate cardiac output using measurements of rebreathed carbon dioxide. (Courtesy Philips Respironics, Eindhoven, The Netherlands.)

Indirect Fick Method

A variation on this method of determining cardiac output is the indirect Fick method, which involves the collection and analysis of exhaled gas in place of arterial and mixed venous blood gas samples. With the indirect Fick method of calculating cardiac output, carbon dioxide production ($\dot{V}CO_2$) is used in place of $\dot{V}O_2$. Arterial and mixed venous O_2 difference is replaced with arterial CO_2 content and mixed venous CO_2 content, respectively. $\dot{V}O_2$ is obtained from continuous measurements of the fractional expired CO_2 (F_ECO_2) and minute ventilation ($\dot{V}_E$). The arterial CO_2 content is calculated from measurements of the partial pressure of mixed expired CO_2 ($P_{\bar{E}}CO_2$), which are obtained while the patient intermittently rebreathes a fixed volume (at 10- to 15-second intervals). Cardiac output is therefore calculated as

$$\dot{Q} = \dot{V}\ CO_2/(P_vCO_2 - P_{ET}CO_2)$$

The application of CO_2 monitoring to estimate cardiac output is currently available with the NM3 monitoring system (Philips Respironics, Eindhoven, The Netherlands; see Fig. 10.12 and Fig. 11.12).

Mixed Venous Oxygen Saturation

If the $\dot{V}O_2$ and cardiac output remain constant, the difference between the arterial O_2 content and the mixed venous O_2 content also remains constant. Mixed venous O_2 values decline when arterial oxygenation is decreased. They also decrease when cardiac output is reduced. With a reduced cardiac output, more time is available for the extraction of O_2 from blood delivered to the tissues. Reductions in $S\bar{v}O_2$ are also associated with increases in metabolic rate in patients with limited cardiac output. Mixed venous O_2 values can be higher than normal in patients with histotoxic hypoxia (e.g., cyanide poisoning) and in situations in which intrapulmonary shunting occurs (i.e., ventilation-perfusion mismatching).

With recent advances in fiberoptic reflectance oximetry, continuous recordings of $S\bar{v}O_2$ can be obtained.[23] Reflectance oximetry technology has been incorporated into specialized balloon-flotation catheters that are used for right heart catheterization. Although the potential for this type of monitoring is promising, additional studies will be required to delineate more clearly the indications for its use in critical care (Case Study 11.3).

Case Study 11.3

Application of the Fick Principle

A patient has a constant $\dot{V}O_2$ of 350 mL/min. At 13:00 hours C_aO_2 is 20 vol% and $C\bar{v}O_2$ is 14 vol%. At 15:00 hours C_aO_2 is 20 vol% and $C\bar{v}O_2$ is 12 vol%. What is one possible cause in the drop in $C\bar{v}O_2$?

Calculate the cardiac output for both times.

Oxygen Delivery

DO_2 is the product of cardiac output and arterial O_2 content. It represents the total amount of O_2 that is carried in the blood to the tissues each minute. Under normal circumstances, DO_2 is approximately 1000 mL/min or about 550 to 650 mL/min/m^2 (Key Point 11.4).[24]

***Key Point* 11.4** DO_2 represents the total amount of O_2 that is carried in the blood to the tissues each minute.

DO_2 is increased in situations in which cardiac output or arterial O_2 content is elevated. A reduced DO_2 indicates a decrease in cardiac output or arterial O_2 content.[24] For example, DO_2 is increased in hyperdynamic states (increased cardiac output) such as septic shock. Conversely, DO_2 is decreased following hemorrhage in which there is a decrease in arterial O_2 content.

Shunt Fraction

A *shunt* is defined as that portion of the cardiac output that does not participate in gas exchange with alveolar air (i.e., *perfusion without ventilation*). Shunts are usually identified as anatomical shunts, intrapulmonary shunts, and physiological shunts, with the latter being the sum of anatomical and intrapulmonary shunts.

Normal anatomical shunts exist because venous blood that would ideally return to the right side of the heart (deoxygenated blood) drains into vessels served by the left side of the heart (oxygenated blood). This venous admixture includes deoxygenated blood from bronchial veins, pleural veins, and Thebesian veins, and it typically only represents about 2% to 3% of the normal cardiac output. Abnormal anatomical shunts can occur when blood is allowed to bypass the pulmonary circulation and enter directly into the left atrium or left ventricle, as occurs with atrial and ventricular septal wall defects.

Intrapulmonary shunts occur when blood passes through pulmonary capillaries that are not ventilated. Shuntlike states can exist in either poorly ventilated alveolar units that are well perfused or in alveolar-capillary units if O_2 diffusion is impaired. Intrapulmonary shunts can be caused by disorders such as atelectasis, pulmonary edema, pneumonia, pneumothorax, complete airway obstruction, consolidation of the lung, ARDS, and on rare occasions by arterial-to-venous fistulas.

The total shunt fraction or, more specifically, **physiological shunt**, can be determined by the following classic shunt equation:

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{(C_cO_2 - C_aO_2)}{C_cO_2 - C\bar{v}O_2}$$

where $\dot{Q}_S$ is the shunted portion of the cardiac output, $\dot{Q}_T$ is total cardiac output, C_cO_2 is the content of O_2 of the pulmonary end-capillary following oxygenation of the blood, C_aO_2 is the arterial O_2 content, and $C\bar{v}O_2$ is the mixed venous O_2 content (i.e., pulmonary capillary blood before oxygenation). C_cO_2 is calculated based on the assumption that pulmonary end-capillary PO_2 is the same as P_AO_2. Mixed venous blood can be obtained from a PA catheter. As discussed later in this text, calculation of shunt fraction can be useful in the differential diagnosis of hypoxemia.

Vascular Resistance

As mentioned earlier, the vascular resistance represents the impedance or opposition to blood flow offered by the systemic and pulmonary vascular beds, and it influences the force that the ventricular muscle must generate during cardiac contractions. Although SVR and PVR have been reported historically as dyne × seconds × cm^{-5}, recent publications have tended to use the units of mm Hg/L/min. In this text, we use the units of dyne × seconds × cm^{-5} for the sake of continuity.

Taking a simple way to approach the calculation of SVR or PVR (R in the following equation), ΔP represents the pressure gradient across the vascular bed and cardiac output is the blood flow through the vascular bed, or

$$R = \frac{\Delta P}{\dot{Q}}$$

Thus SVR can be calculated as follows:

$$SVR = \left(\frac{[MAP - MRAP]}{SBF}\right) \times 80$$

where MAP is the mean aortic or arterial blood pressure, expressed in millimeters of mercury (mm Hg), MRAP is the mean right atrial pressure (in mm Hg), and SBF is the systemic blood flow or cardiac output (in L/min). Multiplying the equation by 80 is routinely used by clinicians to convert the units of mm Hg/L/min to dyne × second × cm^{-5}. Note that CVP may be substituted for MRAP. When CVP is used, the formula can be written as

$$SVR = \left(\frac{[MAP - CVP]}{\dot{Q}}\right) \times 80$$

Similarly, PVR can be calculated as

$$PVR = \left(\frac{[MPAP - MLAP]}{PBF}\right) \times 80$$

where MPAP is the mean PA pressure, MLAP is the mean left atrial pressure (both measured in mm Hg), and PBF is the pulmonary blood flow or cardiac output (CO) (in L/min). In the critical care setting, the PAOP may be used instead of MLAP, and the formula becomes

$$PVR = \left(\frac{[MPAP - PAOP]}{C.O.}\right) \times 80$$

The normal SVR ranges from 900 to 1500 dyne × seconds × cm^{-5}, and the PVR ranges from 100 to 250 dyne × seconds × cm^{-5}. The two most important factors that influence vascular resistance are the caliber of the blood vessels and viscosity of the blood. The SVR is therefore increased in left ventricular failure and hypovolemia arising from vasoconstriction caused by stimulation of the baroreceptor reflex.[16]

The SVR may also be increased if blood viscosity increases, as occurs in polycythemia. SVR decreases during systemic vasodilation, such as occurs with moderate hypoxemia or after the administration of systemic vasodilators such as nitroglycerin or hydralazine.[20]

The PVR increases during periods of alveolar hypoxia or in cases in which high intraalveolar pressures are generated, such as during positive pressure ventilation. A low cardiac output can increase PVR by causing derecruitment of pulmonary vessels. PVR is reduced by the administration of pulmonary vasodilator drugs such as tolazoline and prostacyclin.[25]

Ejection Fraction

The ejection fraction (EF) is a derived variable that provides an estimate of ventricular contractility. It is calculated by dividing the SV by the end-diastolic volume. The EF shows a positive correlation with the CI in most cases and is a valuable measurement in the prognosis of heart failure.[26] Note that the correlation between EF and CI may be inaccurate in cases of mitral regurgitation. EF values of 0.5 to 0.7 are considered normal for healthy adults. EF values lower than 0.30 are associated with compromised cardiovascular function and imminent heart failure.

Cardiac Work

In physics, *work* is defined as the product of a force acting on an object to move it a certain distance. In calculations of **cardiac work**, or more specifically **stroke work**, the pressure generated by the heart during a ventricular contraction is used to quantify the amount of force developed; the SV represents the distance portion of the equation. The amount of work performed by each ventricle during the cardiac cycle is determined by applying the following formulas:

$$LVSW = ([MAP \times SV] \times 0.0136)$$

$$RVSW = ([MPAP \times SV] \times 0.0136)$$

where LVSW and RVSW are left ventricular stroke work and right ventricular stroke work, respectively; MAP is the mean arterial pressure; MPAP is the mean PA pressure; SV represents stroke volume; and 0.00136 is a factor to convert millimeters of mercury (mm Hg)-milliliters (mL) to gram-meters (g-m). In most clinical situations, stroke work measurements are indexed to BSA. Therefore the left ventricular stroke work index (LVSWI) and right ventricular stroke work index (RVSWI) are calculated as follows:

$$LVSWI = \frac{LSW}{BSA}$$

$$RVSWI = \frac{RSW}{BSA}$$

LSWI normally ranges from 40 to 60 g-m/m^2 (0.4–0.6 kg-m/m^2), and RSWI ranges are normally between 7 and 12 g-m/m^2 (0.07–0.12 kg-m/m^2).* Conditions that increase the SV and/or

*Alternative methods for calculation of LVSW and RVSW are LVSW = SV × (BPsys − PAOP) × 0.0136; RVSW = SV (PAsys − CVP) × 0.0136, where BPsys is systolic blood pressure (systemic) and PAsys is PA systolic pressure.

Case Study 11.4

Stroke Work

A patient has a mean arterial pressure of 80 mm Hg and a stroke volume of 60 mL/beat. He is given a cardiac stimulant, and MAP increases to 100 mm Hg and SV to 70 mL/beat. His BSA is 1.5 m^2. Calculate his left ventricular stroke work index before and after delivery of the medication.

mean pressure generated by the ventricles will increase the amount of work the ventricle must perform (Case Study 11.4).

CLINICAL APPLICATIONS

Case Studies 11.5 through 11.7 present clinical cases to demonstrate the application of the concepts reviewed in this chapter. Tables 11.5 and 11.6 may assist the reader in solving the problems presented in these Case Studies.

Case Study 11.5

Hemodynamic Monitoring: After Open-Heart Surgery

A 59-year-old, 154-lb (70-kg) man is being ventilated with a Puritan Bennett 840 ventilator after open-heart surgery for a triple coronary bypass. Vital signs are stable, with a heart rate of 100 beats/min, a temperature of 37.5° C, and a blood pressure of 130/70 mm Hg. Breath sounds are normal. The tidal volume is 550 mL on VC-IMV, with no spontaneous breaths. The respiratory rate is 12 breaths/min. The F_IO_2 is 0.4. The PEEP is set at 5 cm H_2O. Peak airway pressure is 30 cm H_2O, and his pulmonary compliance is 22 mL/cm H_2O. The following data were obtained immediately after surgery:

- Hemoglobin = 13 g%
- Systemic arterial pressure = 135/70 mm Hg
- Pulmonary arterial pressure = 25/10 mm Hg
- Pulmonary artery wedge pressure = 12 mm Hg
- CVP = 2 mm Hg
- pH_a = 7.42; P_aCO_2 = 36 mm Hg; P_aO_2 = 60 mm Hg; S_aO_2 = 90%
- pH $\bar{v}$ = 7.35; $\dot{V}O_2$ = 45 mm Hg; = 40 mm Hg; $S\bar{v}O_2$ = 75%
- P_ECO_2 = 24 mm Hg; $\dot{V}O_2$ = 250 mL/min; $\dot{V}O_2$ = 200 mL/min

The surgeon asks you to increase the PEEP to 10 cm H_2O. After 20 minutes at the increased level of PEEP, the following data are obtained:

- Systemic arterial pressure = 110/65 mm Hg
- Pulmonary arterial pressure = 18/8 mm Hg
- Pulmonary artery wedge pressure = 10 mm Hg
- Central venous pressure = 4 mm Hg
- pH_a = 7.39; P_aCO_2 = 42 mm Hg; P_aO_2 = 70 mm Hg; S_aO_2 = 98%
- pH $\bar{v}$ = 7.32; P $\bar{v}O_2$ = 48 mm Hg; P $\bar{v}O_2$ = 30 mm Hg; $S\bar{v}O_2$ = 65%
- P_ECO_2 = 25 mm Hg; $\dot{V}O_2$ = 230 mL/min; $\dot{V}O_2$ = 180 mL/min

Interpret these findings.

Case Study 11.6

Hemodynamic Monitoring: Chest Injury

An 18-year-old man was admitted to the emergency department with a gunshot wound to the chest. He was transferred to the ICU status post left lower lobectomy, splenectomy, and laparoscopy, with bilateral chest tubes. The patient had bullet fragments at T12 and at the level of the left hemidiaphragm.

He was placed on high-frequency jet ventilation with the following settings:

• Breathing frequency	150 breaths/min
• Pressure	21 cm H_2O
• F_IO_2	0.50
• T_I	20%
• Peak flow	70 L/min
• Exhaled V_T	250 mL
• pH_a	7.46
• P_{aCO_2}	30.2 Torr
• P_aO_2	75.2 Torr
• HCO_3^-	21.8 mEq/L
• S_aO_2	95%
• Hb	9 g%
• $S\bar{v}O_2$	81%

The patient was receiving atracurium (9 mcg), dopamine (3 mcg/kg/min), midazolam (Versed) (3 mcg), dobutamine (6 mcg/kg/min), and morphine sulfate (8 mcg).

After insertion of a pulmonary artery catheter in the patient, the follow data were obtained:

• BSA	1.81 m^2
• Cardiac output	7.58 L/min
• Heart rate	115 beats/min
• MAP	107 mm Hg
• PAP	35 mm Hg
• PAOP	14 mm Hg
• CVP	13 mm Hg
• Hb	8.2 g%
• CI	4.19 L/min/m^2
• SV	70 mL/beat
• SVR	992 dyne × s × cm^{-5}
• PVR	222 dyne × s × cm^{-5}

How would you interpret these data?

Case Study 11.7

ICU and Hemodynamic Assessment

A 72-year-old man is admitted to the ICU, after stabilization in the emergency department (ED), on a nasal cannula at 2 L/min. Arterial blood gases (ABGs) drawn in the ED revealed pH = 7.47, P_aCO_2 = 30 mm Hg, and P_aO_2 = 31 mm Hg. The patient was immediately placed on a nonrebreathing mask, and ABGs and vital signs were assessed, with the following results:

- pH = 7.48, P_aCO_2 = 32 mm Hg, P_aO_2 = 56 mm Hg
- f = 34 breaths/min, heart rate = 116 beats/min
- Blood pressure = 175/58 mm Hg
- The patient was placed on mechanical ventilatory support with the following settings:
  - V_T = 850 mL, VC-CMV with rate = 12 breaths/min
  - F_IO_2 = 0.5, PEEP = +10 cm H_2O

Hemodynamic monitoring, after successful insertion of a pulmonary artery catheter in the subclavian artery, revealed the following data:

- Cardiac output = 7.98 L/min, CI = 4.41 L/min/m^2, HR = 81 beats/min
- BP (systolic) = 159 mm Hg, BP (diastolic) = 64 mm Hg, BP (mean) = 92 mm Hg
- PAP (systolic) = 52 mm Hg, PAP (diastolic) = 18 mm Hg, PAP (mean) = 33 mm Hg
- PAOP = 13 mm Hg, CVP (mean) = 12 mm Hg
- SV = 98.5 mL, SI = 54.4 mL/m^2, SVR = 802 dynes × cm × s^{-5}
- PVR = 201 dynes × cm^{-5} × s
- Hemoglobin = 14.5 g, temperature = 37° C
- Arterial blood gases (ABGs): pH = 7.362, PO_2 = 80 mm Hg, PCO_2 = 46.2 mm Hg, HCO_3^- = 26.5 mEq/L, S_aO_2 = 95.2%
- Mixed venous blood gases: pH = 7.339, PO_2 = 40 mm Hg, PCO_2 = 50.3 mm Hg, HCO_3^- = 27.4 mEq/L, $S\bar{v}O_2$ = 71.2%
- C_aO_2 = 18.5 vol%, $C\bar{v}O_2$ = 13.8 vol%, $C(a\text{-}\bar{v})O_2$ = 4.7 vol%
- O_2 transport = 1476 mL/min, O_2 consumption = 375 mL/min

How would you interpret these findings?

Modified with permission from Deshpande VM, Pilbeam SP, Dixon RJ: *A comprehensive review in respiratory care*, Norwalk, CT, 1988, Appleton & Lange.

TABLE 11.5 Part I: Hemodynamic Parameters That Can Be Calculated

Parameter	Normal Values	Formula	Use
Mean arterial blood pressure (MAP)	70–100 mm Hg	(Systolic pressure + Diastolic pressure)/3	To calculate systemic vascular resistance; used in hemodynamic monitoring when giving vasoactive drugs
Pulse pressure (systemic)	40 mm Hg	Systolic pressure − Diastolic pressure	To estimate the force of the pulse
Stroke volume (SV)	60–100 mL	$\dot{Q}$/heart rate	Provides information about cardiac performance
Cardiac index (CI)	2.5–4 L/min/m^2	$\dot{Q}$ body surface area (BSA)	An important determinant of cardiac performance (removes body size as a variable)
Stroke index (SI)	35–55 mL/beat m^2	SV/BSA	An important determinant of cardiac performance (removes body size as a variable)
Systemic vascular resistance (SVR)	900–1500 dyne × s × cm^{-5}	([MAP − CVP]/CO) × 80	To measure resistance in system circulation; useful in diagnosis of vascular problems
Mean pulmonary artery pressure (MPAP)	10–20 mm Hg	Pulmonary systolic pressure + Pulmonary diastolic pressure	To calculate pulmonary vascular resistance
Pulmonary vascular resistance (PVR)	100–250 dyne × s × cm^{-5}	([MPAP −PAOP] /$\dot{Q}$) × 80	To measure resistance in the pulmonary vascular bed; useful in the diagnosis of pulmonary vascular problems
Oxygen content of arterial blood (C_aO_2)	20 vol%	([S_aO_2 × Hb] × 1.34)[a]	To calculate O_2 delivery, cardiac output, and shunt fraction
Oxygen content of mixed venous blood	15 vol%	([$S\bar{v}O_2$ × Hb] × 1.34)[a]	To calculate cardiac output and shunt fraction
Arterial-to-venous oxygen content difference	3.5–5.0 mL/100 mL or vol%	$C(a\text{-}\bar{v})O_2$	Index of tissue oxygenation
Oxygen transport (DO_2)	500–1000 mL/min[b]	$\dot{Q}$ × CaO_2	Indicates the amount of oxygen delivered to the tissues

Continued

TABLE 11.5 Part I: Hemodynamic Parameters That Can Be Calculated—cont'd

Oxygen consumption ($\dot{V}O_2$)	200–300 mL/min	$\dot{Q} \times (CaO_2 - C\,\bar{v}O_2)$	Indicates the metabolic rate (i.e., the amount of O_2 used by the body); this can be measured indirectly by noninvasive means but only with great difficulty

Part II: Hemodynamic Parameters That Can Be Measured Directly

Parameter	Normal Values	How Measured	Use
Heart rate (HR)	60–100 beats/min	Pulse rate	Early index of tachycardia and bradycardia
Blood pressure (systemic) (BP)	*Systolic:* 90–140 mm Hg *Diastolic:* 60–90 mm Hg	Blood pressure cuff or arterial line	Early index of hypertension or hypotension
Central venous pressure (CVP)	2–6 mm Hg	From CVP catheter or PA 3- or 4-lumen catheter	To estimate right ventricular preload; also for drug and fluid administration
Pulmonary artery pressure (PAP)	*Systolic:* 15–35 mm Hg *Diastolic:* 5–15 mm Hg	From PA catheter	To determine PAP and to pressure PVR
Pulmonary artery occlusion pressure (PAOP)	5–12 mm Hg	From PA catheter in the occluded position (balloon inflated)	To estimate left ventricular filling and preload
Cardiac output (CO)	4–8 L/min	By thermodilution or dye dilution	An important determinant of hemodynamic function
Partial pressure of oxygen in mixed venous blood $P(\bar{v}O_2)$	40 mm Hg	From blood from the distal port of the PA catheter	Overall parameter for assessment of cardiopulmonary function
Partial pressure of oxygen in arterial blood (P_aO_2)	80–100 mm Hg	From a systemic artery	To assess level of arterial oxygenation

[a]Dissolved portion is small and not included here (Dissolved $O_2 = 0.0031 \times P_aO_2$ or $P\,\bar{v}O_2$).
[b]$CO \times C_aO_2 = 5000$ mL/min $\times$ 20 mL/100 mL $= 1000$ mL/min.

TABLE 11.6 Hemodynamic Changes Commonly Seen in Respiratory Diseases

	HEMODYNAMIC INDICES											
Disorder	**CVP**	**RAP**	**PAP**	**PAOP**	**CO**	**SV**	**SVI**	**CI**	**RVSWI**	**LVSWI**	**PVR**	**SVR**
Chronic bronchitis	↑	↑	↑↑	—	—	—	—	—	↑	—	↑	—
Emphysema												
Bronchiectasis												
Cystic fibrosis												
Pulmonary edema (cardiogenic)	—	↑	↑	↑↑	↓	↓	↓	↓	↑	↓	↑	↓
Pulmonary embolism	↑	↑	↑↑	↓	↓	↓	↓	↓	~↑	↓	~↑	~
Acute respiratory distress syndrome (ARDS)—severe	~↑	~↑	~↑	~	~	~	~	~	~↑	~	~↑	~
Lung collapse	↑	↑	↑	↓	↓	↓	↓	↓	↑	↓	↑	↓
Flail chest												
Pneumothorax												
Pleural disease (e.g., hemothorax)												
Kyphoscoliosis	↑	↑	↑	~	~	~	~	~	↑	~	↑	~
Pneumoconiosis	↑	↑	↑↑	~	~	~	~	~	↑	~	↑	~
Chronic interstitial lung diseases	↑	↑	↑↑	~	~	~	~	~	↑	~	↑	~
Lung cancer (tumor mass)	↑	↑	↑	↓	↓	↓	↓	↓	↑	↓	↑	~
Hypovolemia	↓↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	~	↑
Hypervolemia (burns)	↑↑	↑	↑	↑	↑	↑	↑	↑	↑	↑	↑	~
Right heart failure (cor pulmonale)	↑↑	↑↑	↓	↓	~	~	~	~	~	~	~	~

↑, increase; ↓, decrease; ~, unchanged; *CI,* cardiac index; *CO,* cardiac output; *CVP,* central venous pressure; *LVSWI,* left ventricular stroke work index; *PAOP,* pulmonary artery occlusion pressure; *PAP,* pulmonary artery pressure; *PVR,* pulmonary vascular resistance; *RAP,* right atrial pressure; *RVSWI,* right ventricular stroke work index; *SV,* stroke volume; *SVI,* stroke volume index; *SVR,* systemic vascular resistance.
From Des Jardins T, Burton GG: *Clinical manifestations and assessment of respiratory disease,* ed 8, St. Louis, MO, 2019, Elsevier.

SUMMARY

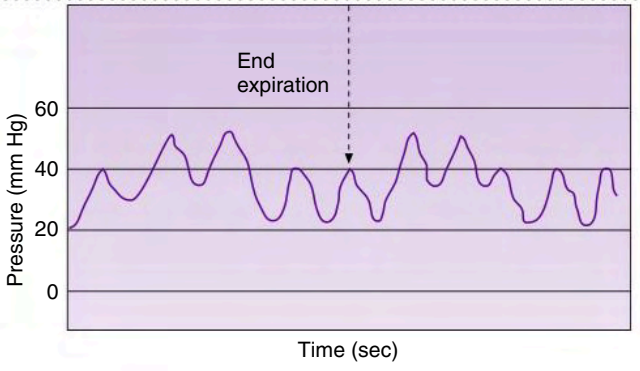
- Hemodynamic monitoring can provide a window to observe the effects of various physiological and pharmacological interventions on cardiovascular function.
- The effective use of hemodynamic monitoring requires knowledge of the basic principles of cardiovascular physiology and an understanding of the physical and technical factors that can influence the measurement conditions.
- Cardiac output is primarily influenced by heart rate and ventricular preload, contractility, and afterload.
- CVP and PAOP can be used to assess right and left ventricular preload, respectively.
- Ejection fraction is a valuable clinical indicator of ventricular contractility.
- The SVR and PVR are used clinically to describe the afterload to the left and right ventricles, respectively.
- Positioning of a PA catheter can be accomplished by fluoroscopy or by monitoring the pressure tracing generated as the catheter is advanced into the right heart and PA.
- Accurate measurements of the PAOP require wedging of the PA catheter in the zone 3 portion of the pulmonary vasculature.
- Pulse pressure is the difference between the systolic and diastolic pressure and is influenced by the stroke volume and arterial compliance.
- Cardiac index and stroke index allow the clinician to compare an individual's cardiac output and stroke output with those of healthy individuals of the same weight and height.
- Cardiac index and ejection fraction are important variables used in determining the prognosis of heart failure.
- Cardiac work is primarily influenced by stroke volume and systolic arterial pressure.
- Calculation of O_2 contents requires the measurement of O_2 partial pressures and saturations in arterial and mixed venous blood.
- Oxygen delivery to the tissues is increased in situations in which the cardiac output and arterial O_2 content are elevated. It is typically decreased when either of these variables is reduced.
- SVR and PVR represent the impedance or opposition to blood flow offered by the systemic and pulmonary vascular beds, respectively. Vascular resistance influences the force that the ventricular muscle must generate during cardiac contractions.
- Cardiac work provides an estimate of the amount of force that must be generated by the ventricles to achieve a given stroke output.

REVIEW QUESTIONS *(See Appendix A for answers.)*

1. Tracings from a patient undergoing cardiac catheterization demonstrated a left ventricular systolic pressure of 180 mm Hg and a peak systolic aortic pressure of 110 mm Hg. The patient complained of shortness of breath, fatigue, and syncope (loss of consciousness). Which of the following would you associate with these findings?
A. Aortic stenosis
B. Mitral regurgitation
C. Pulmonary stenosis
D. Tricuspid insufficiency

2. Which of the following is *incorrectly* matched for a resting healthy 154-lb (70-kg), 25-year-old sedentary person?
A. Peak systolic left ventricular pressure = 120 mm Hg
B. Mean right atrial pressure = 5 mm Hg
C. Left ventricular stroke volume = 120 mL
D. Left ventricular end-systolic volume = 50 mL

3. The following tracing was obtained during the placement of a pulmonary artery catheter. The contour of the tracing suggests that the catheter is in the:
A. Right atrium
B. Right ventricle
C. Pulmonary artery
D. Pulmonary wedge position



4. For a heart rate of 75 beats/min, the cardiac cycle will last approximately _____ seconds.
A. 0.4
B. 0.8
C. 1.0
D. 1.2

5. Which of the following conditions will cause a decrease in cardiac output?
A. Exercise
B. Hypovolemia
C. Increased sympathetic tone
D. Fever

6. Which of the following is a characteristic finding in patients with hypovolemia?
   A. Elevated PAOP
   B. Low RAP
   C. Decreased heart rate
   D. Increased pulmonary artery pressure
7. Which of the following will cause an elevation in pulmonary artery pressure?
   A. Hemorrhage
   B. Fluid overload
   C. Administering nitric oxide
   D. Breathing an enriched oxygen mixture
8. Which of the following measurements is a good indicator of left ventricular contractility?
   1. dP/dT
   2. Ejection fraction
   3. Stroke volume
   4. LVEDV
      A. 1 and 2 only
      B. 1 and 3 only
      C. 1, 2, and 3 only
      D. 2, 3, and 4 only
9. Which of the following variables are required to calculate PVR?
   1. Cardiac output
   2. Mean left atrial pressure
   3. Mean pulmonary artery pressure
   4. Mean right atrial pressure
      A. 1 and 2 only
      B. 1 and 3 only
      C. 1, 2, and 3 only
      D. 1, 2, and 4 only
10. Which of the following will typically lead to a *decrease* in cardiac output?
    A. Increase in preload
    B. Increase in afterload
    C. Increase in contractility
    D. Increase in heart rate
11. When properly inserted, the proximal lumen of the pulmonary artery catheter will be positioned in the:
    A. Right atrium
    B. Right ventricle
    C. Pulmonary artery
    D. Left atrium
12. The proximal lumen can be used for all of the following *except:*
    A. Monitoring of RA pressure
    B. Fluid administration
    C. Cardiac output injectate insertion
    D. Monitoring of wedge pressures
13. The primary function of the transducer dome in a fluid-filled system is to:
    A. Amplify the weak biological signal
    B. Filter clots from the system
    C. Convert a pressure signal into an electrical signal
    D. Respond to pressure changes in the fluid column
14. If the transducer level is lower than the tip of the catheter during pulmonary artery pressure monitoring,
    A. The readings will be falsely high
    B. An overwedged waveform will appear
    C. The waveform will be damped
    D. Catheter whip will appear on the waveform
15. The dicrotic notch on the pulmonary artery waveform may disappear in all of the following conditions *except:*
    A. Systemic vasoconstriction
    B. Pulmonary vasodilation
    C. Measurements obtained from a femoral artery
    D. Pulmonary stenosis
16. Left ventricular stroke work is decreased by increases in which of the following?
    A. Mean aortic pressure
    B. Ventricular end-diastolic pressure
    C. Heart rate
    D. Systemic vasodilation
17. Pulmonary hypertension will have which of the following effects?
    A. Increase afterload of the left side of the heart
    B. Increase afterload of the right side of the heart
    C. Decrease preload of the right side of the heart
    D. No effect on myocardial function
18. If a patient has a cardiac output of 5.6 L/min and a BSA of 2.1 m^2, what is the patient's CI?
    A. 2.67 L/min/m^2
    B. 3.50 L/min/m^2
    C. 7.70 L/min/m^2
    D. 11.76 L/min/m^2
19. Which of the following statements is true regarding the effects of mechanical ventilation on hemodynamic measurements?
    A. Lower mean inspiratory pressures present with PSV minimize the effects of positive intrathoracic pressure
    B. Applied PEEP should be discontinued when making PAOP measurements
    C. PC-IRV is associated with increases in CI and DO_2
    D. PAOP should be measured at the end of a quiet inspiration
20. Which of the following could be used to estimate left ventricular end-diastolic pressure?
    1. PAOP
    2. PA diastolic pressure
    3. PA systolic pressure
    4. RV systolic pressure
       A. 1 and 2 only
       B. 2 and 3 only
       C. 3 and 4 only
       D. 1, 2, 3, and 4

References

1. Bernard GR, Sopko G, Cerra F, et al.: Pulmonary artery catheterization and clinical outcomes, *JAMA* 19:2568–2572, 2000.
2. Boron WF, Boulpaep GL: *Medical physiology*, ed 3, Philadelphia, PA, 2016, Saunders.
3. Cairo JM: *Mosby's respiratory care equipment*, ed 11, St. Louis, MO, 2022, Elsevier.
4. Wiedeman HP, Matthay MA, Matthay RA: Cardiovascular-pulmonary monitoring in the intensive care unit. Part 1, *Chest* 85:537–549, 1984.
5. Wiedeman HP, Matthay MA, Matthay RA: Cardiovascular-pulmonary monitoring in the intensive care unit. Part 2, *Chest* 85:656–668, 1984.
6. Agee KR, Balk RA: Central venous catheters in the critically ill patient, *Crit Care Clin* 8:677–686, 1992.

7. Swan HJC, Ganz W, Forrester J, et al.: Catheterization of the heart in man with the use of a flow-directed balloon tipped catheter, *N Engl J Med* 75:83—89, 1975.
8. Mathews L, Singh RK: Swan-Ganz catheter in hemodynamic monitoring, *J Anaesth Clin Pharmacol* 22:335—345, 2006.
9. Kacmarek RM, Stoller JK, Heuer AJ: *Egan's fundamentals of respiratory care*, ed 12, St, Louis, MO, 2021, Elsevier.
10. Brierre SP, Summer W, Happel KI, et al.: Interpretation of pulmonary artery catheter tracings, *Clin Pulmon Med* 9:335—341, 2002.
11. Marini J: Hemodynamic monitoring with the pulmonary artery catheter, *Crit Care Clin* 3:551—572, 1986.
12. Pilbeam SP: *Mechanical ventilation: physiological and clinical applications*, ed 3, St. Louis, MO, 1998, Mosby.
13. DesJardins T, Burton GG: *Clinical manifestations and assessment of respiratory disease*, ed 8, St. Louis, MO, 2019, Mosby.
14. Rubenstein JS, Hageman JR: Monitoring of critically ill infants and children, *Crit Care Clin* 4:621—639, 1988.
15. Morhman DE, Heller LJ: *Cardiovascular physiology*, ed 9, New York, NY, 2018, McGraw-Hill.
16. Rushmer RF: *Cardiovascular dynamics*, ed 4, Philadelphia, PA, 1976, WB Saunders.
17. Hoyt JD, Leatherman JW: Interpretation of pulmonary artery occlusion pressure in mechanically ventilated patients with large respiratory excursions in intrathoracic pressure, *Intensive Care Med* 23:1125—1131, 1997.
18. Daily EK: Hemodynamic waveform analysis, *J Cardiovasc Nurs* 15:6—22, 2001.
19. Murphy BA, Durbin CG: Using ventilator and cardiovascular graphics in the patient who is hemodynamically unstable, *Respir Care* 50:262—274, 2005.
20. Sternberg R, Sahebjami H: Hemodynamic and oxygen transport characteristics of common ventilator modes, *Chest* 105:1798—1803, 1994.
21. Chan K, Abraham E: Effects of inverse ratio ventilation on cardiorespiratory parameters in severe respiratory failure, *Chest* 102:1556—1561, 1992.
22. Mercat A, Graïni L, Teboul JL, et al.: Cardiorespiratory effects of pressure-controlled ventilation with and without inverse ratio in the adult respiratory distress syndrome, *Chest* 104:871—875, 1993.
23. Pinsky MR, Teboul JL, Vincent JL: *Hemodynamic monitoring*, Danvers, MA, 2019, Springer.
24. Wilkins RL, Dexter JR, Heuer AJ: *Clinical assessment in respiratory care*, ed 6, St. Louis, MO, 2009, Mosby.
25. Brunton L, Knollmann B, Hilal-Dandan R: *Goodman and Gilman's the pharmacological basis of therapeutics*, ed 13, New York, NY, 2017, McGraw-Hill.
26. Libby P, Bonow RO, Mann DL, et al.: *Braunwald's heart disease: a textbook of cardiovascular medicine*, ed 11, Philadelphia, PA, 2018, Elsevier.

CHAPTER 12

Methods to Improve Ventilation in Patient-Ventilator Management

OUTLINE

KEY TERMS

- Alveolar dead space
- Asynchrony
- Hyperosmolar
- Iatrogenic
- Ischemic
- Ketoacidosis
- Minute ventilation
- Permissive hypercapnia
- Transpyloric

LEARNING OBJECTIVES

On completion of this chapter, the reader will be able to do *the following:*

1. Recommend ventilator adjustments to reduce work of breathing and improve ventilation based on patient diagnosis, arterial blood gas results, and ventilator parameters.
2. Calculate the appropriate suction catheter size, length, and amount of suction pressure needed for a specific size endotracheal tube and patient.
3. Compare the benefits of closed-suction catheters with the open-suction technique.
4. List the pros and cons of instilling normal saline to loosen secretions before suctioning.
5. List the clinical findings used to establish the presence of a respiratory infection.
6. Compare and contrast the protocols for using metered-dose inhalers and small-volume nebulizers during mechanical ventilation.
7. Describe complications associated with using small-volume nebulizers powered by external flowmeters during mechanical ventilation.
8. Discuss the importance of patient-centered mechanical ventilation in the treatment of critically ill patients.
9. Discuss the complications associated with the in-house transport of a mechanically ventilated patient.

Clinicians generally use the first 30 to 60 minutes after initiation of mechanical ventilation to gather information that can be used to evaluate the effectiveness of ventilatory support. As discussed in Chapter 8, these data typically involve vital signs, breath sounds, and assessment of respiratory mechanics (i.e., lung compliance [C_L] and airway resistance [R_{aw}]). Ventilator graphics can also be a valuable resource when evaluating the patient–ventilator interaction (see Chapter 9).

This chapter provides an overview of ventilatory strategies that can be used to manage patients with various acid-base disturbances. It also includes a discussion of airway clearance techniques, aerosol administration, flexible fiberoptic bronchoscopy during ventilation, patient positioning, and techniques used to assess fluid balance. The importance of ensuring patient comfort and safety, in addition to in-house transport of the patient on ventilation, is also discussed.

CORRECTING VENTILATION ABNORMALITIES

Once an initial physical assessment is performed, an arterial blood gas (ABG) sample should be obtained to evaluate the patient's respiratory and acid-base status. Evaluation of ABG results can be divided into three parts: acid-base status, ventilation, and oxygenation status—pH (alkalinity and acidity), and bicarbonate; P_aCO_2 (partial pressure of carbon dioxide [CO_2]); and oxygenation status (P_aO_2 [partial pressure of O_2], S_aO_2 [arterial O_2 saturation], C_aO_2 [arterial content of O_2], and O_2 delivery [DO_2]). The following discussion focuses on those factors that can alter P_aCO_2 during mechanical ventilation, including **minute ventilation**, physiological dead space, and CO_2 production (Fig. 12.1). Methods to improve oxygenation are reviewed in Chapter 13.

COMMON METHODS OF CHANGING VENTILATION BASED ON P_aCO_2 AND pH

A change in minute ventilation ($\dot{V}_E$) is often required after a patient is placed on mechanical ventilation. It is not uncommon to use full ventilatory support initially and then adjust parameters after an initial assessment is performed. The examples presented in this discussion represent full support of an apneic patient.

During mechanical ventilation, changing the set tidal volume (V_T) or rate (*f*) can be used to correct respiratory alkalosis or acidosis. These changes are based on the following equation*:

$$\text{known } P_aCO_2 \times \text{known } \dot{V}_E = \text{Desired } P_aCO_2 \times \text{Desired } \dot{V}_E$$

If we assume that physiological dead space† and CO_2 production (resulting from metabolism) do not change significantly during a short period, the equation can be modified to read:

$$\text{Known } P_aCO_2 \times \text{Known alveolar ventilation per minute } (\dot{V}_A) =$$

$$\text{Desired } P_aCO_2 \times \text{Desired } V_A$$

If it is appropriate to keep rate (*f*) constant and change V_T, the equation becomes:

$$\text{Desired } V_T = \frac{\text{Known } P_aCO_2 \times \text{Known } V_T}{\text{Desired } P_aCO_2}$$

If it is appropriate to keep V_T constant and change *f*, the equation is written:

$$\text{Desired f} = \frac{\text{Known } P_aCO_2 \times \text{Known f}}{\text{Desired } P_aCO_2}$$

Respiratory Acidosis: Volume and Pressure Ventilation Changes

When P_aCO_2 is elevated (>45 mm Hg) and pH is decreased (<7.35), respiratory acidosis is present and $\dot{V}_A$ is inadequate. Acute respiratory acidosis is associated with the following:

- Parenchymal lung problems (e.g., pulmonary edema, pneumonia)
- Airway disease (e.g., severe asthma attack)
- Pleural abnormalities (e.g., effusions)
- Chest wall abnormalities
- Neuromuscular disorders (e.g., spinal cord injury, myasthenia gravis)
- Central nervous system problems (e.g., drug overdose)[1]

See Chapter 4 for additional information.

For patients receiving mechanical ventilation, respiratory acidosis can generally be corrected by adjusting the set V_T or *f* to achieve the desired minute ventilation. Regardless of whether the patient is receiving volume-controlled or pressure-controlled ventilation, increasing $\dot{V}_E$ will decrease the P_aCO_2.

Recommended guidelines are to target a V_T of 6 to 8 mL/kg ideal body weight (IBW) while ensuring that the plateau pressure (P_{plat}) is maintained at less than 30 cm H_2O. As discussed in Chapter 6, V_T and breathing frequency adjustments should be based on the patient's pulmonary pathology. It is important to understand that the 6 to 8 mL/kg range is an average. If the V_T is at

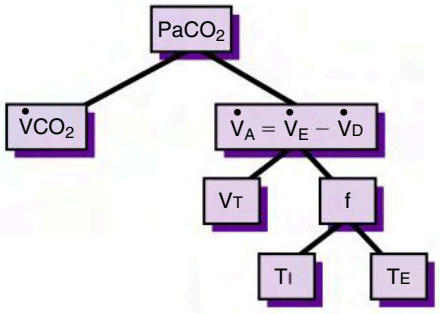


Fig. 12.1 Factors that affect the partial pressure of arterial carbon dioxide (P_aCO_2) during mechanical ventilation. $\dot{V}CO_2$, Carbon dioxide production; $\dot{V}_A$, alveolar ventilation; $\dot{V}_E$, minute ventilation; $\dot{V}_D$, dead space ventilation; V_T, tidal volume; T_I, inspiratory time; T_E, expiratory time; *f*, respiratory rate. (From Hess DR, MacIntyre NR, Mishoe SC, et al.: *Respiratory care principles and practice,* ed 2, Sudbury, MA, 2012, Jones and Bartlett.)

*This equation is based on the following: $\dot{V}_E$, where $\dot{V}_A$ is alveolar ventilation, 0.863 is a conversion factor, and $\dot{V}co_2$ is CO_2 production. In addition, $\dot{V}co_2$. Assuming dead space and $\dot{V}co_2$ remain constant, increasing ventilation results in a decreased CO_2.

†Physiological dead space = Anatomical dead space + Alveolar dead space.

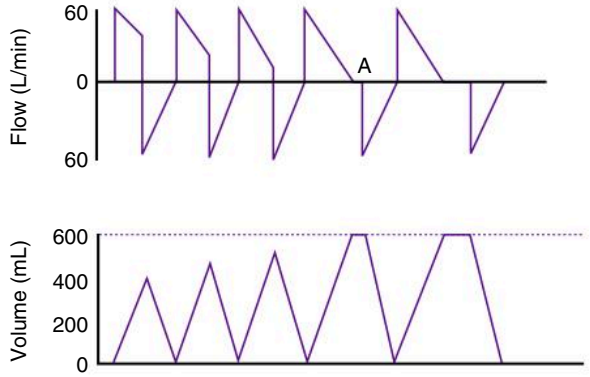


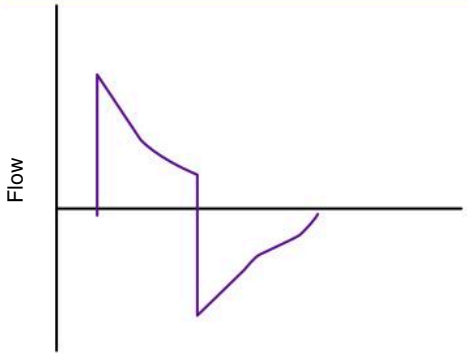
Fig. 12.2 Effect of inspiratory time (T_I) and inspiratory plateau on delivered tidal volume (V_T) in the pressure-controlled ventilation (PC-CMV) mode. Initially, as T_I is increased, V_T increases. Once an inspiratory plateau *(A)* is reached, a further increase in T_I will not increase V_T. Kacmarek RM, Stoller JK, Heuer AJ: *Egan's fundamentals of respiratory care,* ed 12, St. Louis, MO, 2021, Elsevier.

the upper limit of this range and P_{plat} is greater than 30 cm H_2O, V_T may be decreased to achieve a lower P_{plat}.

With pressure-controlled continuous mandatory ventilation (PC-CMV), the set pressure is generally adjusted to obtain the targeted V_T. PC-CMV is time cycled. If inspiratory time (T_I) is short, increasing it may also increase volume delivery, without requiring an increase in pressure (P) (Fig. 12.2).

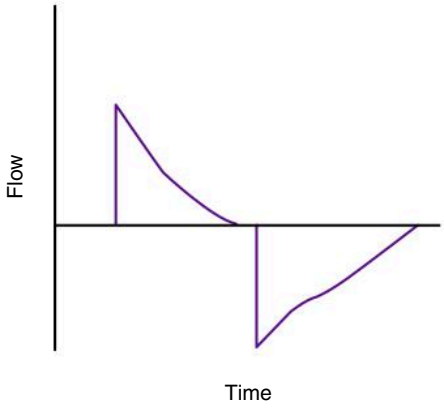
Clinical Scenario: Adjusting PC-CMV in a Patient With Respiratory Acidosis

A 165-lb (75-kg, IBW) patient is placed on PC-CMV with a rate of 10 breaths/min, a set pressure of 25 cm H_2O, and a measured V_T of 425 mL. The flow-time curve for this patient is shown in *Curve A*. T_I is 0.7 seconds. The ABGs are as follows: pH = 7.30; P_aCO_2 = 50 mm Hg; HCO_3^- = 23 mEq/L.



Curve A

To improve ventilation, two things can be adjusted. The T_I can be increased so that the set pressure reaches the alveolar level. This would be apparent when the flow scalar shows a period of zero flow during inspiration *(curve B)*. This strategy may be tried first. If this does not improve volume delivery sufficiently, the set pressure can be increased.



Curve B

Clinical Scenario: Respiratory Acidosis—Increasing Tidal Volume

A 50-year-old man with respiratory acidosis is receiving mechanical ventilatory support. He is 6-ft, 2-in tall and weighs 190 lb (81 kg, IBW) (anatomical dead space [V_{Danat}] = 190 mL). Exhaled V_T measured at the endotracheal tube (ET) is 400 mL. His respiratory rate is 16 breaths/min. The patient is receiving VC-CMV. ABGs show a P_aCO_2 of 55 mm Hg and a pH of 7.33. The patient has no pulmonary disease. His desired P_aCO_2 is 40 mm Hg. What ventilator change must be made to decrease the P_aCO_2?

Because the V_T setting is less than 8 mL/kg and the patient has no pulmonary disease, it is appropriate to hold the rate constant and change V_T:

$$\text{Desired } V_T = \frac{\text{Known } P_aCO_2 \times \text{Known } V_T}{\text{Desired } P_aCO_2}$$

$$\text{Desired } V_T = \frac{55 \times 400}{400} = 550 \text{ mL}$$

The new V_T is set at 550 mL (~7 mL/kg IBW) to achieve a desired P_aCO_2 of 40 mm Hg. The alveolar ventilation has been increased appropriately to correct the respiratory acidosis.

Clinical Scenario: Respiratory Acidosis—Increasing Rate

A 48-year-old woman who is 5-ft, 2-in tall is receiving VC-CMV and generating no spontaneous breaths. She has a P_aCO_2 of 58 mm Hg, pH is 7.28, and V_T at the ET is 425 mL. IBW is 115 lb (52 kg), and V_{Danat} is 115 mL. Respiratory rate is 15 breaths/min. How can the ventilator settings be adjusted to achieve a desired P_aCO_2 of 40 mm Hg?

In this case the patient's V_T is set at about 8 mL/kg, so it is appropriate to change *f* and not increase V_T.

$$\text{Desired f} = \frac{\text{Known f} \times \text{Known } P_aCO_2}{\text{Desired } P_aCO_2}$$

Increasing the breathing frequency to 23 breaths/min should decrease the P_aCO_2 to 40 mm Hg. It is important to recognize, however, that setting high respiratory rates may be associated with air trapping because of a reduced expiratory time. (NOTE: It is useful to monitor the flow scalar when setting breathing frequencies greater than 20 breaths/min.)

Clinical Scenario: Changes During Pressure-Targeted Ventilation

During PC-CMV, the same types of adjustments for V_T and f are made on the basis of ABGs, except that the set pressure is increased or decreased to achieve a desired V_T. Exhaled volume is monitored until the desired V_T is obtained. (NOTE: It is important to ensure that T_I is long enough to gain the most benefit from the set pressure [see Case Study 12.1].)

A 165-lb (75-kg, IBW) patient with respiratory acidosis is receiving PC-CMV ($P_aCO_2 = 59$ mm Hg; pH = 7.31; desired $P_aCO_2 = 40$ mm Hg). At a set pressure of 10 cm H_2O, the exhaled V_T is 400 mL (5.3 mL/kg) and the rate is 16 breaths/min. The patient is not spontaneously breathing. T_I is set so that the flow returns to zero before the end of inspiration and a P_{plat} period can be measured. What ventilator adjustment must be made to decrease his P_aCO_2? The V_T setting is less than 8 mL/kg, so it is appropriate to hold f constant and change V_T.

$$\text{Desired } V_T = \frac{59 \times 400}{40} = 590 \text{ mL or about } 600 \text{ mL}$$

A set pressure (P) of 10 cm H_2O results in a V_T of 400 mL (0.4 L); what is an estimated pressure that will achieve a V_T of 600 mL (0.6 L)? Assume that compliance does not change. Remember static compliance (C_S) = $V_T/(P_{plat} - \text{positive end-expiratory pressure [PEEP]})$, $C_S = 400/10$, and $C_S = 40$ mL/cm H_2O

$$\text{Desired P} = \frac{\text{Desired } V_T}{C_s}$$

$$\text{Desired P} = \frac{600 \text{ mL}}{\left(\frac{40 \text{ mL}}{\text{cm } H_2O}\right)} \text{ and P} = 15 \text{ cm } H_2O$$

Desired pressure is 15 cm H_2O

Increasing pressure during PC-CMV will normally increase V_T. Conversely, decreasing pressure decreases V_T for patients with respiratory alkalosis during PC-CMV.

Case Study 12.1

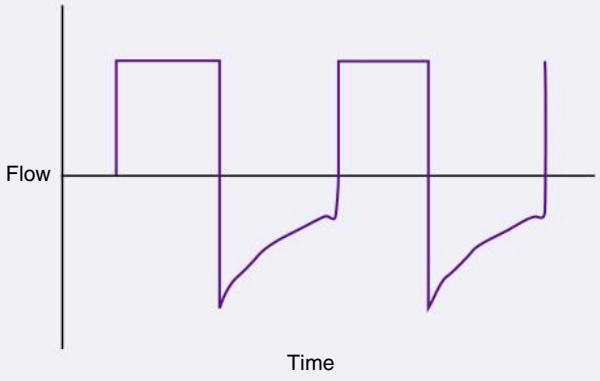
Hyperventilation

A 190-lb (86-kg), 68-year-old man (IBW = 80 kg) is admitted to the hospital. He is placed on mechanical ventilation for acute respiratory failure compounded by metabolic acidosis. It is determined that he has a renal disorder. The physician orders peritoneal dialysis. In the interim, the physician asks the respiratory therapist to target a pH of 7.35 with assisted ventilation. Initial assessment of the patient resulted in the following ABG: pH = 7.22, $P_aCO_2 = 38$ mm Hg, $HCO_3^- = 15$ mEq/L, $P_aO_2 = 98$ mm Hg on $F_IO_2 = 0.25$.

The ventilator settings are VC-CMV, $f = 24$ breaths/min, $V_T = 600$ mL, $F_IO_2 = 0.25$.

ABG results on this new setting are pH = 7.37, P_aCO_2 mm Hg = 23, $HCO_3^- = 13.5$ mEq/L, $P_aO_2 = 115$ mm Hg.

A flow-time graphic shows the following:



What is the problem, and what would you suggest to correct this problem and still help the patient?

Respiratory Alkalosis: VC-CMV and PC-CMV Changes

When P_aCO_2 is decreased (<35 mm Hg) and pH increases (>7.45), respiratory alkalosis is present, indicating that the alveolar ventilation is excessive. Common causes of respiratory alkalosis include the following[2]:

- Hypoxia with compensatory hyperventilation
- Parenchymal lung disease
- Medications (salicylate, xanthines, analeptics)
- Mechanical ventilation
- Central nervous system disorders (meningitis, encephalitis, head trauma)
- Anxiety
- Metabolic problems (sepsis, hepatic disease)

In mechanically ventilated patients, hyperventilation is often the cause of respiratory alkalosis. To correct respiratory alkalosis in this situation, the clinician should decrease minute ventilation during volume-controlled ventilation by decreasing f and, if necessary, decreasing V_T. If pressure-controlled ventilation is used, the clinician should decrease f first and then decrease set pressure, if necessary.

Clinical Scenario: Respiratory Alkalosis—Decreasing the Rate

A 35-year-old man with respiratory alkalosis (P_aCO_2 = 20 mm Hg; pH = 7.60) is on CMV with no spontaneous breaths and a delivered V_T of 550 mL, a rate of 18 breaths/min, and V_{Danat} at 150 mL. His desired P_aCO_2 is 40 mm Hg. IBW is 70 kg. V_T setting is approximately 8 mL/kg. It is appropriate to decrease f and leave V_T constant:

$$\text{Desired f} = \frac{18 \times 20}{40} = 9 \frac{\text{breaths}}{\text{min}}$$

A new rate of 9 breaths/min should achieve the desired P_aCO_2 of 40 mm Hg. (NOTE: This case did not state whether this was volume-controlled or pressure-controlled ventilation. It does not matter in this situation, however, because the targeted change was the rate.)

Clinical Scenario: Respiratory Alkalosis: Decreasing the Volume in Pressure-Controlled Ventilation

A 50-year-old woman on PC-CMV originally had a set pressure of 20 cm H_2O, which resulted in a V_T of 450 mL (C_S = 28 mL/cm H_2O). The set rate was 12 breaths/min with no spontaneous efforts. T_I is long enough to provide a slight pause (zero flow) at end inspiration. ABGs on this setting showed a pH = 7.41 and a P_aCO_2 of 44 mm Hg. The patient's IBW is 60 kg.

After 2 days of ventilation on these settings, the pH = 7.51 and P_aCO_2 = 29 mm Hg. The pressure is still at 20 cm H_2O and V_T is 625 mL (10.4 mL/kg IBW). C_S is now 32 mL/cm H_2O. This is a substantial improvement. The V_T is high, based on IBW. What would be an appropriate target volume for this patient?

$$\text{Desired } V_T = \sim 450 \text{ mL}$$

How do you set a pressure to get this V_T?

$$\text{Remember } C_s = V_T/\Delta P, \text{ so } P = V_T/C_s$$

and

$$\text{Desired P} = 450 \text{ mL}/(32 \text{ mL / cm } H_2O) = 14 \text{ cm } H_2O$$

A decrease in pressure to 14 cm H_2O should achieve the new targeted V_T of 450 mL, which is 7.5 mL/kg IBW for this patient.

Clinical Scenario: Respiratory Alkalosis During Spontaneous Efforts

A 25-year-old man on VC-CMV has a set rate of 14 breaths/min and a V_T of 560 mL. The patient's IBW is 80 kg (176 lb). The patient is triggering the ventilator an additional 4 breaths/min. Total rate is 18 breaths/min; P_aCO_2 is 30 mm Hg; pH is 7.50. What can the respiratory therapist do to improve ventilation? The V_T is 7 mL/kg IBW, which is appropriate for this patient. Suppose the therapist reduces the set *f* on the ventilator to 10 breaths/min. What effect would this have on ventilation? As long as the patient continues to trigger additional breaths, reducing the set *f* will have no effect.

Decreasing V_T (or, more specifically, pressure during PC-CMV) may be effective in reducing minute ventilation unless the patient increases his spontaneous rate, thus maintaining high alveolar ventilation. In addition, lowering the V_T to less than 7 mL/kg may result in atelectasis from low V_T ventilation.

In this situation, several alternatives are available: (1) reduce the V_T but ensure an adequate level of PEEP is being administered to reduce the potential for atelectasis; (2) institute another mode, such as synchronized intermittent mandatory ventilation (IMV) or pressure-supported ventilation (PSV), to allow the patient to breathe without receiving a mandatory breath with every inspiration; or (3) sedate the patient to control breathing better. Sedation may be needed for patients demonstrating extreme agitation, increased work of breathing (WOB), or patient-ventilator asynchrony. It is important to recognize that sedation may not always be the best choice. Whenever possible, the cause of hyperventilation should be investigated and treated. Common causes of hyperventilation include hypoxemia, pain, anxiety, fever, agitation, and asynchrony.

Head injury that results in a high $\dot{V}_E$ and hyperventilation is sometimes difficult to control, regardless of the mode selected. Some patients with brain injury have a tendency to breathe with a high V_T and *f*, which is the result of a central nervous system lesion and cannot be corrected.[3]

Metabolic Acidosis and Alkalosis

Treatment of metabolic acidosis and alkalosis should focus on identifying metabolic factors that can cause these acid-base disturbances. Although it may not be apparent, a respiratory component may also be involved, and this possibility should be addressed.

Metabolic Acidosis

Patients in apparent respiratory distress may present with metabolic acidosis. Blood gases indicate pH = 7.00 to 7.34 and a bicarbonate in the range of about 12 to 22 mEq/L. These patients are often struggling to lower their P_aCO_2 to achieve some degree of hyperventilation to compensate for the metabolic acidosis. As a consequence, these patients are at risk for developing respiratory muscle fatigue. Thus, in this situation, mechanical ventilation is indicated to meet the minimum goal of compensated hypocapnia.[4] Whether this can be accomplished with noninvasive or invasive ventilation depends on whether the patient meets the criteria for this mode of ventilation (see Chapter 19). If invasive ventilation is required, the risks must be balanced against the temporary benefits.

Causes of metabolic acidosis include the following processes:

- **Ketoacidosis** (alcoholism, starvation, diabetes)
- Uremic acidosis (renal failure to excrete acid)
- Loss of bicarbonate (diarrhea)
- Renal loss of base after administration of carbonic anhydrase inhibitors (e.g., acetazolamide [Diamox])
- Lactic acidosis
- Toxins ingested that produce acidosis (salicylate, ethylene glycol [antifreeze], methanol)

Treatment for metabolic acidosis includes initiating effective therapy to address the cause of the acidosis and assessing the need to reverse the acidemia with the administration of an alkalizing agent. Treating the underlying cause of acidemia and making sure vascular volume and cardiac output are normal, in addition to ensuring adequate oxygenation, are essential. These actions allow time for the normal metabolism of organic acids (lactic acid and ketoacids) and allow time for the kidneys to generate bicarbonate to replace losses.[4]

Controversy abounds regarding the benefit of using alkalinizing agents, such as bicarbonate administration, in the treatment of metabolic acidosis. Lowering arterial CO_2 is also controversial, but if the patient is losing the struggle to maintain high $\dot{V}_E$ with spontaneous breathing, assisted ventilation may be necessary to avoid respiratory failure. In this situation it is appropriate to keep the pH within normal limits (7.35–7.45) (Case Study 12.1).

Metabolic Alkalosis

Metabolic alkalosis is present when pH (7.45–7.70) and bicarbonate (26–48 mEq/L) are elevated above normal. Common causes include the following:

- Loss of gastric fluid and stomach acids (vomiting, nasogastric suctioning)
- Acid loss in the urine (diuretic administration)
- Acid shift into the cells (potassium deficiency)
- Lactate, acetate, or citrate administration
- Excessive bicarbonate loads (bicarbonate administration)

As with metabolic acidosis, treatment involves identifying the underlying cause and reversing those factors leading to the alkalosis. In severe cases, carbonic anhydrase inhibitors, acid infusion, and low bicarbonate dialysis may be required.[5]

Uncomplicated metabolic alkalosis is not usually associated with alveolar hypoventilation as a compensatory mechanism because of the resulting hypoxemia that occurs with severe hypoventilation. For example, P_aCO_2 will usually not rise higher than 55 mm Hg. Remember that the P_aO_2 will fall as the CO_2 rises. Thus hypoventilation is accompanied by hypoxemia in a patient who is breathing room air. The hypoxemia will cause stimulation of the peripheral chemoreceptors, resulting in increased minute ventilation. Only in rare circumstances does full compensation of metabolic alkalosis occur.[5]

Mixed Acid-Base Disturbances

Some patients with acute respiratory failure have mixed respiratory and metabolic disturbances, such as respiratory acidosis combined with metabolic alkalosis. Notice that the pH may actually be near normal. The following case can be used to illustrate this point. Two additional case studies are presented to illustrate the management of patients with (1) combined respiratory alkalosis with metabolic acidosis and (2) combined respiratory acidosis with metabolic alkalosis.

Clinical Scenario: Mixed Respiratory and Metabolic Disturbances

A 65-year-old patient on mechanical ventilation has a nasogastric tube in place with suction. He is also receiving diuretics for congestive heart failure (CHF). The pH is 7.36, P_aCO_2 is 58 mm Hg, HCO_3^- 31 mEq/L, and P_aO_2 is 111 mm Hg ($F_IO_2 = 0.30$). V_T is 400 mL, and the rate is 12 breaths/min. Is it appropriate to increase $\dot{V}_E$ so that the P_aCO_2 can be returned to 40 mm Hg? In this case it is *not* appropriate to increase $\dot{V}_E$ to decrease the P_aCO_2. This can rapidly result in alkalosis with accompanying cardiac dysrhythmias, seizures, and other neurological disturbances. The cause of both problems should be determined and corrected.

Clinical Scenario: Combined Respiratory Alkalosis and Metabolic Acidosis

A 55-year-old man is transported by helicopter to a large metropolitan hospital. His initial diagnosis is acute myocardial infarction. Paramedics intubate the patient, start an intravenous (IV) drip with normal saline, and begin manual ventilation with 100% O_2. After admission to the hospital, the patient has a vascular stent placed in one of the coronary vessels to improve its patency. The patient is stabilized, and mechanical ventilation is initiated with the following settings: IMV 16 breaths/min and $V_T = 750$ mL ($\approx$10 mL/kg, IBW). No spontaneous breaths are present. No changes are made in the ventilator settings, and the V_T is maintained at approximately 10 mL/kg IBW.

Two days later the respiratory therapist obtains a 6:00 a.m. ABG for this patient. The ventilator settings are still the same. ABG results are as follows:

$$P_aCO_2 = 25 \text{ mm Hg}; \text{pH} = 7.42; HCO_3^- \ 17 \text{ mEq/L}$$

The patient does not have a history of pulmonary or metabolic disease. His normal P_aCO_2 is about 40 mm Hg. Both the V_T and rate are high for this patient. Because the P_{plat} is only 20 mm Hg, the therapist decides to reduce the rate initially to begin targeting the patient's normal P_aCO_2.

$$\text{Desired f} = (\text{Actual rate} \times \text{Actual } P_aCO_2)/\text{Desired } P_aCO_2$$

$$\text{Desired f} = (16 \times 25)/40$$
$$\text{Desired f} = 10 \text{ breaths/min}$$

Attempts to reduce ventilator rate to 10 breaths/min result in an increased spontaneous rate of more than 25 breaths/min. The patient is conscious, appears anxious, and is laboring to breathe. Why is it difficult to reduce the mandatory rate in this situation?

The patient has a compensated respiratory alkalosis. The initial $\dot{V}_E$ was set too high for this patient (set $\dot{V}_E = 12$ L/min; predicted $\dot{V}_E = 10$ L/min). After 2 days on these settings, the kidneys have compensated for an iatrogenic hyperventilation by excreting excess bicarbonate. To allow the reverse process to take place, the mandatory rate will have to be reduced slowly (1–2 breaths about every 2–4 hours) so that normal ventilation can be restored and the kidneys given time to increase the bicarbonate back to a normal level.

Clinical Scenario: Combined Respiratory Acidosis and Metabolic Alkalosis

A 35-year-old woman is receiving mechanical ventilation. ABG analysis shows a P_aCO_2 of 60 mm Hg, pH of 7.41, $HCO_3^- = 35$ mEq/L. (NOTE: The patient's normal P_aCO_2 is 40 mm Hg.) The fact that the high P_aCO_2 is not reflected in the pH indicates that the patient appears to have a metabolic alkalosis and a respiratory acidosis. Before the P_aCO_2 can be corrected with ventilator adjustment, the metabolic alkalosis must be corrected. If the P_aCO_2 were decreased to 40 mm Hg, the pH would increase and the patient would develop severe alkalosis.

Increased Physiological Dead Space

If pure respiratory acidosis persists even after alveolar ventilation has been increased, the patient may have a problem that is the result of increased alveolar dead space. Increased alveolar dead space can be caused by a pulmonary embolism or low cardiac output resulting in low pulmonary perfusion.

Key Point 12.1 Increased alveolar dead space can occur when high levels of positive end-expiratory pressure compress pulmonary capillaries and reduce pulmonary blood flow to the lungs.

Increased **alveolar dead space** can also occur when ventilatory support reduces pulmonary blood flow to the lungs by causing high alveolar pressures, such as in the application of high PEEP (Key Point 12.1). Reduced pulmonary perfusion can be associated with air trapping that results from a high $\dot{V}_E$, a low inspiratory gas flow (high inspiratory-to-expiratory [I/E] ratio, such as 3:1), or an uneven distribution of ventilation because of a pathological lung problem.

In the case of air trapping (auto-PEEP), increasing the flow or decreasing the I/E ratio (to 1:3 or 1:4) may correct the problem. (Increasing inspiratory flow can shorten T_I and allow more time for exhalation.) Sometimes repositioning the patient so that the disease-compromised lung receives minimal blood flow (nondependent position) while the nondiseased lung receives greater blood flow can significantly improve gas exchange and help address the problem. (See section on positioning later in this chapter.)

The normal ratio of dead space to tidal volume (V_D/V_T) is 0.2 to 0.4. In critically ill patients, this value can be greater than 0.7. Calculation of V_D/V_T ratio uses the Enghoff modification of the Bohr equation: $V_D/V_T = (P_aCO_2 - P_ECO_2)/P_aCO_2$, where P_ECO_2 is the partial pressure of CO_2 in the mixed expired gases collected from the patient.* A blood gas sample is collected and the average V_T is measured while the P_ECO_2 is being measured. Note that although this method of measuring the V_D/V_T ratio can provide useful information on mechanically ventilated patients, it typically requires additional equipment and is not routinely performed.

It is more common in patients on ventilation to monitor the end-tidal CO_2 ($EtCO_2$; normal $P_{et}CO_2 = 35–43$ mm Hg) and the gradient between arterial and $EtCO_2$ to determine whether dead space is changing (normal P_aCO_2-to-$P_{et}CO_2$ gradient = 4–6 mm Hg). A decrease in $EtCO_2$ and an increase in the P_aCO_2-to-$P_{et}CO_2$ gradient suggest increased dead space. Some clinicians use the mean value of the $P_{et}CO_2$ instead of measuring mixed expired gas to calculate the V_D/V_T ratio.[6] Advances in capnography using single-breath volumetric CO_2 monitoring offer still another alternative for estimating V_D/V_T. (See Chapter 10 for more details on $P_{et}CO_2$ and volumetric CO_2 monitoring.)

Increased Metabolism and Increased Carbon Dioxide Production

Metabolic rate and $\dot{V}CO_2$ are elevated in patients who have fever, burns, multiple trauma, sepsis, hyperthyroidism, muscle tremors or seizures, or agitation and in patients who have undergone multiple surgical procedures. Regardless of the cause, it is clear that $\dot{V}_E$ will be increased and WOB will be elevated. Increasing the ventilator rate will decrease the patient's WOB, but auto-PEEP may occur. If auto-PEEP is a factor, it may be beneficial to add enough pressure support (PS) for the spontaneous breaths to reduce WOB through the ET and circuit. Other options might include switching to PC-CMV and using sedation to reduce the patient's work.

Clinical Scenario: Increased Metabolism and Increased CO_2 Production

A 25-year-old patient with burns on IMV has a V_T of 0.7 L, rate of 10 breaths/min, P_aCO_2 of 40 mm Hg, and pH of 7.39. The patient has a spontaneous rate of 15 breaths/min and a spontaneous V_T of 600 mL. His P_aO_2 is 88 mm Hg on 0.5 F_IO_2. This patient has a high total $\dot{V}_E$ (ventilator $\dot{V}_E$ = 7 L/min + patient $\dot{V}_E$ = 9 L/min; total $\dot{V}_E$ = 16 L/min). Given this level of $\dot{V}_E$, one would expect the P_aCO_2 to be lower. The reason that it is not lower may be either an increased $\dot{V}CO_2$ or an increased V_D/V_T.

Intentional Iatrogenic Hyperventilation

Historically, **iatrogenic** hyperventilation has been used in patients with acute head injury and increased intracranial pressure (ICP). Hyperventilation lowers CO_2 in the blood, which in turn is associated with constriction of cerebral blood vessels, resulting in a reduction in blood flow to the brain. Although this approach was believed by many clinicians to help lower increased ICP, therapeutic guidelines for head injuries with increased ICP do *not* recommend prophylactic hyperventilation (P_aCO_2 <25 mm Hg) during the first 24 hours.[7,8] Hyperventilation during the first few days after severe traumatic brain injury may actually increase cerebral **ischemia** and cause cerebral hypoxemia (see Chapter 7 section on closed head injury).

Hyperventilation may be required for brief periods when acute neurological deterioration is present and ICP is elevated. Mild hyperventilation (P_aCO_2 30–35 mm Hg) may be used for longer periods in situations in which increased ICP is refractory to standard treatment, including sedation and analgesia, neuromuscular blockade, cerebrospinal fluid drainage, and **hyperosmolar** therapy.[7] (NOTE: Guidelines for ventilation therapies used in the treatment of brain injuries also include recommendations for monitoring jugular venous O_2 saturation [SjO_2] and the partial pressure of brain tissue O_2 [$BtpO_2$] to measure O_2 delivery to the brain if hyperventilation is used.[8]) It is important to mention that the practice of iatrogenic hyperventilation is debatable because of the effects associated with reductions in cerebral blood flow and resultant development of cerebral ischemia. The following case study provides an example of the use of iatrogenic hyperventilation.

Clinical Scenario: Iatrogenic Hyperventilation

A 42-year-old woman who is 5-ft, 4-in tall (IBW = 125 lb [57 kg]) is on controlled ventilation for 12 hours after a severe cerebral concussion. Her P_aCO_2 is 48 mm Hg, her pH is 7.32, and her V_T is 400 mL. Respiratory rate is 12 breaths/min. What are your recommendations? Current V_T is 7 mL/kg. Is it appropriate to change *f*? In this case it would be appropriate to increase the *f* and maintain the P_aCO_2 at normal levels for the first 24 hours.

*Mixed expired gas includes alveolar gas and gas from the anatomical dead space. Historically, mixed expired analyses were obtained using a large airtight collection bag, such as a Douglas bag. Exhaled gases were collected over a 3-minute period, and then a sample of the exhaled gas was analyzed using a standard blood gas analyzer. With most intensive care unit (ICU) ventilators, mixed expired gas samples can be obtained using rapid-responding CO_2 analyzers and computer software incorporated into the ventilator design.

If we change the rate, the following recommendation would apply:

$$\text{Desired } f = 12 \times 48/40 = 14 \text{ breaths/min}$$

Permissive Hypercapnia

Occasionally, it becomes impossible to maintain normal P_aCO_2 levels in a patient without risking lung damage from high P_{plat} (>30 cm H_2O) and volumes. Patients with acute respiratory distress syndrome (ARDS) or status asthmaticus and sometimes patients with chronic obstructive pulmonary disease (COPD) who require ventilatory support are at risk for ventilator-induced injury. Inappropriate ventilator settings can result in severe lung injury and activation of inflammatory mediators and can even potentially lead to multisystem organ failure.[9] (See Chapter 17 for additional information on ventilator-induced lung injury.)

A technique referred to as **permissive hypercapnia** (PHY) is an alternative form of protective ventilator patient management.[10] PHY is a deliberate limitation of ventilatory support to avoid lung overdistention and injury of the lung. During PHY arterial P_aCO_2 values are allowed to rise above normal (e.g., ≥50 and ≤150 mm Hg), and pH values are allowed to fall below normal (e.g., ≥7.10–7.30). Patients who do not have renal failure or cardiovascular problems usually tolerate a pH of 7.20 to 7.25. Younger patients may tolerate even lower pH values. Many clinicians who use PHY allow for a gradual rise in P_aCO_2 because an abrupt increase in P_aCO_2 is usually not well tolerated.[11]

The physiologic effects of hypercapnia are complex. It can affect multiple organ systems.[10,12] Although most investigators agree that a pH of 7.25 or greater is acceptable, no one is certain whether a lower pH is acceptable. Survival without complications has been demonstrated in isolated cases in which the pH dropped as low as 6.6 and the P_aCO_2 rose as high as 375 mm Hg when oxygenation was well maintained.[11,13] Similar findings have been shown in studies of patients with ARDS and acute asthma.[14-17]

During hypoventilation, increases in P_aCO_2 are accompanied by a decrease in P_aO_2, so O_2 administration must be provided and oxygenation status monitored carefully. Increases in P_aCO_2 and decreases in pH that occur in acute respiratory acidosis also cause a right shift in the oxyhemoglobin dissociation curve. Although this shift in the curve facilitates unloading of O_2 at the tissue level, it also reduces O_2 loading at the lungs and can further compromise gas exchange.

Increases in CO_2 have an additional physiological effect. A higher-than-normal P_aCO_2 stimulates the drive to breathe. Therefore it is appropriate to provide sedation to patients with acute lung injury in whom permissive hypercapnia is being used. The sedation may improve patient comfort. (Note that extremely high levels of P_aCO_2 can result in an anesthesia effect referred to as *CO_2 narcosis* [Key Point 12.2].)

Procedures for Managing Permissive Hypercapnia

Efforts to maintain eucapnic breathing (i.e., near a patient's normal level of P_aCO_2) might include removing sources of mechanical dead space and increasing the frequency of mandatory breaths.[18] When the decision is made to allow P_aCO_2 to rise above normal, the following strategy may be used[19,20]:

1. Allow P_aCO_2 to rise and pH to fall without changing the mandatory rate or volume. Do nothing other than sedate the patient, avoid high ventilating pressures, and maintain oxygenation.
2. Reduce CO_2 production by using paralytic agents, cooling the patient, and restricting glucose intake.
3. Administer agents such as sodium bicarbonate, *tris*(hydroxymethyl)aminomethane (tromethamine [THAM], an amino buffer), or Carbicarb (a mixture of sodium carbonate and bicarbonate) to keep pH greater than 7.25.
4. In some cases, extracorporeal CO_2 removal ($ECCO_2R$) may be a viable alternative method of CO_2 removal to maintain a lung protective ventilation strategy.

Note that use of buffering agents remains debatable and not well studied in PHY. A short-term increase in P_aCO_2 might occur when bicarbonate is administered. This is exhaled over time if the level of ventilation is constant. The use of THAM is not associated with an increased P_aCO_2. It produces intracellular and extracellular buffering of pH. Whether buffers have any effect on the tolerance of permissive hypercapnia is not known. A protocol for the implementation of permissive hypercapnia is provided in Box 12.1.[21]

Key Point 12.2 Extremely high levels of P_aCO_2 (>200 mm Hg) can result in an anesthesia effect also known as *CO_2 narcosis*.

BOX 12.1 Protocol for the Implementation of Permissive Hypercapnia

When adequate ventilation cannot be maintained within acceptable limits for pressures and volumes, permissive hypercapnia (PHY) can be implemented using the following steps[21]:

1. Hypercapnia should be implemented progressively in increments of 10 mm Hg/h to a maximum of 80 mm Hg/day.
2. If hypercapnia should exceed 80 mm Hg, progress more slowly.
3. F_IO_2 is adjusted to maintain arterial O_2 saturation (S_aO_2) at 85% to 90%. Adequate oxygenation is imperative and can require the intermittent use of 100% O_2.
4. If PHY is used for less than 24 hours, P_aCO_2 can be allowed to decrease by 10 to 20 mm Hg/h, provided that P_aCO_2 is greater than 80 mm Hg. The closer the patient is to normocapnia, the slower the process should be.
5. If PHY is used for more than 24 hours or if large amounts of buffer agents are used, discontinue PHY even more slowly (in 1–3 days).

Contraindications of Permissive Hypercapnia

CO_2 is a powerful vasodilator of cerebral vessels. Thus increasing CO_2 levels can result in cerebral edema and increased ICP, which can aggravate cerebral disorders, such as cerebral trauma or hemorrhage, and cerebral-occupying lesions.[19,21] For this reason the use of PHY is contraindicated in the presence of disorders such as head trauma and intracranial disease. Indeed, it is generally avoided for those patients who demonstrate intracranial lesions (Key Point 12.3).[22]

PHY is relatively contraindicated in patients with preexisting cardiovascular instability. Circulatory effects of PHY can include