Chapter 9

Monitoring of the Heart and Vascular System

David L. Reich, MD • Alexander J. Mittnacht, MD •
Martin J. London, MD • Joel A. Kaplan, MD

HEMODYNAMIC MONITORING

For patients with severe cardiovascular disease and those undergoing surgery associated with rapid hemodynamic changes, adequate hemodynamic monitoring should be available at all times. With the ability to measure and record almost all vital physiologic parameters, the development of acute hemodynamic changes may be observed and corrective action may be taken in an attempt to correct adverse hemodynamics and improve outcome. Although outcome changes are difficult to prove, it is a reasonable assumption that appropriate hemodynamic monitoring should reduce the incidence of major cardiovascular complications. This is based on the presumption that the data obtained from these monitors are interpreted correctly and that therapeutic decisions are implemented in a timely fashion.

Many devices are available to monitor the cardiovascular system. These devices range from those that are completely noninvasive, such as the blood pressure (BP) cuff and ECG, to those that are extremely invasive, such as the pulmonary artery (PA) catheter. To make the best use of invasive monitors, the potential benefits to be gained from the information must outweigh the potential complications. In many critically ill patients, the benefit obtained does seem to outweigh the risks, which

---

BOX 9-1 *Standard Monitoring for Cardiac Surgical Patients*

- (Invasive) blood pressure
- Electrocardiogram
- Pulse oximetry
- Capnometry
- Temperature
- Central venous pressure
- Urine output
- Intermittent arterial blood gas analysis

BOX 9-2 *Extended Monitoring for Patients Based on Case-Specific Factors*

- Pulmonary artery catheter
- Cardiac output measurements
- Transesophageal echocardiography
- Left atrial pressure
- Bispectral index monitoring
- Cerebral oximetry
- Jugular venous saturation or pressure
- Spinal drain (intrathecal) pressure

---

explains the widespread use of invasive monitoring. Transesophageal echocardiography (TEE), a minimally invasive technology, has gained in popularity as an alternative to the PA catheter and is considered a standard-of-care monitoring device in the perioperative management for certain procedures, such as mitral valvuloplasty or surgery for congenital heart defects.

Standard monitoring for cardiac surgical patients includes BP, ECG, central venous pressure (CVP), urine output, temperature, capnometry, pulse oximetry, and intermittent arterial blood gas analysis (Box 9-1). The next tier of monitoring includes PA catheters, left atrial pressure (LAP) catheters, thermodilution cardiac output (CO) measurements, TEE, and indices of tissue oxygen transport (Box 9-2). All of these measurements and their derivatives can be obtained and recorded. The interpretation of these complex data, however, requires an astute clinician who is aware of the patient's overall condition and the limitations of the monitors.[1]

ARTERIAL PRESSURE MONITORING

Blood pressure monitoring is the most commonly used method of assessing the cardiovascular system. The magnitude of the BP is directly related to the CO and the systemic vascular resistance (SVR). This is analogous to Ohm's law of electricity (voltage = current × resistance), in which BP is analogous to voltage, CO to flow, and SVR to resistance. An increase in the BP may reflect an increase in CO or SVR, or both. Although BP is one of the easiest cardiovascular variables to measure, it gives only indirect information about the patient's cardiovascular status.

Mean arterial pressure (MAP) is probably the most useful parameter to measure in assessing organ perfusion, except for the heart, in which the diastolic BP is the

most important. MAP is measured directly by integrating the arterial waveform tracing over time, or using the formula:

$$MAP = (SBP + [2 \times DBP])/3$$

or

$$MAP = DBP + \left(\frac{SBP - DBP}{3} \right)$$

where SBP is systolic blood pressure and DBP is diastolic blood pressure. The pulse pressure is the difference between SBP and DBP.

Anesthesia for cardiac surgery is frequently complicated by rapid and sudden lability of the BP because of several factors, including direct compression of the heart, impaired venous return due to retraction and cannulation of the venae cavae and aorta, arrhythmias from mechanical stimulation of the heart, and manipulations that may impair right ventricular (RV) outflow and pulmonary venous return. Sudden losses of significant amounts of blood may induce hypovolemia at almost any time. The cardiac surgical population also includes many patients with labile hypertension and atherosclerotic heart disease. A safe and reliable method of measuring acute changes in the BP is required during cardiac surgery with cardiopulmonary bypass (CPB).

Continuous BP monitoring with noninvasive devices have not proven to be suitable for cardiac surgery. Intra-arterial monitoring provides a continuous, beat-to-beat indication of the arterial pressure and waveform, and having an indwelling arterial catheter enables frequent sampling of arterial blood for laboratory analyses. Direct intra-arterial monitoring remains the gold standard for cardiac surgical procedures.

Arterial Cannulation Sites

Factors that influence the site of arterial cannulation include the location of surgery, the possible compromise of arterial flow due to patient positioning or surgical manipulations, and any history of ischemia of or prior surgery on the limb to be cannulated. Another factor that may influence the cannulation site is the presence of a proximal arterial cutdown. The proximal cutdown may cause damped waveforms or falsely low BP readings due to stenosis or vascular thrombosis.

The radial artery is the most commonly used artery for continuous BP monitoring because it is easy to cannulate with a short (20-gauge) catheter. It is readily accessible during surgery, and the collateral circulation is usually adequate and easy to check. It is advisable to assess the adequacy of the collateral circulation and the absence of proximal obstructions before cannulating the radial artery for monitoring purposes.

The ulnar artery provides most blood flow to the hand in about 90% of patients. The radial and ulnar arteries are connected by a palmar arch, which provides collateral flow to the hand in the event of radial artery occlusion. It has been shown that if there is adequate ulnar collateral flow, circulatory perfusion pressure to the fingers is adequate after radial arterial catheterization. Many clinicians routinely perform Allen's test before radial artery cannulation to assess the adequacy of collateral circulation to the hand.

Allen's test is performed by compressing the radial and ulnar arteries and by exercising the hand until it is pale. The ulnar artery is then released (with the hand open loosely), and the time until the hand regains its normal color is noted. With a normal collateral circulation, the color returns to the hand in about 5 seconds. If, however, the hand takes longer than 15 seconds to return to its normal color, cannulation of the radial artery on that side is controversial. The hand may remain pale if the fingers are hyperextended or

widely spread apart, even in the presence of a normal collateral circulation. Variations on Allen's test include using a Doppler probe or pulse oximeter to document collateral flow. If Allen's test demonstrates that the hand depends on the radial artery for adequate filling, and other cannulation sites are not available, the ulnar artery may be selected.[2]

Chest wall retractors, such as the Favaloro retractor, may impede radial arterial pressure monitoring in cardiothoracic procedures in some patients. The arm on the affected side may have diminished perfusion during extreme retraction of the chest wall. If the left internal mammary artery is used during myocardial revascularization, the right radial artery could be monitored to avoid this problem. Alternatively, a noninvasive BP cuff on the right side could be used to confirm the accuracy of the radial artery tracing during extreme chest wall retraction.

Monitoring of the radial artery distal to a brachial arterial cutdown site is not recommended. Acute thrombosis or residual stenosis of the brachial artery will lead to falsely low radial arterial pressure readings. Other considerations related to the choice of a radial arterial monitoring site include prior surgery of the hand, selection of the nondominant hand, and the preference of the surgeon, the anesthesiologist, or both.

The brachial artery lies medial to the bicipital tendon in the antecubital fossa, in close proximity to the median nerve. Brachial artery pressure tracings resemble those in the femoral artery, with less systolic augmentation than radial artery tracings. Brachial arterial pressures were found to more accurately reflect central aortic pressures than radial arterial pressures before and after CPB. The complications from percutaneous brachial artery catheter monitoring are lower than those after brachial artery cutdown for cardiac catheterization.[3] A few series of perioperative brachial arterial monitoring have documented the relative safety of this technique.

The femoral artery may be cannulated for monitoring purposes but is usually reserved for situations in which other sites are unable to be cannulated or it is specifically indicated (e.g., descending thoracic aortic aneurysm surgery for distal pressure monitoring). Peripheral artery cannulation for hemodynamic monitoring, including 3899 femoral artery cannulations, has been studied. Temporary occlusion was found in 10 patients (1.45%), whereas serious ischemic complications requiring extremity amputation were reported in 3 patients (0.18%). Other complications were pseudoaneurysm formation in 6 patients (0.3%), sepsis in 13 patients (0.44%), local infection (0.78%), bleeding (1.58%), and hematoma (6.1%). The femoral artery for hemodynamic monitoring purposes was as safe as radial artery cannulation.

In patients undergoing thoracic aortic surgery, distal aortic perfusion (using partial CPB, left-heart bypass, or a heparinized shunt) may be performed during aortic cross-clamping to preserve spinal cord and visceral organ blood flow. In these situations, it is useful to measure the distal aortic pressure at the femoral artery (or, dorsalis pedis or posterior tibial artery) to optimize the distal perfusion pressure. In repairs of aortic coarctation, simultaneous femoral and radial arterial monitoring may help determine the adequacy of the surgical repair by documenting the pressure gradient after the repair. It is necessary to consult with the surgeon before cannulating the femoral vessels because these vessels may be used for extracorporeal perfusion or placement of an intra-aortic balloon pump during the surgical procedure.

Indications

The indications for invasive arterial monitoring are provided in Box 9-3.

BOX 9-3   *Indications for Intra-Arterial Monitoring*

- Major surgical procedures involving large fluid shifts or blood loss
- Surgery requiring cardiopulmonary bypass
- Surgery of the aorta
- Patients with pulmonary disease requiring frequent arterial blood gases
- Patients with recent myocardial infarctions, unstable angina, or severe coronary artery disease
- Patients with decreased left ventricular function (congestive heart failure) or significant valvular heart disease
- Patients in hypovolemic, cardiogenic, or septic shock or with multiple organ failure
- Procedures involving the use of deliberate hypotension or deliberate hypothermia
- Massive trauma cases
- Patients with right-sided heart failure, chronic obstructive pulmonary disease, pulmonary hypertension, or pulmonary embolism
- Patients requiring inotropes or intra-aortic balloon counterpulsation
- Patients with electrolyte or metabolic disturbances requiring frequent blood samples
- Inability to measure arterial pressure noninvasively (e.g., morbid obesity)

Insertion Techniques

Direct Cannulation

Proper technique is helpful in obtaining a high degree of success in arterial catheterization. The wrist should be placed in a dorsiflexed position on an armboard and immobilized in a supinated position. It is helpful to draw the course of the artery for 1 inch and to be comfortably seated. Doppler devices and ultrasonic vessel finders may also be of value. Local anesthetic is injected intradermally over the artery, and a small skin nick may be made to allow passage of the catheter-over-needle assembly into the subcutaneous tissue without crimping secondary to penetration of the unit through the skin. A 20-gauge or smaller, 3- to 5-cm, nontapered Teflon catheter over needle is used, without a syringe attached, to make the puncture. If a syringe is used, the plunger should be removed to allow free flow of blood to detect when the artery has been punctured. The angle between the needle and the skin should be shallow (30 degrees or less), and the needle should be advanced parallel to the course of the artery. When the artery is entered, the angle between the needle and skin is reduced to 10 degrees, the needle is advanced another 1 to 2 mm to ensure that the tip of the catheter also lies within the lumen of the vessel, and the outer catheter is then threaded off the needle while watching that blood continues to flow out of the needle hub (Fig. 9-1). After insertion of the catheter, the wrist should be taken out of the dorsiflexed position, because continued extreme dorsiflexion can lead to median nerve damage by stretching of the nerve over the wrist. An armboard may still be used to prevent the wrist from flexing, which causes kinking of the catheter and damping of the arterial waveform.

Transfixation

If blood ceases flowing while the needle is being advanced, the needle has penetrated the back wall of the vessel. In this technique, the artery has been transfixed by passage of the catheter-over-needle assembly "through-and-through" the artery. The needle is then completely withdrawn. As the catheter is slowly withdrawn, pulsatile blood flow emerges from the catheter when its tip is within the lumen of the artery. The catheter is then slowly advanced into the artery. A guidewire may be helpful at this point if the catheter does not advance easily into the artery. Alternatively, the catheter-over-needle
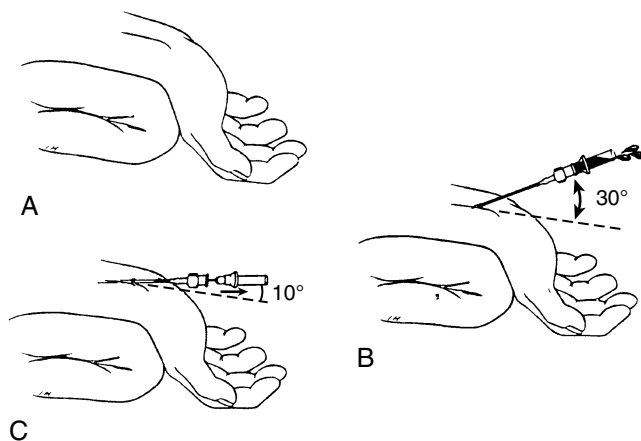
9

Figure 9-1   The direct-cannulation technique for the radial artery. **A,** The wrist is dorsi-flexed and loosely taped to a stable surface such as an armboard. **B,** The artery is directly cannulated at a 30- to 40-degree angle to the plane of the wrist. Arterial blood flows steadily into the "flashback" chamber. **C,** The catheter-over-needle assembly is lowered until the angle is approximately 10 degrees to the plane of the wrist. The entire assembly is advanced another 1 to 2 mm, until the tip of the catheter lies within the lumen of the artery. The catheter is then advanced into the artery completely while the needle is held motionless. (From Lake CL: Cardiovascular Anesthesia. New York, Springer-Verlag, 1985, p 54.)

assembly may be withdrawn slowly as one unit until flow of blood has returned. As soon as this occurs, the needle and catheter are most likely in the lumen of the artery and the catheter may be gently threaded off the needle into the artery.

Seldinger Technique

The artery is localized with a needle, and a guidewire is passed through the needle into the artery. A catheter is then passed over the guidewire into the artery. Alternatively, a catheter-over-needle assembly may be inserted in the artery in a through-and-through fashion, the needle withdrawn, and the wire passed through the catheter after pulsatile flow is encountered. It is very important when using this technique to avoid withdrawal of guidewires through needles to prevent shearing of the wire and embolization.

CENTRAL VENOUS PRESSURE MONITORING

Central venous pressure catheters are used to measure the filling pressure of the right ventricle, give an estimate of the intravascular volume status, and assess RV function. For accurate pressure measurement, the distal end of the catheter must lie within one of the large intrathoracic veins or the right atrium. Although water manometers have been used in the past, an electronic system is preferred because it allows the observation of the right atrial (RA) waveform, which provides additional information. In any pressure monitoring system, it is necessary to have a reproducible landmark (e.g., the midaxillary line) as a zero reference. This is especially important in monitoring venous pressures, because small changes in the height of the zero reference point produce proportionately larger errors compared with arterial pressure monitoring.

The normal CVP waveform consists of three upward deflections (A, C, and V waves) and two downward deflections (X and Y descents) (Fig. 9-2). The A wave is produced by right atrial contraction and occurs just after the P wave on the ECG.
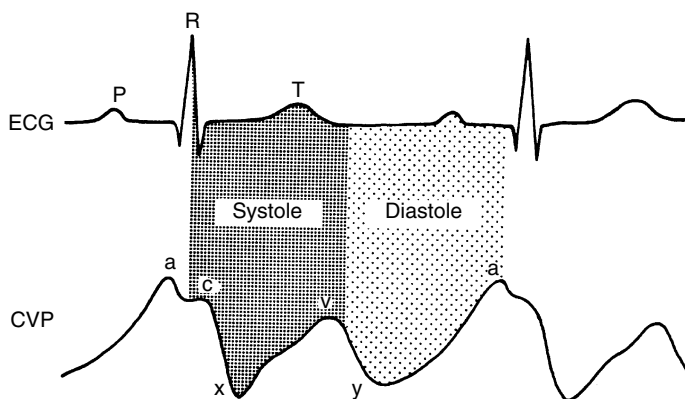
III

Figure 9-2 The relationship of the central venous pressure (CVP) tracing to the electrocardiogram (ECG) in normal sinus rhythm. The normal CVP waveform consists of three upward deflections (A, C, and V waves) and two downward deflections (X and Y descents). The A wave is produced by right atrial contraction and occurs just after the P wave on the ECG. The C wave occurs because of the isovolumic ventricular contraction forcing the tricuspid valve to bulge upward into the right atrium. The pressure within the right atrium then decreases as the tricuspid valve is pulled away from the atrium during right ventricular ejection, forming the X descent. The right atrium continues to fill during late ventricular systole, forming the V wave. The Y descent occurs when the tricuspid valve opens and blood from the right atrium empties rapidly into the right ventricle during early diastole. (Adapted from Mark JB: Central venous pressure monitoring: Clinical insights beyond the numbers. J Cardiothorac Vasc Anesth 5:163, 1991.)

The C wave occurs because of the isovolumic ventricular contraction forcing the tricuspid valve to bulge upward into the right atrium. The pressure within the right atrium then decreases as the tricuspid valve is pulled away from the atrium during RV ejection, forming the X descent. RA filling continues during late ventricular systole, forming the V wave. The Y descent occurs when the tricuspid valve opens and blood from the right atrium empties rapidly into the right ventricle during early diastole.

The CVP is a useful monitor if the factors affecting it are recognized and its limitations are understood. The CVP reflects the patient's blood volume, venous tone, and RV performance. Following serial measurements (trends) is more useful than individual numbers. The response of the CVP to a volume infusion is a useful test.

The CVP does not give a direct indication of left-heart filling pressure, but it may be used as an estimate of left-sided pressures in patients with good LV function. A good correlation has been shown between the CVP and left-sided filling pressures during a change in volume status in patients with coronary artery disease and left ventricular ejection fraction (LVEF) greater than 0.4.

Internal Jugular Vein

Cannulation of the internal jugular vein (IJV) was first described by English and coworkers in 1969. Its popularity among anesthesiologists has steadily increased since that time. Advantages of this technique include the high success rate as a result of the relatively predictable relationship of the anatomic structures; a short, straight course to the right atrium that almost always assures RA or superior vena cava (SVC) localization of the catheter tip; easy access from the head of the operating room table; and fewer complications than with subclavian vein catheterization. The IJV is located under the medial border of the lateral head of the sternocleidomastoid (SCM) muscle (Fig. 9-3). The carotid artery is usually deep and medial to the IJV. The right IJV is
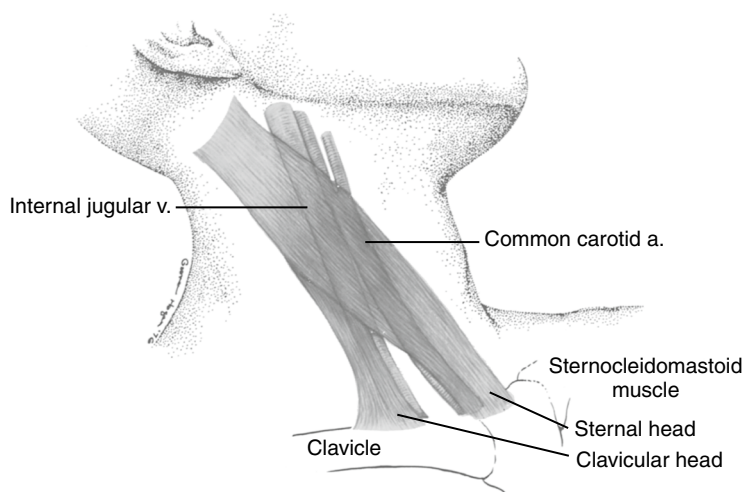
Figure 9-3   The internal jugular vein is usually located deep to the medial border of the lateral head of the sternocleidomastoid muscle, just lateral to the carotid pulse.

Figure 9-4   The preferred middle approach to the right internal jugular vein. The needle enters the skin at the apex of the triangle formed by the sternal and clavicular heads of the sternocleido-mastoid muscle. The needle is held at a 30- to 45-degree angle to the skin and directed toward the ipsilateral nipple.

preferred, because this vein takes the straightest course into the SVC, the right cupola of the lung may be lower than the left, and the thoracic duct is on the left side.

The preferred *middle approach* to the right IJV is shown in Figure 9-4. With the patient supine or in Trendelenburg position and the head turned toward the contralateral side, the fingers of the left hand are used to palpate the two heads of the SCM muscle and the carotid pulse. These fingers then hold the skin stable over the underlying structures while local anesthetic is infiltrated into the skin and subcutaneous tissues. A 22-gauge "finder" needle is placed at the apex of the triangle formed by the two heads of the SCM muscle at a 45-degree angle to the skin and directed toward the ipsilateral nipple. If venous blood return is not obtained, the needle is withdrawn to the subcutaneous tissue and then passed in a more lateral

or medial direction until the vein is located. This needle reduces the risk of consequences related to inadvertent carotid arterial puncture and tissue trauma if localization of the vein is difficult. When venous blood is aspirated through the "finder" needle, the syringe and needle are withdrawn, leaving a small trail of blood on the drape to indicate the direction of the vein. Alternatively, the needle and syringe can be fixated and used as an identifying needle. Then, a syringe attached to an 18-gauge intravenous catheter-over-needle is inserted in an identical fashion. When venous return is present, the whole assembly is lowered to prevent the needle from going through the posterior wall of the central vein and advanced an additional 1 to 2 mm until the tip of the catheter is within the lumen of the vein. The catheter is then threaded into the vein.

Once the catheter is advanced into the vein, the needle is removed, and an empty syringe is attached to the cannula to withdraw a sample of blood. To confirm that an artery has not been inadvertently cannulated, comparison of the color of the blood sample to an arterial sample drawn simultaneously is recommended. If this is inconclusive or there is no arterial catheter in place, the cannula may be attached to a transducer by sterile tubing to observe the pressure waveform. Another option is to attach the cannula to sterile tubing and allow blood to flow retrograde into the tubing. The tubing is then held upright as a venous manometer, and the height of the blood column is observed. If the catheter is in a vein, it will stop rising at a level consistent with the CVP and demonstrate respiratory variation. A guidewire is then passed through the 18-gauge catheter, and the catheter is exchanged over the wire for a CVP catheter. The use of more than one technique to confirm the venous location of the catheter may provide additional reassurance of correct placement before cannulation of the vein with a larger cannula.

ULTRASONIC GUIDANCE OF INTERNAL JUGULAR VEIN CANNULATION

Ultrasound has been increasingly used to define the anatomic variations of the IJV. A review and meta-analysis of randomized controlled trials looking at ultrasound-guided central venous cannulation found that real-time two-dimensional ultrasound for IJV cannulation had a significantly higher success rate overall and on the first attempt compared with the landmark method in adults.[4] Most studies have demonstrated that two-dimensional ultrasonic guidance of IJV cannulation is helpful in locating the vein, permits more rapid cannulation, and decreases the incidence of arterial puncture.[5] Circumstances in which ultrasonic guidance of IJV cannulation can be advantageous include patients with difficult neck anatomy (e.g., short neck, obesity), prior neck surgery, anticoagulated patients, and infants.

Ultrasound has provided more precise data regarding the structural relationship between the IJV and the carotid artery (Fig. 9-5). Troianos and associates found that in more than 54% of patients, more than 75% of the IJV overlies the carotid artery. Patients who were older than 60 years were more likely to have this type of anatomy.[6] There was greater overlap of the IJV and the carotid artery when the head is rotated 80 degrees compared with head rotation of only 0 to 40 degrees. The data from 2 and 4 cm above the clavicle did not differ, and the percentage overlap was larger on the left side of the neck compared with the right. Excessive rotation of the head of the patient toward the contralateral side may distort the normal anatomy in a manner that increases the risk of inadvertent carotid artery puncture.[7] Doppler ultrasonography has also been used to demonstrate that the Valsalva maneuver increases IJV cross-sectional area by approximately 25% and that the Trendelenburg position increases it by approximately 37%.
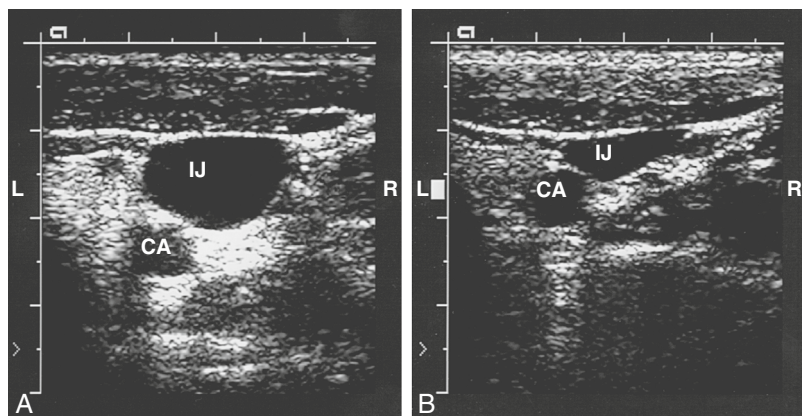
Figure 9-5 The ultrasound images depict the anatomic relationship of the right internal jugular (IJ) vein and the carotid artery (CA). **A,** With the Valsalva maneuver or in Trendelenburg position, the IJ is distended and circular. **B,** As gentle pressure is applied with the ultrasound probe, the IJ is partially compressed and flattened anteriorly and the CA size is unaffected. The compressibility of the IJ is its most characteristic feature during ultrasonic imaging.

External Jugular Vein

Although the EJV is another means of reaching the central circulation, the success rate with this approach is lower because of the tortuous path followed by the vein. A valve is usually present at the point where the EJV perforates the fascia to join with the subclavian vein. However, a success rate of 90% has been reported using a J-wire to manipulate past obstructions into the central circulation. The main advantage of this technique is that there is no need to advance a needle into the deeper structures of the neck.

For this approach, the patient is placed supine or in the Trendelenburg position until the EJV becomes distended. The vein is then cannulated with an intravenous catheter. A guidewire with curved tip (i.e., J-wire) is passed through the cannula and manipulated into the central circulation. The curved tip is necessary to negotiate the tortuous course between the EJV and the SVC. Manipulation of the shoulder and rotation of the guidewire between the operator's fingers may be useful maneuvers when difficulty is encountered in passing the wire into the superior vena cava.

Subclavian Vein

The subclavian vein is readily accessible from supraclavicular or infraclavicular approaches and has long been used for central venous access. The success rate is higher than the EJV approach but lower than the right IJV approach. Cannulation of the subclavian vein is associated with a higher incidence of complications than the IJV approach, especially pneumothorax. Other complications associated with subclavian vein cannulation are arterial punctures, misplacement of the catheter tip, aortic injury, cardiac tamponade, mediastinal hematoma, and hemothorax. This may be the cannulation site of choice, however, when CVP monitoring is indicated in patients undergoing carotid artery surgery. It is also useful for parenteral nutrition or for prolonged CVP access because the site is easier to maintain and well tolerated by patients.

The *infraclavicular approach* is performed with the patient supine or in the Trendelenburg position with a folded sheet between the scapulae and the shoulder lowered. The head is turned to the contralateral side. A thin-walled needle or intravenous catheter is inserted 1 cm below the midpoint of the clavicle and advanced toward the suprasternal notch under the posterior surface of the clavicle.

III

> **BOX 9-4** *Indications for Central Venous Catheter Placement*
>
> - Major operative procedures involving large fluid shifts or blood loss in patients with good heart function
> - Intravascular volume assessment when urine output is not reliable or unavailable (e.g., renal failure)
> - Major trauma
> - Surgical procedures with a high risk of air embolism, such as sitting-position craniotomies. The central venous pressure catheter also may be used to aspirate intracardiac air.
> - Frequent venous blood sampling
> - Venous access for vasoactive or irritating drugs
> - Chronic drug administration
> - Inadequate peripheral intravenous access
> - Rapid infusion of intravenous fluids (using large cannulae)
> - Total parenteral nutrition

When a free flow of venous blood is obtained, the guidewire is passed into the subclavian vessel and is exchanged for a CVP catheter.

Indications

Central venous pressure monitoring is often performed to obtain an indication of intravascular volume status. The accuracy and reliability of CVP monitoring depend on many factors, including the functional status of the right and left ventricles, the presence of pulmonary disease, and ventilatory factors, such as positive end-expiratory pressure (PEEP). The CVP may reflect left-sided heart filling pressures, but only in patients with good LV function. Elderly patients have a high incidence of coronary artery and pulmonary disease, and the CVP is therefore less likely to accurately reflect left-sided filling pressures in this population. Perioperative indications for the insertion of a central venous catheter are listed in Box 9-4.

The CVP should be monitored in all patients during CPB. When the catheter tip is in the SVC, it indicates RA pressure and cerebral venous pressure. Significant increases in CVP can produce critical decreases in cerebral perfusion pressure. This is occasionally caused by a malpositioned SVC cannula during CPB and must be corrected immediately by the surgeon to avoid cerebral edema and poor cerebral perfusion.

Complications

The complications of central venous cannulation can be roughly divided into three categories: complications of vascular access, complications of catheter insertion, or complications of catheter presence. These are summarized in Box 9-5.

Inadvertent arterial puncture during central venous cannulation is not uncommon. The two main reasons why this phenomenon occurs are that all veins commonly used for cannulation lie in close proximity to arteries and that the venous anatomy is quite variable. Localized hematoma formation is the usual consequence. This may be minimized if a small-gauge needle is initially used to localize the vein or ultrasonic guidance is employed.

If RA or RV perforation occurs during central venous cannulation, pericardial effusion or tamponade may result. The likelihood of this complication is increased when inflexible guidewires, long dilators, or catheter are used.

BOX 9-5 *Complications of Central Venous Catheterization*

Complications of Central Venous Cannulation

- Arterial puncture with hematoma
- Arteriovenous fistula
- Hemothorax
- Chylothorax
- Pneumothorax
- Nerve injury
- Brachial plexus
- Stellate ganglion (Horner's syndrome)
- Air emboli
- Catheter or wire shearing
- Right atrial or right ventricular perforation

Complications of Catheter Presence

- Thrombosis, thromboembolism
- Infection, sepsis, endocarditis
- Arrhythmias
- Hydrothorax

The dilators used in many of the central catheters kits may be a major cause of vessel perforation. The dilator may bend the guidewire, creating its own path, causing it to perforate a vessel wall. Several kits have dilators that are much longer than the catheters, and they constitute a further risk factor for possible perforation of the heart or vessels.

The physiology of fluid accumulation in the pericardial sac is such that sudden cardiovascular collapse occurs once a critical volume has been reached. This is explained by the compliance curve of the normal pericardium. The curve is flat until the critical volume is reached and then rises steeply with any further increment in volume. If pericardial tamponade is imminent, immediate pericardiocentesis is indicated. Withdrawal of small volumes of blood results in marked hemodynamic improvement because of the nature of pericardial compliance.

Transient atrial and ventricular arrhythmias commonly occur as the guidewire is passed into the right atrium or right ventricle during central venous cannulation using the Seldinger technique. This most likely results from the relatively inflexible guidewire causing extrasystoles as it contacts the endocardium. Ventricular fibrillation during guidewire insertion has been reported. The same investigators reported a 70% reduction in the incidence of arrhythmias when guidewire insertion was limited to 22 cm.

There are also reports of complete heart block due to guidewire insertion during central venous cannulation. These cases can be successfully managed using a temporary transvenous or external pacemaker. This complication has previously been reported with PA catheterization. The problem most likely resulted from excessive insertion of the guidewire, with impingement of the wire in the region of the right bundle branch. It is recommended that the length of guidewire insertion be limited to the length necessary to reach the SVC-RA junction to avoid these complications. It is also imperative to monitor the patient appropriately (e.g., ECG and pulse monitoring) and to have resuscitative drugs and equipment immediately available when performing central venous catheterization.

Strict aseptic technique is required to minimize catheter-related infections. Full barrier precautions during insertion of central venous catheters have been

shown to decrease the incidence of catheter-related infections.[8] Subcutaneous tunneling of central venous catheters inserted into the internal jugular and femoral veins, antiseptic barrier-protected hub for central venous catheters, and antiseptic/antibiotic-impregnated short-term central venous catheters have been shown to reduce catheter-related infections. Hospital policies differ with respect to the permissible duration of catheterization at particular sites, but routine replacement of central venous catheters to prevent catheter-related infections is not recommended.

PULMONARY ARTERIAL PRESSURE MONITORING

The introduction of the flow-directed PA catheter was a quantum advance in the monitoring of patients in the perioperative period. Since the 1970s, its use has increased the amount of diagnostic information that can be obtained at the bedside in critically ill patients. It is impressive to observe large changes in the PAP and PCWP with almost no reflection in the CVP. Connors and coworkers prospectively analyzed 62 consecutive PA catheterizations. They found that fewer than one half of a group of clinicians correctly predicted the PCWP or CO and that more than 50% made at least one change in therapy based on data from the PA catheterization. Waller and Kaplan demonstrated that a group of experienced cardiac anesthesiologists and surgeons who were blinded to the information from PA catheterization during CABG surgery were unaware of any problem during 65% of severe hemodynamic abnormalities. Similarly, Iberti and Fisher showed that ICU physicians were unable to accurately predict hemodynamic data on clinical grounds and that 60% made at least one change in therapy and 33% changed their diagnosis based on PA catheter data*. The clinical significance of these changes has been questioned because the weight of evidence-based medicine on the subject does not support improvements in outcome related to PA catheter monitoring, and the overall use of the PA catheter has decreased 60–80% over the past decade. Nevertheless, with increasing numbers of patients with multisystem organ dysfunction undergoing cardiac surgical procedures, PA catheter monitoring is prevalent in cardiac surgical settings. An understanding of the potential benefits and pitfalls of PA catheterization is therefore essential for anesthesiologists.

Specific information that can be gathered with the PA catheter and the quantitative measurements of cardiovascular and pulmonary function that can be derived from this information are listed in Tables 9-1 and 9-2.

One of the main reasons that clinicians measure PCWP and PA diastolic pressure (PADP) is that these parameters are estimates of LAP, which is an estimate of left ventricular end-diastolic pressure (LVEDP). LVEDP is an index of left ventricular end-diastolic volume (LVEDV), which correlates well with left ventricular preload. The relationship between LVEDP and LVEDV is described by the left ventricular compliance curve. This nonlinear curve is affected by many factors, such as ventricular hypertrophy and myocardial ischemia. The PCWP and PADP do not directly measure LV preload. The relationship of these parameters is diagrammed in Figure 9-6.

The PCWP and PADP pressures will not accurately reflect LVEDP in the presence of incorrect position of the PA catheter tip, pulmonary vascular disease, high levels of PEEP, or mitral valvular disease. The patency of vascular channels between the distal port of the PA catheter and the LA is necessary to ensure a close relationship between the PCWP and LAP. This condition is met only in the dependent portions

9

Table 9-1 Normal Intracardiac Pressures

Location/Pressure	Mean (mm Hg)	Range (mm Hg)
Right atrial	5	1-10
Right ventricular	25/5	15-30/0-8
Pulmonary arterial systolic/ diastolic	23/9	15-30/5-15
Mean pulmonary arterial	15	10-20
Pulmonary capillary wedge	10	5-15
Left atrial	8	4-12
Left ventricular end-diastolic	8	4-12
Left ventricular systolic	130	90-140

Table 9-2 Derived Hemodynamic Parameters

Parameter/Formula	Normal Values
Cardiac index: $CI = CO/BSA$	2.8-4.2 L/min/m^2
Stroke volume: $SV = CO \times 1000/HR$	50-110 mL/beat
Stroke index: $SI = SV/BSA$	30-65 mL/beat/m^2
Left ventricular stroke work index: $LVSWI = 1.36 \times (MAP - PCWP) \times SI/100$	45-60 g•m/m^2
Right ventricular stroke work index: $RVSWI = 1.36 \times (MPAP - CVP) \times SI/100$	5-10 g•m/m^2
Systemic vascular resistance: $SVR = (MAP - CVP) \times 80/CO$	900-1400 dynes•s•cm^{-5}
Systemic vascular resistance index: $SVRI = (MAP - CVP) \times 80/CI$	1500-2400 dynes•s•cm^{-5}/m^2
Pulmonary vascular resistance: $PVR = (MPAP - PCWP) \times 80/CO$	150-250 dynes•s•cm^{-5}
Pulmonary vascular resistance index: $PVRI = (MPAP - PCWP) \times 80/CI$	250-400 dynes•s•cm^{-5}/m^2

BSA = body surface area; CI = cardiac index; CO = cardiac output; CVP = central venous pressure; HR = heart rate; LVSWI = left ventricular stroke work index; MAP = mean arterial pressure; PAP = pulmonary arterial pressure; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; PVRI = pulmonary vascular resistance index; RVSWI = right ventricular stroke work index; SI = stroke index; SV = stroke volume; SVR = systemic vascular resistance; SVRI = systemic vascular resistance index.

III

of the lung (West's zone III), in which the pulmonary venous pressure exceeds the alveolar pressure. Otherwise, the PCWP will reflect the alveolar pressure, not the LAP. Because PEEP decreases the size of West's zone III, it has been shown to adversely affect the correlation between the PCWP and LAP, especially in the hypovolemic patient. Nevertheless, the correlation of PCWP and LAP could be maintained in the presence of PEEP by placing the catheter tip below the left atrium. The acute respiratory distress syndrome (ARDS) seems to prevent the transmission of increased alveolar pressure to the pulmonary interstitium. This preserves the relationship between the PCWP and LAP during the application of PEEP. It is not considered prudent, however, to temporarily disconnect patients from PEEP to measure preload.
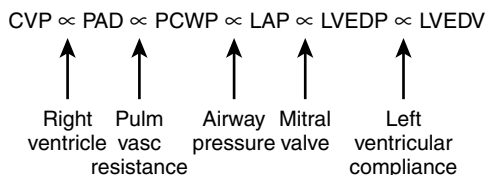
$$CVP \propto PAD \propto PCWP \propto LAP \propto LVEDP \propto LVEDV$$

| Right ventricle | Pulm vasc resistance | Airway pressure | Mitral valve | Left ventricular compliance |

Figure 9-6 The left ventricular end-diastolic volume (LVEDV) is related to left ventricular end-diastolic pressure (LVEDP) by the left ventricular compliance. The LVEDP is related to the left atrial pressure (LAP) by the diastolic pressure gradient across the mitral valve. The pulmonary capillary wedge pressure (PCWP) is related to the LAP by the pulmonary capillary resistance. The pulmonary artery diastolic pressure (PAD) is an estimate of the PCWP. The central venous pressure (CVP) reflects the PAD if right ventricular function is normal.

BOX 9-6 *Conditions Resulting in Discrepancies between Pulmonary Capillary Wedge Pressure and Left Ventricular End-Diastolic Pressure*

PCWP > LVEDP

- Positive-pressure ventilation
- Positive end-expiratory pressure
- Increased intrathoracic pressure
- Non–West lung zone III pulmonary artery catheter placement
- Chronic obstructive pulmonary disease
- Increased pulmonary vascular resistance
- Left atrial myxoma
- Mitral valve disease (e.g., stenosis, regurgitation)

PCWP < LVEDP

- Noncompliant left ventricle (e.g., ischemia, hypertrophy)
- Aortic regurgitation (premature closure of the mitral valve)

LVEDP > 25 mm Hg

LVEDP = left ventricular end-diastolic pressure; PCWP = pulmonary capillary wedge pressure.

Adapted from Tuman KJ, Carrol CC, Ivankovich AD: Pitfalls in interpretation of pulmonary artery catheter data. Cardiothorac Vasc Anesth Update 2:1, 1991.

The presence of large V waves in the PCWP tracing of patients with mitral regurgitation leads to an overestimation of the LVEDP. In patients with mitral stenosis, using the PCWP instead of the LAP to assess the transmitral gradient has been shown to overestimate the severity of mitral stenosis. However, when the PCWP was adjusted for the time delay through the pulmonary vasculature, the mean LAP and mean PCWP correlated well. It has been demonstrated that there is a significant positive gradient between the PCWP and the LAP in the initial hour after CPB. Box 9-6 is a summary of conditions that may alter the relationship between the PCWP and the LVEDP.

Placement of the Pulmonary Artery Catheter

The considerations for the insertion site of a PA catheter are the same as for CVP catheters. The right IJV approach remains the technique of choice because of the direct path between this vessel and the right atrium. The placement of PA catheters
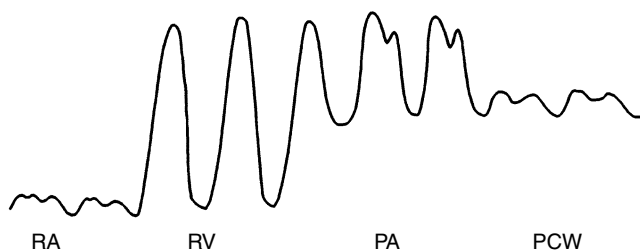
Figure 9-7 The waveforms encountered during the flotation of a PA catheter from the venous circulation to the pulmonary capillary wedge (PCW) position. Notice the sudden increase in systolic pressure as the catheter enters the right ventricle (RV), the sudden increase in diastolic pressure as the catheter enters the pulmonary artery (PA), and the decrease in mean pressure as the catheter reaches the PCW position. RA = right atrium.

through subclavian vein introducers may be complicated by kinking of the catheter when the sternum is retracted during cardiothoracic surgery.

Passage of the PA catheter from the vessel introducer to the PA can be accomplished by monitoring the pressure waveform from the distal port of the catheter or under fluoroscopic guidance. Waveform monitoring is the more common technique for perioperative right-sided heart catheterization. First, the catheter must be advanced through the vessel introducer (15 to 20 cm) before inflating the balloon. The inflation of the balloon facilitates further advancement of the catheter through the right atrium and right ventricle into the PA. Normal intracardiac pressures are shown in Table 9-1. The pressure waveforms seen during advancement of the PA catheter are illustrated in Figure 9-7. The RA waveform is seen until the catheter tip crosses the tricuspid valve and enters the right ventricle. In the right ventricle, there is a sudden increase in SBP but little change in DBP compared with the RA tracing. Arrhythmias, particularly premature ventricular complexes, usually occur at this point but almost always resolve without treatment once the catheter tip has crossed the pulmonary valve. The catheter is rapidly advanced through the right ventricle toward the PA.

As the catheter crosses the pulmonary valve, a dicrotic notch appears in the pressure waveform and there is a sudden increase in diastolic pressure. The PCWP tracing is obtained by advancing the catheter 3 to 5 cm farther until there is a change in the waveform associated with a drop in the measured mean pressure. Deflation of the balloon results in reappearance of the PA waveform and an increase in the mean pressure value. Using the right IJV approach, the right atrium is entered at 25 to 35 cm, the right ventricle at 35 to 45 cm, the PA at 45 to 55 cm, and the PCWP at 50 to 60 cm in most patients.

If the catheter does not enter the PA by 60 cm, the balloon should be deflated. It should be withdrawn into the right atrium, and another attempt should be made to advance the catheter into proper position. Excessive coiling of the catheter in the right ventricle should be avoided to prevent catheter knotting. The balloon should be inflated only for short periods to measure the PCWP. The PA waveform should be continually monitored to be certain that the catheter does not float out into a constant wedge position because this may lead to PA rupture or pulmonary infarction. The PA catheter is covered by a sterile sheath that must be secured at both ends to prevent contamination of the external portion of the catheter. Not infrequently, the PA catheter must be withdrawn a short distance because the extra catheter in the right ventricle floats out more peripherally into the PA over time as the catheter softens.

The time for the entire PA catheter insertion procedure is 10 to 15 minutes in experienced hands. Many clinicians believe that the PA catheter should be inserted before the induction of anesthesia. Studies showed that PA catheter insertion in the awake patient did not result in myocardial ischemia or deleterious hemodynamic

> **BOX 9-7** *Indications for Pulmonary Artery Catheterization*
>
> - Assessment of volume status
> - Cardiac output measurements
> - Hemodynamic measurements
> - Respiratory or oxygen transport measurements

changes after preanesthetic medication and with continuation of all preoperative cardiac medications. A chest radiograph should be obtained postoperatively in all patients to check the position of the PA catheter. Most catheters pass into the right middle or lower lobes. The location of the PA catheter can also be checked by TEE imaging of the catheter in the RA, RV, and pulmonary artery.

Indications

The indications for PA catheterization are given in Box 9-7. The ability of PA catheters to positively influence patient outcome has never been conclusively proved in large-scale, prospective studies. There remains considerable controversy regarding the risk/benefit ratio of PA catheters. Many studies have reported no change or even worse outcome in patients who were monitored with PA catheters. Randomized trials on patients with myocardial infarction seemed to confirm these data, whereas earlier prospective studies on surgical patients showed improved outcome.[9-11]

Major problems with PA catheter outcome studies include flaws in study design and insufficient statistical power. The most common design flaws were a lack of therapeutic protocols or treatment algorithms and inadequate randomization, which introduce observer bias.[12] Physician knowledge is another confounding variable, as demonstrated in multicenter studies that indicated competency in interpreting PA catheter–derived data was lacking in many individuals and depended on such factors as the level of training and the frequency of use. As many as 47% of physicians could not correctly determine the PCWP to within 5 mm Hg.

Another problem with PA catheter outcome studies is the clinical setting, specifically operating room versus ICU. Patients in the ICU might have disease too far advanced to make invasive hemodynamic monitoring useful. Studies that have reported improved outcome used invasive hemodynamic monitoring to optimize oxygen delivery in the perioperative period.

The operative procedures and medical conditions that are cited as indications for PA catheterization in the perioperative period remain controversial and vary by institution. In a global sense, the indications for using a PA catheter are assessing volume status, measuring CO, measuring $S\overline{v}o_2$, and deriving hemodynamic parameters. In 2003, the American Society of Anesthesiologists (ASA) Task Force on Pulmonary Artery Catheterization published updated practice guidelines for PA catheterization (http://www.asahq.org/publicationsAndServices/pulm_artery.pdf).[13] These guidelines emphasized that patient, surgery, and practice setting had to be considered. Generally, the routine use of PA catheters is indicated in high-risk patients (e.g., ASA 4 or 5) and high-risk procedures (e.g., where large fluid changes or hemodynamic disturbances are expected). The practice setting is important, because there is evidence that inadequate training or experience may increase the risk for perioperative complications associated with the use of a PA catheter. It is recommended that the routine use of a PA catheter should be confined to centers with adequate training and experience in the perioperative management of patients with the PA catheter (Box 9-8). A summary of procedural indications that is relatively aggressive is shown in Box 9-9.

BOX 9-8   *American Society of Anesthesiologists' Practice Guidelines for Pulmonary Artery Catheter Use*

Opinions

- PA catheterization provides new information that may change therapy, with poor clinical evidence of its effect on clinical outcome or mortality.
- There is no evidence from large, controlled studies that preoperative PA catheterization improves outcome regarding hemodynamic optimization.
- Perioperative PA catheter monitoring of hemodynamic parameters leading to goal-directed therapy has produced inconsistent data in multiple studies and clinical scenarios.
- Having immediate access to PA catheter data allows important preemptive measures for selected subgroups of patients who encounter hemodynamic disturbances that require immediate and precise decisions about fluid management and drug treatment.
- Experience and understanding are the major determinants of PA catheter effectiveness.
- PA catheterization is inappropriate as routine practice in surgical patients and should be limited to cases in which the anticipated benefits of catheterization outweigh the potential risks.
- PA catheterization can be harmful.

Recommendations

- The appropriateness of routine PA catheterization depends on a combination of patient-related, surgery-related, and practice setting–related factors.
- Perioperative PA catheterization should be considered in patients who present with significant organ dysfunction or major comorbidity that poses an increased risk for hemodynamic disturbances or instability (e.g., ASA IV or V patients).
- Perioperative PA catheterization in surgical settings should be considered based on the hemodynamic risk of the individual case rather than generalized surgical setting–related recommendations. High-risk surgical procedures are those in which large fluid changes or hemodynamic disturbances can be anticipated and procedures that are associated with a high risk of morbidity and mortality.
- Because of the risk of complications from PA catheterization, the procedure should not be performed by clinicians or nursing staff or done in practice settings where competency in safe insertion, accurate interpretation of results, and appropriate catheter maintenance cannot be guaranteed.
- Routine PA catheterization is not recommended when the patient, procedure, or practice setting poses a low or moderate risk for hemodynamic changes.

BOX 9-9   *Clinical Indications for Pulmonary Artery Catheter Monitoring*

Major Procedures Involving Large Fluid Shifts or Blood Loss in Patients with

- Severe unstable coronary artery disease or poor left ventricular function (congestive heart failure)
- Cardiogenic or septic shock or with multiple organ failure
- Right-sided heart failure, pulmonary hypertension, or pulmonary embolism
- Hemodynamic instability requiring inotropes or intra-aortic balloon counterpulsation
- Surgery of the aorta requiring cross-clamping
- Hepatic transplantation
- Massive ascites requiring major surgery

The use of the PA catheter has significantly contributed to the understanding and care of patients with cardiac disease. Nevertheless, further large-scale randomized controlled trials are needed to clearly define which, if any, patient populations benefit from PA catheter monitoring. The body of evidence is inconclusive. The risks associated with perioperative PA catheter monitoring may outweigh the benefits in low-to-moderate risk patients, whereas high-risk patients undergoing major surgery probably benefit from right-sided heart catheterization. One major caveat is that the data derived from PA catheter monitoring must be interpreted correctly and treatment protocols should be followed to derive maximal benefit. In summary, using an evidence-based medicine approach, PA catheter monitoring does not improve patient outcome in most patient populations and may actually be harmful in certain circumstances.

Complications

The complications associated with PA catheter placement include almost all of those detailed in the section on CVP placement. The ASA Task Force on Pulmonary Artery Catheterization concluded that serious complications due to PA catheterization occur in 0.1% to 0.5% of patients monitored with a PA catheter. Higher estimates are found in the literature and probably represent different patient populations, hospital settings, level of experience with PA catheter management, and other factors.

Arrhythmias

The most common complications associated with PA catheter insertion are transient arrhythmias, especially premature ventricular contractions (PVCs). However, fatal arrhythmias have rarely been reported. Intravenous lidocaine has been used in attempts to suppress these arrhythmias, with mixed results. However, a positional maneuver entailing 5-degree head-up and right lateral tilt was associated with a statistically significant decrease in malignant arrhythmias (compared with the Trendelenburg position) during PA catheter insertion.

Complete Heart Block

Complete heart block may develop during PA catheterization in patients with preexisting left bundle-branch block (LBBB). This potentially fatal complication is most likely due to electrical irritability from the PA catheter tip causing transient right bundle-branch block (RBBB) as it passes through the RV outflow tract. The incidence of developing RBBB was 3% in a prospective series of patients undergoing PA catheterization. However, none of the patients with preexisting LBBB developed complete heart block in that series. In another study of 47 patients with LBBB, complete heart block occurred in 2 patients with recent-onset LBBB. It is imperative to have an external pacemaker immediately available or to use a pacing PA catheter when placing a PA catheter in patients with LBBB.

Endobronchial Hemorrhage

Iatrogenic rupture of the PA has become more common since the advent of PA catheter monitoring in the ICU and operating room.[14] Several risk factors have emerged: advanced age, female sex, pulmonary hypertension, mitral stenosis, coagulopathy, distal placement of the catheter, and balloon hyperinflation. Balloon inflation in distal pulmonary arteries is probably accountable for most episodes of PA rupture because of the high pressures generated by the balloon. Hypothermic CPB may also increase risk due to distal migration of the catheter tip with movement of the heart and hardening of the PA catheter. It is now common practice to pull the PA catheter back 3 to 5 cm when CPB is instituted.

It is important to consider the cause of the hemorrhage when forming a therapeutic plan. If the hemorrhage is minimal and a coagulopathy coexists, correction of the coagulopathy may be the only necessary therapy. Protection of the uninvolved lung is of prime importance. Tilting the patient toward the affected side, placement of a double-lumen endotracheal tube, and other lung-separation maneuvers should protect the contralateral lung. Strategies proposed to stop the hemorrhage include the application of PEEP, placement of bronchial blockers, and pulmonary resection. The clinician is obviously at a disadvantage unless the site of hemorrhage is known. A chest radiograph will usually indicate the general location of the lesion. Although the cause of endobronchial hemorrhage may be unclear, the bleeding site must be unequivocally located before surgical treatment is attempted. A small amount of radiographic contrast dye may help to pinpoint the lesion if active hemorrhage is present. In severe hemorrhage and with recurrent bleeding, transcatheter coil embolization has been used. This may emerge as the preferred treatment method.

Pulmonary Infarction

Pulmonary infarction is a rare complication of PA catheter monitoring. An early report suggested that there was a 7.2% incidence of pulmonary infarction with PA catheter use. However, continuously monitoring the PA waveform and keeping the balloon deflated when not determining the PCWP (to prevent inadvertent wedging of the catheter) were not standard practice at that time. Distal migration of PA catheters may also occur intraoperatively owing to the action of the right ventricle, uncoiling of the catheter, and softening of the catheter over time. Inadvertent catheter wedging occurs during CPB because of the diminished RV chamber size and retraction of the heart to perform the operation. Embolization of thrombus formed on a PA catheter could also result in pulmonary infarction.

Catheter Knotting and Entrapment

Knotting of a PA catheter usually occurs as a result of coiling of the catheter within the right ventricle. Insertion of an appropriately sized guidewire under fluoroscopic guidance may aid in unknotting the catheter. Alternatively, the knot may be tightened and withdrawn percutaneously along with the introducer if no intracardiac structures are entangled. If cardiac structures, such as the papillary muscles, are entangled in the knotted catheter, then surgical intervention may be required. Sutures placed in the heart may inadvertently entrap the PA catheter.

Valvular Damage

Withdrawal of the catheter with the balloon inflated may result in injury to the tricuspid or pulmonary valves. Placement of the PA catheter with the balloon deflated may increase the risk of passing the catheter between the chordae tendineae. Septic endocarditis has also resulted from an indwelling PA catheter.

Pacing Catheters

The possible indications for placement of a pacing PA catheter are shown in Box 9-10. The actual use in a group of 600 patients undergoing cardiac surgery is shown in Table 9-3.[15]

A multipurpose PA catheter contains five electrodes for bipolar atrial, ventricular, or atrioventricular (AV) sequential pacing. The intraoperative success rates for atrial, ventricular, and AV sequential capture have been reported as 80%, 93%, and 73%, respectively.

The Paceport and A-V Paceport catheters have lumina for the introduction of a ventricular wire (Paceport) or for atrial and ventricular wires (A-V Paceport) for temporary transvenous pacing. The success rate for ventricular pacing capture was

III

BOX 9-10   *Indications for Perioperative Placement of Pacing Pulmonary Artery Catheters*

- Sinus node dysfunction or bradycardia
- Second-degree (Mobitz II) atrioventricular block
- Complete (third-degree) atrioventricular block
- Digitalis toxicity
- Need for atrioventricular sequential pacing
- Aortic stenosis (need to maintain sinus rhythm)
- Severe left ventricular hypertrophy or noncompliant left ventricle
- Idiopathic hypertrophic subaortic stenosis/hypertrophic obstructive cardiomyopathy
- Need for an intracardiac electrogram

Table 9-3   **Use of Pacing Pulmonary Artery Catheters According to the Presence or Absence of Different Indications**

Indication	Indication Present*	Indication Present/ Pacing PAC Used† (%)	Indication Absent*	Indication Absent/ Pacing PAC Used† (%)	P Value
Sinus node dysfunction	24	6 (25.0)	576	32 (5.5)	0.002
First-degree AV block	52	1 (1.9)	548	37 (6.7)	0.24
Second-degree AV block	1	1 (100)			
Complete AV block	15	5 (33.3)	585	33 (5.6)	0.001
LBBB	41	5 (12.1)	559	33 (5.9)	0.17
RBBB	32	0 (0)	568	38 (6.6)	0.25
LAH	17	1 (5.8)	583	37 (6.3)	1.0
RBBB and LAH	5	0 (0)	595	38 (6.3)	1.0
Reoperation/ with other indications present	61	14 (23.0)	539	24 (4.4)	<0.001
Reoperation/ no other indications present	51	1 (1.9)	549	37 (6.7)	0.24
Aortic stenosis	88	11 (12.0)	512	27 (5.2)	0.02
Mitral stenosis	17	1 (5.8)	583	37 (6.3)	1.0
Aortic insufficiency	40	9 (22.5)	560	29 (5.1)	<0.001
Mitral regurgitation	65	7 (10.7)	535	31 (5.7)	0.17

*Total number of patients.
†Total number and percentage of patients with or without each indication.
AV = atrioventricular; LAH = left anterior block; LBBB = left bundle-branch block; PAC = pulmonary artery catheter; RBBB = right bundle-branch block.
From Risk SC, Brandon D, D'Ambra MN, et al: Indications for the use of pacing pulmonary artery catheters in cardiac surgery. J Cardiothorac Vasc Anesth 6:275, 1992.

9

96% for the Paceport. The success rates for atrial and ventricular pacing capture before CPB were 98% and 100%, respectively, in a study of the A-V Paceport.

Mixed Venous Oxygen Saturation Catheters

Monitoring the $S\overline{v}o_2$ is a means of providing a global estimation of the adequacy of oxygen delivery relative to the needs of the various tissues. The formula for $S\overline{v}o_2$ calculation can be derived by modifying the Fick equation and assuming that the effect of dissolved oxygen in the blood is negligible:

$$S\overline{v}o_2 = Sao_2 - \frac{Vo_2}{CO \cdot 1.34 \cdot Hb}$$

A decrease in the $S\overline{v}o_2$ can indicate one of the following situations: decreased CO; increased oxygen consumption; decreased arterial oxygen saturation; or decreased hemoglobin (Hb) concentration. To measure $S\overline{v}o_2$, blood is aspirated from the distal port of the PA catheter slowly, so as not to contaminate the sample with oxygenated alveolar blood.

The addition of fiberoptic bundles to PA catheters has enabled the continuous monitoring of $S\overline{v}o_2$ using reflectance spectrophotometry. The catheter is connected to a device that includes a light-emitting diode and a sensor to detect the light returning from the PA. $S\overline{v}o_2$ is calculated from the differential absorption of various wavelengths of light by the saturated and desaturated hemoglobin.

If it is assumed that there is constant oxygen consumption and arterial oxygen content, changes in $S\overline{v}o_2$ should reflect changes in CO. Several investigators have come to the conclusion that it provides a valuable measure of CO during surgery. The $S\overline{v}o_2$ has been shown to correlate with cardiac index during CABG surgery when oxygen consumption is constant, but not with the cardiac index when oxygen consumption is changing, such as during shivering after anesthesia. The usefulness of the catheter may primarily be its ability to continuously monitor the balance between oxygen delivery and consumption,[16] and $S\overline{v}o_2$ may also help predict survival after acute myocardial infarction.

CARDIAC OUTPUT MONITORING

The CO is the amount of blood pumped to the peripheral circulation by the heart each minute. It is a measurement that reflects the status of the entire circulatory system, not just the heart, because it is governed by autoregulation from the tissues. The CO is equal to the product of the SV and the heart rate. Preload, afterload, heart rate, and contractility are the major determinants of the CO. The measurement of CO is of particular interest in patients with cardiac disease.

Indicator Dilution

The indicator dilution method is based on the observation that, for a known amount of indicator introduced at one point in the circulation, the same amount of indicator should be detectable at a downstream point. The amount of indicator detected at the downstream point is equal to the product of CO and the change in indicator concentration over time. CO is calculated using the Stewart-Hamilton equation:

$$CO = I \times 60 \Big/ \oint C \, dt$$

in which CO is cardiac output, I is amount of indicator injected, and ∫C dt is the integral of indicator concentration over time (60 converts seconds to minutes).

Cold saline (i.e., thermodilution) or lithium ions are used as the indicator, whereas dye (e.g., indocyanine green) or radioisotopes are rarely used in current practice. Blood flow is directly proportional to the amount of the indicator delivered and inversely proportional to the amount of indicator that is present at a sampling site distal to the injection site.

Thermodilution

INTERMITTENT THERMODILUTION CARDIAC OUTPUT

The thermodilution method, using the PA catheter, is the most commonly used method at present for invasively measuring CO in the clinical setting. With this technique, multiple COs can be obtained at frequent intervals using an inert indicator, and without blood withdrawal. A bolus of cold fluid is injected into the right atrium, and the resulting temperature change is detected by the thermistor in the PA. When a thermal indicator is used, the modified Stewart-Hamilton equation is used to calculate CO:

$$CO = \frac{V(T_B - T_I) \times K_1 \times K_2}{\int_0^\infty \Delta T_B(t)dt}$$

in which CO is the cardiac output (L/min), V is the volume of injectate (mL), T_B is the initial blood temperature (°C), T_I is the initial injectate temperature (°C), K_1 is the density factor, K_2 is the computation constant, and $\int_0^\infty \Delta T_B(t)dt$ is the integral of blood temperature change over time.

Solution of this equation is performed by a computer that integrates the area under the temperature versus time curve. CO is inversely proportional to the area under the curve.

The temperature-versus-time curve is the crux of this technique, and any circumstances that affect it have consequences for the accuracy of the CO measurement. Specifically, anything that results in less "cold" reaching the thermistor, more "cold" reaching the thermistor, or an unstable temperature baseline will adversely affect the accuracy of the technique. Less "cold" reaching the thermistor would result in overestimation of the CO. This could be caused by a smaller amount of indicator, an indicator that is too warm, a thrombus on the thermistor, or partial "wedging" of the catheter. Conversely, underestimation of the CO will occur if excessive volume of injectate, or injectate that is too cold, is used to perform the measurement. Intracardiac shunts have unpredictable effects that depend on the anatomy and physiology of individual patients. Variations of up to 80% in measured CO occur when the rate of administration of intravenous crystalloid infusions caused fluctuations in baseline blood temperature. The rapid temperature decrease seen after hypothermic CPB has been shown to result in the underestimation of CO by 0.6 to 2.0 L/min. The normal changes in the PA that occur with each respiratory cycle appear to be exaggerated in the early phase after hypothermic CPB. This may cause peak-to-peak errors in estimation of intermittent CO of up to 50% if initiated at different times during the ventilatory cycle. This effect was significantly decreased with thermal equilibration, approximately 30 minutes after CPB. This problem is less prevalent currently, because hypothermic CPB is used less commonly.

The precision of the thermodilution CO technique is not very good but can be improved by ensuring that, for each determination, the rate and duration of the injection are kept as constant as possible. Whenever possible, 10-mL volumes of

injecate should be used and the timing of the injection in the respiratory cycle should be the same.

CONTINUOUS THERMODILUTION CARDIAC OUTPUT

Pulmonary artery catheters with the ability to measure CO continuously were introduced into clinical practice in the 1990s. The method that has gained the most clinical use functions by mildly heating the blood in a pseudorandom stochastic fashion. In vitro as well as in vivo studies have shown that a good correlation exists between this method and other measures of CO.

There was a poor correlation between intermittent and continuous thermodilution CO ($r = 0.273$) in the first 45 minutes after CPB. In contrast, there was an excellent correlation between intermittent and continuous CO measurements obtained in more physiologically stable periods. Perhaps the reason for this observation lies in the unstable thermal baseline after hypothermic CPB.

The routine use of continuous CO catheters in cardiac surgery patients has not been shown to improve outcome, and they are more expensive than standard PA catheters. Bolus thermodilution CO still holds its place as the gold standard of CO measurements in the clinical setting.[17]

ARTERIAC PRESSURE-BASED CARDIAC OUTPUT

Recently, the FloTrac/Vigileo system has been introduced into clinical pratice. It is a unique system that measures CO from the radial artery catheter and does not require calibration. This device has been shown to be accurate in cardiac surgial patients. It measures beat-to-beat stroke volume, and calculate stroke volume variation as a predictor of fluid responsiveness or dynamic preload. Abnormal arterial pressure waveforms (eg: aortic regurgitation) will lead to incorrect CO measurements with this pulse contour technique.[18]

ANALYSIS AND INTERPRETATION OF HEMODYNAMIC DATA

III

The information provided by hemodynamic monitoring permits the calculation of various derived parameters that assist in evaluating patients clinically. The formulas, normal values, and units for the calculation of various hemodynamic parameters are presented in Table 9-2. These parameters include the SVR, pulmonary vascular resistance (PVR), SV, left ventricular stroke work (LVSW), and right ventricular stroke work (RVSW). As an example of information that may be obtained, graphs of PCWP versus SV can be constructed for individual patients; these "Starling curves" provide insight into the contractile state of the heart. Although these parameters are easily derived using the standard formulas, many modern monitors perform these calculations. To compare data among patients of different body weights and types, the various hemodynamic parameters may be normalized by indexing them to body surface area.

Systemic and Pulmonary Vascular Resistances

Systemic vascular resistance represents an estimation of the afterload of the left ventricle. Afterload is roughly defined as the force that impedes or opposes ventricular contraction. Higher SVR results in increased LV systolic wall stress. This has clinical significance because LV wall stress is one of the major determinants of

myocardial oxygen consumption. Elevations in wall stress have been observed in patients with LV enlargement due to systemic hypertension, aortic stenosis, and aortic regurgitation.

Clinically, calculations of SVR are used to assess the response to inotropic, vaso-dilatory, and vasoconstrictive agents.[19] For example, a patient who is hypotensive despite a high normal CO has a low SVR. The SVR is calculated, and then therapy is instituted (e.g., a vasoconstrictor). A repeat calculation of the SVR enables the clinician to titrate the therapy to the appropriate endpoint. Despite this common use in the operating room and ICU setting, there is good evidence that SVR is not an accurate indicator of true afterload. Nevertheless, SVR remains the clinical technique for measuring afterload at the present time.

PVR remains the traditional measure of afterload of the right ventricle. Systolic PAP may provide a better estimation of RV afterload. PVR and PAP do provide some clinically useful information regarding the pulmonary vasculature and are readily available in patients with PA catheters. The PVR should be used in conjunction with other hemodynamic data to assess the response of the pulmonary vasculature to pharmacologic therapy and physiologic changes.

Frank-Starling Relationships

Myocardial function depends on the contractile state and the preload of the ventricle (sarcomere length at end-diastole). The relationship between the ventricular preload and myocardial work (ventricular stroke work) is the Frank-Starling relationship. The slope of the curve indicates the contractile state of the myocardium (Fig. 9-8). For clinical purposes, it is usually not feasible to measure actual end-diastolic volumes (can be estimated with TEE), and approximations of end-diastolic pressure, such as the PCWP or LAP, are often substituted. This introduces error, because the relationship between end-diastolic pressure and volume is usually nonlinear (as described by the diastolic ventricular compliance curve) and is dynamic. Unfortunately, the Frank-Starling relationship is extremely sensitive to changes in afterload. Patients with LV or RV dysfunction may have severe decrements in SV with increased SVR or PVR, respectively.

MONITORING CORONARY PERFUSION

The coronary perfusion pressure (CPP) is usually defined as the aortic diastolic blood pressure (DAP) minus the LVEDP:

$$CPP = DAP - LVEDP$$

Elevation of the LVEDP will decrease the gradient of blood flow to the vulnerable subendocardial tissue during diastole as will a decrease in the DBP. If coronary artery disease is present, significant stenosis will decrease the coronary artery DBP well below the aortic DBP, and elevation of LVEDP can seriously jeopardize the subendocardium. An increase in the LVEDP is detrimental in two ways: decreased coronary blood flow and increased myocardial oxygen demand ($M\bar{v}o_2$), which explain the severe ischemia seen with overdistention of the left ventricle. Tachycardia is also extremely detrimental because it decreases coronary filling time and increases oxygen demand. Subendocardial ischemia is commonly produced by a combination of tachycardia and elevated LVEDP.
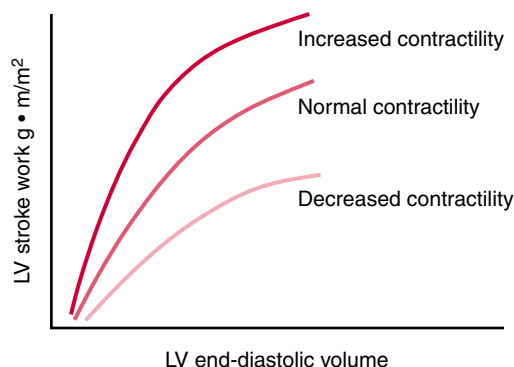
Figure 9-8   The graph of left ventricular (LV) stroke work versus LV preload (i.e., end-diastolic volume) is known as the Frank-Starling relationship. Shifting of the curve upward and to the left represents increased contractility. Shifting of the curve downward and to the right represents decreased contractility. Alterations in afterload significantly affect the curve.

ELECTROCARDIOGRAPHY

Interpretation of the ECG is often considered the domain of the cardiologist, but clinicians providing perioperative and critical care also derive important information from it by the standard 12-lead tracing or as a continuous "monitoring" modality.

The practicing anesthesiologist relies on the ECG to make critical decisions at many phases of the perioperative period in patients undergoing cardiac or noncardiac (particularly vascular) surgery.[20]

Lead Systems

Einthoven established electrocardiography using three extremities as references: the left arm, right arm, and left leg. He recorded the difference in potential between the left arm and right arm (lead I), between the left leg and right arm (lead II), and between the left leg and left arm (lead III). Because the signals recorded were differences between two electrodes, these leads were called *bipolar*. The positive or negative polarity of each of the limbs was chosen by Einthoven to result in positive deflections of most of the waveforms and has no innate physiologic significance. He postulated that the three limbs defined an imaginary equilateral triangle with the heart at its center.

Wilson refined and introduced the unipolar precordial leads into clinical practice. To implement these leads, he postulated a mechanism whereby the absolute level of electrical potential could be measured at the site of the exploring precordial electrode (the positive electrode). A negative pole with zero potential was formed by joining the three limb electrodes in a resistive network in which equally weighted signals cancel each other out. He called this the *central terminal*. He described three additional limb leads (VL, VR, and VF). These leads measured new vectors of activation, and in this way the hexaxial reference system for determination of electrical axis was established. He subsequently introduced the six unipolar precordial V leads in 1935 (Fig. 9-9).

Detection of Myocardial Ischemia

Pathophysiology of ST-Segment Responses

The ST segment is the most important portion of the QRS complex for evaluating ischemia (Box 9-11). The origin of this segment, at the J point, is easy to locate. Its end, which is generally accepted as the beginning of any change of slope of the
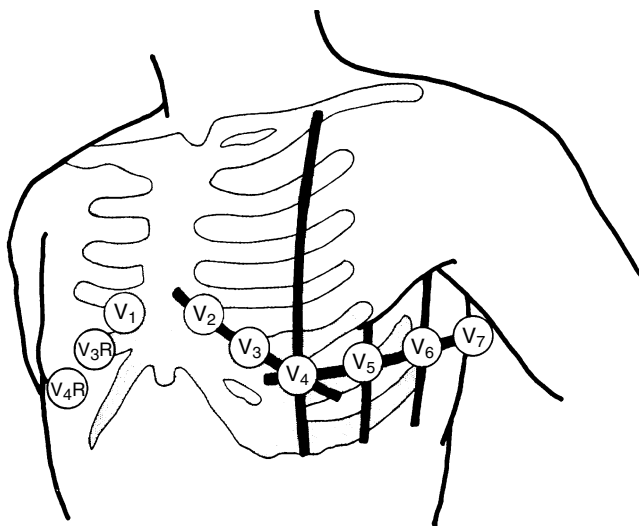
Figure 9-9 The locations of the precordial leads. *Heavy vertical lines* represent the midclavicular, anterior, axillary, and midaxillary lines (*from left to right*). V_1 and V_2 are referenced to the fourth intercostal space and V_4 to the fifth space. V_3 lies on a line between V_2 and V_4. V_5 and V_6 lie on a horizontal line from V_4. Additional precordial leads can be obtained on the right side (V_3R, V_4R), as well as extending farther left from V_6 (V_7). (From Friedman HH: Diagnostic Electrocardiography and Vectorcardiography. New York, McGraw-Hill, 1985, p 41; with permission of The McGraw-Hill Companies.)

BOX 9-11 *Monitoring for Myocardial Ischemia*

- (Preoperative) clinical symptoms
- Electrocardiogram (leads II, V_3, V_4, V_5)
- Transesophageal echocardiography
- Pulmonary artery catheterization

9

T wave, is more difficult to determine. In normal individuals there may be no discernible ST segment as the T wave starts with a steady slope from the J point, especially at rapid heart rates. The TP segment has been used as the isoelectric baseline from which changes in the ST segment are evaluated, but with tachycardia this segment is eliminated, and during exercise testing the PR segment is used. The PR segment is used in all ST-segment analyzers.

Repolarization of the ventricle proceeds from the epicardium to the endocardium, opposite to the vector of depolarization. The ST segment reflects the midportion, or phase 2, of repolarization during which there is little change in electrical potential. It is usually isoelectric. Ischemia causes a loss of intracellular potassium, resulting in a *current of injury*. The electrophysiologic mechanism accounting for ST-segment shifts (elevation or depression) remains controversial. The two major theories are based on a loss of resting potential as current flows from the uninjured to the injured area (i.e., diastolic current) and on a true change in phase 2 potential as current flows from the injured to the uninjured area (i.e., systolic current). With subendocardial injury, the ST segment is depressed in the surface leads. With epicardial or transmural injury, the ST segment is elevated.[21,22]

Electrocardiographic Manifestations of Ischemia

With myocardial ischemia, repolarization is affected, resulting in downsloping or horizontal ST-segment depression. Various local effects and differences in vectors during repolarization result in different ST morphologies that are recorded by the different leads. It is generally accepted that ST changes in multiple leads are associated with more severe degrees of coronary artery disease.

The classic criterion for ischemia is 0.1 mV (1 mm) of ST-segment depression measured 60 to 80 ms after the J point.[23-25] The slope of the segment must be horizontal or downsloping. Downsloping depression may be associated with a greater number of diseased vessels and a worse prognosis than horizontal depression. Slowly upsloping depression with a slope of 1 mV/s or less is also used but is considered less sensitive and specific (and difficult to assess clinically). Nonspecific ST-segment depression can be related to drug use, particularly digoxin. Interpretation of ST-segment changes in patients with LV hypertrophy is particularly controversial given the tall R-wave baseline, J-point depression, and steep slope of the ST segment. Although a number of studies have excluded such patients, others (including those using other modalities or epidemiologic studies) observed that LV hypertrophy is a highly significant predictor of adverse cardiac outcome.

The criteria for ischemia with ST-segment elevation ($\geq$0.1 mV in $\geq$2 contiguous leads) are used in conjunction with clinical symptoms or elevation of biochemical markers to diagnose acute coronary syndromes. It usually results from transmural ischemia, but it may potentially represent a *reciprocal change* in a lead oriented opposite to the primary vector with subendocardial ischemia. Perioperative ambulatory monitoring studies have also included more than 0.2 mV in any single lead as a criterion, but ST-segment elevation is rarely reported in the setting of noncardiac surgery. It is commonly observed, however, during weaning from CPB in cardiac surgery and during CABG surgery (on and off pump) with interruption of coronary flow in a native or graft vessel. ST-segment elevation in a Q-wave lead should not be analyzed for acute ischemia, although it may indicate the presence of a ventricular aneurysm.

Clinical Lead Systems for Detecting Ischemia

Early clinical reports of intraoperative monitoring using the V_5 lead in high-risk patients were based on observations during exercise testing, in which bipolar configurations of V_5 demonstrated high sensitivity for myocardial ischemia detection (up to 90%). Subsequent studies using 12-lead monitoring (torso mounted for stability during exercise) confirmed the sensitivity of the lateral precordial leads. Some studies, however, reported higher sensitivity for leads V_4 or V_6 compared with V_5, followed by the inferior leads (in which most false-positive responses were reported).

The factors responsible for precipitating ischemia during exercise testing and surgical settings may differ. For example, during exercise stress testing, most ischemia is demand related, whereas in the perioperative period a larger proportion may be related to reduced oxygen supply. The most sensitive leads during exercise testing, however, are useful in the perioperative setting.

Intraoperative Lead Systems

Detection of perioperative myocardial ischemia has received considerable attention over the past several decades and more recently with publication of several studies of clinical monitoring and therapy (e.g., perioperative β-blockade). Many of these studies demonstrated associations of perioperative ischemia with adverse cardiac

outcomes in adults undergoing a variety of cardiac and noncardiac surgical procedures. The ease of use of new ST-segment trending software in operating room monitors has resulted in its routine use.

The recommended leads for intraoperative monitoring, based on several clinical studies, do not differ substantially from those used during exercise testing.[26] Clinical studies using continuous 12-lead ECG analysis reported that almost 90% of responses involved ST-segment depression alone (75% in V_5 and 61% in V_4). In approximately 70% of patients, significant changes were observed in multiple leads. The sensitivity of each of the 12 leads is shown in Figure 9-10. When considered in combination (as occurs clinically), the use of leads V_4 and V_5 increased sensitivity to 90%, whereas sensitivity for the standard clinical combination of leads II and V_5 was 80%. Use of leads V_2 through V_5 and lead II captured all episodes (Table 9-4).

A larger clinical study of patients undergoing vascular surgery using a longer period of monitoring (up to 72 hours) with more specific criteria for ischemia (>10 minute duration of episode) extended these observations. It was reported that V_3 was most sensitive for ischemia (87%) followed by V_4 (79%), whereas V_5 alone was only 66% sensitive. In the subgroup of patients in whom prolonged ischemic episodes ultimately culminated in infarction, V_4 was most sensitive (83%). In this study, all myocardial infarctions were non–Q-wave events detected by troponin elevation. Use of two precordial leads detected 97% to 100% of changes. Based on analysis of the resting isoelectric levels of each of the 12 leads (a unique component of this study), it was recommended that V_4 was the best single choice for monitoring of a single precordial lead, because it was most likely to be isoelectric relative to the resting 12-lead preoperative ECG. In contrast, the baseline ST segment was more likely above isoelectric in V_1 through V_3 and below isoelectric in V_5 and V_6. Surprisingly, no episodes of ST elevation occurred in this study.

A cohort of vascular patients monitored in the ICU for the first postoperative day with continuous 12-lead monitoring used a threshold of 20 minutes for an ischemic
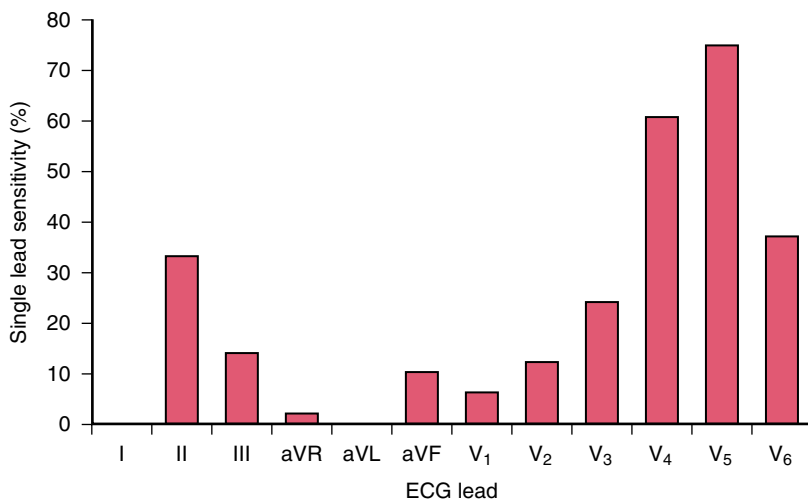

Figure 9-10 Single-lead sensitivity for the intraoperative detection of ischemia based on 51 episodes detected in 25 patients undergoing noncardiac surgery. Sensitivity was calculated by dividing the number of episodes detected in that lead by the total number of episodes. Sensitivity was greatest in lead V_5, and the lateral leads (I, aVL) were insensitive. (From London MJ, Hollenberg M, Wong MG, et al: Intraoperative myocardial ischemia: Localization by continuous 12-lead electrocardiography. Anesthesiology 69:232, 1988.)

Table 9-4   Sensitivity for Different Electrocardiographic Lead Combinations

Number of Leads	Combination	Sensitivity (%)
1 lead	II	33
	V_4	61
	V_5	75
2 leads	II/V_5	80
	II/V_4	82
	V_4/V_5	90
3 leads	V_3/V_4/V_5	94
	II/V_4/V_5	96
4 leads	II/V_2-V_5	100

Data from London MJ, Hollenberg M, Wong MG, et al: Intraoperative myocardial ischemia: Localization by continuous 12-lead electrocardiography. Anesthesiology 69:232, 1988.

episode. Eleven percent of 149 patients met the criteria, with ST-segment depression in 71% and ST-segment elevation alone in 18% (12% had both). Most changes were detected in V_2 (53%) and V_3 (65%). Using the standard two-lead system (II and V_5), only 41% of episodes would have been detected.

The use of multiple precordial leads, although appealing, is not likely to become common clinical practice, owing to the limitations of existing monitors (and cables). Even if such equipment were available, it is likely that considerable resistance would occur from practitioners due to the extra effort associated with this approach. Perhaps, in the future, when lower cost wireless technologies are perfected, this approach may become a clinical reality; in the meantime, a combination of leads V_5 and II remains the clinical choice of most practitioners.

Arrhythmia and Pacemaker Detection

Use of inferior leads allows superior discrimination of P-wave morphology, facilitating visual diagnosis of arrhythmias and conduction disorders. Although esophageal (and even intracardiac) leads allow the greatest sensitivity in detecting P waves, these are rarely used clinically. Nevertheless, they should be kept in mind for difficult diagnoses. With the increasing use of implantable defibrillators and automatic external defibrillators to treat ventricular fibrillation and ventricular tachycardia, there is considerable interest in the refinement of arrhythmia detection algorithms and their validation. As expected, the devices' accuracy for detecting ventricular arrhythmias is high but is much lower for detecting atrial arrhythmias. In the settings of critical care and ambulatory monitoring, a variety of artifacts are common causes of false-positive responses.

Detection of pacemaker spikes may be complicated by very-low-amplitude signals related to bipolar pacing leads, amplitude varying with respiration, and total-body fluid accumulation. Most critical care and ambulatory monitors incorporate pacemaker spike enhancement for small high-frequency signals (typically 5 to 500 mV with 0.5- to 2-ms pulse duration) to facilitate recognition. However, this can lead to artifact if there is high-frequency noise within the lead system.

SUMMARY

- Patients with severe cardiovascular disease and those undergoing surgery associated with rapid hemodynamic changes should be adequately monitored at all times.
- Adequate monitoring is based on specific patient, surgical, and environmental factors.
- Standard monitoring for cardiac surgery patients includes invasive blood pressure, electrocardiography, central venous pressure, urine output, temperature, capnometry, pulse oximetry, and intermittent blood gas analysis.
- Electrocardiography remains the gold standard for myocardial ischemia monitoring. Thermodilution PA catheters and left atrial pressure catheters are invasive monitors. The risks and benefits should be considered for each patient.
- The Society of Cardiovascular Anesthesiologists and the American Society of Echocardiography have published recommendations for intraoperative PA catheter and TEE use in various clinical settings.
- Evidence-based data on clinical outcome and monitoring in cardiac anesthesia are difficult to obtain owing to difficulties in conducting large prospective trials.

REFERENCES

1. Dorje P, Tremper K: Systolic pressure variation: A dynamic measure of the adequacy of intravascular volume. Semin Anesth Periop Med Pain 24:147, 2005
2. Barbeau GR, Arsenault F, Dugas L, et al: Evaluation of the ulnopalmar arterial arches with pulse oximetry and plethysmography: Comparison with the Allen's test in 1010 patients. Am Heart J 147:489, 2004
3. Armstrong PJ, Han DC, Baxter JA, et al: Complication rates of percutaneous brachial artery access in peripheral vascular angiography. Ann Vasc Surg 17:107, 2003
4. Hind D, Calvert N, McWilliams R, et al: Ultrasonic locating devices for central venous cannulation: Meta-analysis. BMJ 327:361, 2003
5. Riopelle J, Ruiz D, Hunt J, et al: Circumferential adjustment of ultrasound probe position to determine the optimal approach to the internal jugular vein: A noninvasive geometric study in adults. Anesth Analg 100:512, 2005
6. Troianos CA, Kuwik R, Pasqual J, et al: Internal jugular vein and carotid artery anatomic relation as determined by ultrasonography. Anesthesiology 85:43, 1996
7. Parry G: Trendelenburg position, head elevation and a midline position optimize right internal jugular vein diameter. Can J Anaesth 51:379, 2004
8. Pawar M, Mehta Y, Kapoor P, et al: Central venous catheter-related bloodstream infections: Incidence, risk factors, outcome, and associated pathogens. J Cardiothorac Vasc Anesth 18:304, 2004
9. Sandham JD: Pulmonary artery catheter use—refining the question. Crit Care Med 32:1070, 2004
10. Chittock DR, Dhingra VK, Ronco JJ, et al: Severity of illness and risk of death associated with pulmonary artery catheter use. Crit Care Med 32:911, 2004
11. Sandham JD, Hull RD, Brant RF, et al: A randomized, controlled trial of the use of pulmonary artery catheters in high-risk surgical patients. N Engl J Med 348:5, 2003
12. Vender J: Pulmonary artery catheter utilization: The use, misuse, or abuse, J Cardiothorac Vasc Anesth 20:295-299, 2006
13. American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization: Practice guidelines for pulmonary artery catheterization: An updated report by the American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. Anesthesiology 99:988, 2003
14. Abreu AR, Campos MA, Krieger BP: Pulmonary artery rupture induced by a pulmonary artery catheter: A case report and review of the literature. J Intensive Care Med 19:291, 2004
15. Risk SC, Brandon D, D'Ambra MN, et al: Indications for the use of pacing pulmonary artery catheters in cardiac surgery. J Cardiothorac Vasc Anesth 6:275, 1992
16. Reinhart K, Kuhn HJ, Hartog C, Bredle DL: Continuous central venous and pulmonary artery oxygen saturation monitoring in the critically ill. Intensive Care Med 30:1572, 2004
17. Zollner C, Goetz AE, Weis M, et al: Continuous cardiac output measurements do not agree with conventional bolus thermodilution cardiac output determination. Can J Anaesth 48:1143, 2001

18. Manecke GR: Cardiac output from the arterial catheter: Deceptively simple. J Cardiothorac Vasc Anesth 21:629, 2007
19. Bashore T: Afterload reduction in chronic aortic regurgitation: It sure seems like a good idea. J Am Coll Cardiol 45:1031, 2005
20. Weinfurt PT: Electrocardiographic monitoring: An overview. J Clin Monit 6:132, 1990
21. Tsuda H, Tobata H, Watanabe S, et al: QRS complex changes in the V_5 ECG lead during cardiac surgery. J Cardiothorac Vasc Anesth 6:658, 1992
22. Crescenzi G, Scandroglio AM, Pappalardo F, et al: ECG changes after CABG: The role of the surgical technique. J Cardiothorac Vasc Anesth 18:38, 2004
23. Horacek BM, Wagner GS: Electrocardiographic ST-segment changes during acute myocardial ischemia. Card Electrophysiol Rev 6:196, 2002
24. Carley SD: Beyond the 12 lead: Review of the use of additional leads for the early electrocardiographic diagnosis of acute myocardial infarction. Emerg Med (Fremantle) 15:143, 2003
25. Zimetbaum PJ, Josephson ME: Use of the electrocardiogram in acute myocardial infarction. N Engl J Med 348:933, 2003
26. London MJ: Multilead precordial ST-segment monitoring: "The next generation?" Anesthesiology 96:259, 2002

III