

CHAPTER 20 Cardiac Catheterization

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Indications for Diagnostic Cardiac Catheterization

As with any procedure, the decision to recommend cardiac catheterization is based on an appropriate risk/benefit ratio. In general, diagnostic cardiac catheterization is recommended whenever it is clinically important to define the presence or severity of a suspected cardiac lesion that cannot be evaluated adequately by noninvasive techniques. Because the risk of a major complication from cardiac catheterization is less than 1% with mortality of less than 0.08%, there are few patients who cannot be studied safely in an active laboratory. Intracardiac pressure measurements and coronary arteriography are procedures that can be performed with reproducible accuracy best by invasive catheterization. Noninvasive estimation of intracardiac pressures can be obtained with echocardiography (see Chap. 15). Coronary computed tomography (CT) angiography can also be used for assessment of coronary anatomy (see Chap. 19) and provides complementary information of plaque distribution and composition. However, current limitations of spatial resolution, heart rate variability, patient cooperation, and radiation dosing limit the ability of CT to replace cardiac catheterization.

To understand the various indications for diagnostic cardiac catheterization, integration of knowledge from multiple American College of Cardiology/American Heart Association (ACC/AHA) guidelines is necessary. The diagnostic coronary angiography guidelines¹ have been updated by other groups that have addressed specific cardiac catheterization indications, including the guidelines for management of patients with valvular heart disease,² chronic heart failure,³ ST elevation myocardial infarction,⁴ percutaneous coronary intervention⁵ and coronary artery bypass surgery,⁶ unstable angina or non-ST elevation myocardial infarction,⁷ and congenital heart disease.⁸

Indications for cardiac catheterization include divergent populations. At one extreme, many critically ill and hemodynamically unstable patients are evaluated during acute coronary syndromes, severe heart failure, or cardiogenic shock. At the other end of the spectrum, many procedures are performed in an outpatient setting. These settings include hospitals with or without cardiac surgical capability and freestanding or mobile laboratories.⁸

Cardiac catheterization should be considered a diagnostic test used in combination with complementary noninvasive tests. For example, cardiac catheterization in patients with valvular or congenital heart disease is best performed with full prior knowledge of noninvasive imaging and functional information. This allows catheterization to be directed and simplified without obtaining redundant anatomic information that is reliably available through echocardiography, cardiac magnetic resonance (see Chap. 18), or CT.

Identification of coronary artery disease and assessment of its extent and severity are the most common indications for cardiac catheterization in adults. The information obtained is crucial to optimize the selection of mechanical or medical therapy. In addition, dynamic coronary vascular lesions, such as spasm, myocardial bridging, and plaque rupture with thrombosis, can be identified. The consequences of coronary heart disease, such as ischemic mitral regurgitation and left ventricular (LV) dysfunction, can be defined. During percutaneous catheter intervention for acute coronary syndromes, patients are studied during evolving acute myocardial infarction, with unstable angina, or in the early period after acute myocardial injury. The optimal timing for catheterization and revascularization has been described in various guidelines²⁻⁸ (see Chaps. 21, 55, 57, and 58).

In patients with myocardial disease and LV dysfunction, cardiac catheterization provides critical information. It can evaluate whether coronary artery disease is the cause of symptoms and quantify LV function, right-heart pressures, and cardiac outputs. In patients with angina and impaired LV function, noninvasive testing is of limited value and clinicians should proceed directly to coronary angiography.³ Cardiac catheterization also permits quantification of the severity of both diastolic and systolic dysfunction, differentiation of myocardial restriction from pericardial constriction, assessment of the extent of valvular regurgitation, and assessment of the cardiovascular response to acute pharmacologic intervention.

In patients with valvular heart disease, cardiac catheterization provides data both confirmatory of and complementary to noninvasive echocardiography, cardiac magnetic resonance, and nuclear studies (see Chap. 66). Cardiac catheterization can define the severity of valvular stenosis or regurgitation, particularly when noninvasive studies are inconclusive or the results are disparate to clinical findings. Knowledge of coronary artery anatomy is critical in most adults older than 35 years when valve surgery is planned.² However, catheterization may be unnecessary in some preoperative situations, such as for patients with an atrial myxoma or young patients (<35 years) with endocarditis or acute valvular regurgitation. The identification of anomalies, the quantification of the hemodynamic consequences of the valvular lesions (such as pulmonary hypertension), and the acute hemodynamic response to pharmacologic therapy can provide useful preoperative information that helps define the operative risk and response to surgery and permits a more directed surgical approach.²

The current role of cardiac catheterization in certain congenital disease states has been addressed in guidelines for adults with congenital heart disease (see Chap. 65). Echocardiography with Doppler study and cardiac magnetic resonance often provide adequate information. Because gross cardiac anatomy can generally be well defined by these methods, catheterization is required only if certain hemodynamic information (e.g., quantification of shunt size,

**TABLE 20-1** Relative Contraindications to Diagnostic Cardiac Catheterization

Acute gastrointestinal bleeding
Severe hypokalemia
Uncorrected digitalis toxicity
Anticoagulation with international normalized ratio >1.8 or severe coagulopathy
Previous anaphylactoid reaction to contrast media
Acute stroke
Acute renal failure or severe chronic non-dialysis-dependent kidney disease
Unexplained fever or untreated active infection
Severe anemia
Uncooperative patient

pulmonary vascular resistance, and reversibility of pulmonary arterial hypertension with a vasodilator) is important in determining the indications for surgical procedures or if percutaneous interventional methods are being used.

There is no true absolute contraindication to cardiac catheterization other than refusal of the competent patient. The procedure can be successfully performed even in the most critically ill patient with a relatively low risk. The relative contradictions to cardiac catheterization are summarized in **Table 20-1**.

Technical Aspects of Cardiac Catheterization

Catheterization Laboratory Facilities

Cardiac catheterization facilities have several venues, including traditional hospital-based laboratories with in-house cardiothoracic surgical programs, hospital-based laboratories without on-site surgical programs, freestanding laboratories, and mobile laboratories. At present, about 75% of cardiac catheterization laboratories have on-site surgical backup. The goal of the freestanding and mobile cardiac catheterization facilities is to reduce cost while offering services in a convenient location for low-risk patients. The safety of mobile catheterization in properly selected low-risk patients has been well established and appears comparable to other settings.

As a result of the documented safety and cost-effectiveness of diagnostic cardiac catheterization in the outpatient setting, there has been increasing use of this approach. About 50% of hospital-based practices are currently performed as outpatient procedures. In general, patients who require preprocedural hospitalization for diagnostic catheterization are uncommon. These include patients with severe congestive heart failure and patients with congestive heart failure renal insufficiency requiring prehydration. The need for hospitalization to change from warfarin to heparin has been replaced by use of low-molecular-weight heparin as an outpatient bridge for anticoagulation.²

Noninvasive testing can identify patients who would be more appropriately evaluated in a setting where cardiac surgery is available. This includes severe ischemia discovered during stress testing, ischemia at rest, known or highly suspected severe left main or proximal three-vessel disease, critical aortic stenosis, and severe comorbid disease. Most patients can be discharged on the same day within 2 to 6 hours after the procedure.

The most common reason for postprocedural hospitalization is hematoma formation necessitating additional bed rest and observation. Also, diagnosis from the procedure may require hospitalization, including the findings of severe left main or three-vessel disease. Other considerations for postprocedure hospitalization include uncompensated heart failure, unstable ischemic symptoms, severe aortic stenosis with LV dysfunction, renal insufficiency requiring further hydration, and need for continuous anticoagulation.⁷

PERSONNEL. Personnel in the catheterization laboratory include the medical director, physicians, nurses, cardiovascular fellows,

physician extenders including nurse practitioners and physician assistants, and radiologic technologists. All members should be trained in cardiopulmonary resuscitation and preferably in advanced cardiac life support. For full-service laboratories, it is highly desirable for facilities to be associated with a cardiothoracic surgical program. High-risk diagnostic studies and all elective percutaneous interventions should be performed in laboratories with on-site surgical facilities.⁵

LABORATORY CASELOAD. For proficiency to be maintained, laboratories for adult studies should perform a minimum of 300 procedures per year. According to the Accreditation Council for Graduate Medical Education guidelines for diagnostic catheterization, physicians in training must spend a total of 8 months and perform more than 300 cases, including more than 200 as a primary operator, to be credentialed for level II diagnostic cardiac catheterization procedures in practice.⁹ However, the minimum caseload for established physicians in practice has not been established.¹⁰ Regular evaluation with quality indicator assessment of laboratory, physician, nurse, and technologist performance and outcomes is mandatory. The laboratory director should possess at least 5 years of catheterization experience. In a laboratory performing percutaneous coronary intervention, the director should be board certified in interventional cardiology. The director is responsible for credentialing of physicians; for review of laboratory, physician, and ancillary personnel performance; and for provision of necessary training. Other responsibilities involve establishing and maintaining quality control of staff and equipment, patient outcome monitoring, and budget oversight.

EQUIPMENT. Necessary equipment for cardiac catheterization includes the radiographic system and physiologic data monitoring, including recording and acquisition instrumentation, sterile supplies, and an emergency cart. Also necessary is support equipment consisting of a power injector, image processing with digital archiving, viewing stations, and a uniform method of report generation.

Radiographic Equipment

High-resolution x-ray imaging is required for optimal performance of catheterization procedures. The necessary equipment includes a generator, x-ray tube, image intensifier or flat panel detector, expansive modulation, video image capture, imaged display, and either digital archiving or a cine camera (see Fig. 21-5).¹¹ Presently, x-ray systems use a flat panel detector rather than an image intensifier and therefore do not use video cameras. The flat panel detector produces a direct digital video signal from the original visible light fluorescence without the intermediate visible light stage.

Digital acquisition and archiving permits immediate on-line review, quantitative computer analysis, image manipulation capabilities, road maps, and flicker-free images at low frame rates, thus minimizing exposure. Transfer of images between cardiac catheterization laboratories, hospitals, and physician offices can be accomplished with use of a common network. The development of Digital Imaging and Communication (DICOM) standards for cardiac angiography has allowed compatibility among different vendor systems. Increased computer storage capabilities have allowed storage with immediate access to thousands of archived cases.

Physiologic Monitors

Continuous monitoring of blood pressure and the electrocardiogram (ECG) is required during cardiac catheterization. Systemic, pulmonary, and intracardiac pressures are generally recorded by use of fluid-filled catheters connected to strain-gauge pressure transducers and then transmitted to a monitor. Equipment for determination of thermodilution and Fick cardiac output and blood gas determination as well as a standard 12-lead ECG machine is necessary.

RADIATION SAFETY. Radiation effects can be classified as either deterministic effects or stochastic effects. Both have a delay between radiation and effect. The delay may be hours to years. Deterministic effects are dose related in that below a certain dose, there is no effect. However, when a threshold is exceeded, the severity increases with



dose. Examples of deterministic effects include skin erythema, desquamation, cataracts, hair loss, and skin necrosis. Skin injury is the most common deterministic effect from radiation. Early transient erythema can develop within hours, but most skin injuries do not appear for 2 to 3 weeks after exposure. The main guiding principle of x-ray exposure is ALARA (as low as reasonably achievable). This implies that no level of radiation is completely safe to patients or providers. The dose-area product (DAP) is the absorbed dose to air (air kerma) multiplied by the x-ray beam cross-sectional area at the point of measurement. It is an approximation of the total x-ray energy delivered to the patient and is a measure of the patient's risk of stochastic effect.¹¹

Another measure of skin dose is the interventional reference point (IRP). This is located 15 cm from the isocenter of the x-ray tube and is an estimation of the skin entrance point of the beam.

Stochastic effects are related to probability and not proportional to dose, although the likelihood of an effect is related to dose. Examples of this effect include neoplasms and genetic defects. The estimated dose range for cardiac catheterization is 1 to 10 millisievert (mSv), which is the equivalent of 2 to 3 years of natural background radiation.¹¹ The typical dose is 3 to 5 mSv.

The basic principles of minimizing radiation exposure include minimizing fluoroscopic beam time for fluoroscopy, using beam collimation, positioning the x-ray source and image reception optimally, using the least magnification possible, changing the radiographic projection in long procedures to minimize entrance port skin exposure, recording the estimated patient dose, and selecting equipment with dose reduction features including low fluoroscopy mode.

For laboratory personnel, the most important factors are maximizing distance from the source of x-rays and using appropriate shielding, including lead aprons, thyroid collars, lead eyeglasses, and movable leaded barriers. Severely angulated views, particularly the left anterior oblique view, substantially increase the radiation exposure of the operators.

A method for measuring radiation exposure for personnel is required. It is recommended that two film badges be worn, one on the outside of the apron at the neck and another under the apron at the waist. The latter monitors the effectiveness of the lead apron. The maximum allowable whole-body radiation dose per year for those working with radiation is 5 roentgen-equivalents-man (rem = 50 mSv) or a maximum of 50 rem in a lifetime.⁷

Catheterization Laboratory Protocol

PREPARATION OF THE PATIENT FOR CARDIAC CATHETERIZATION. Before arrival in the catheterization laboratory, the cardiologist responsible for the procedure should explain the procedure fully, including the risks and benefits, and answer questions from the patient and family. Precatheterization evaluation includes obtaining the patient's history, physical examination, and ECG. Routine laboratory studies include complete blood count with platelets, serum electrolyte determinations with creatinine and glucose concentrations, prothrombin time with international normalized ratio (INR), and partial thromboplastin time (in patients receiving heparin). Important components of the history that need to be addressed include diabetes mellitus (insulin or non-insulin requiring), kidney disease, anticoagulation status, and peripheral arterial disease as well as previous contrast media or latex allergy. Full knowledge of any prior procedures, including cardiac catheterizations, percutaneous coronary interventions, peripheral arterial interventions or surgery, and cardiac surgery, is necessary.

Patients should be fasting at least 6 hours, and an intravenous line should be established. Oral or intravenous sedation is usually administered (e.g., benzodiazepine). Pulse oximetry should be used to monitor respiratory status. Some laboratories premedicate patients with antihistamines such as diphenhydramine (25 mg intravenous push) for its anti-allergic properties and to assist in sedation. Oral anticoagulants should be discontinued and the INR should be less than 1.8 to avoid increased risk of bleeding. Aspirin or other oral antiplatelet agents are continued before the procedure. Patients with diabetes receiving metformin should have the medication discontinued the morning of the procedure and not restarted until renal function is stable at least 48 hours after the procedure.¹² All patients should receive hydration before and after the procedure. The amount of hydration is dependent on the ventricular function and baseline fluid status. However, if tolerated, a total of 1 liter of normal saline administered between initiation and completion of the procedure

is recommended. Another hydration regimen that has been shown to be effective in preventing contrast nephropathy in patients with chronic kidney disease is the use of sodium bicarbonate at 3 mL/kg/km for 1 hour before the procedure and 1 mL/kg/km for 6 hours after.¹³

Those with a prior history of contrast medium allergy need prophylaxis before the procedure.¹⁴ A recommended regimen is administration of either prednisone (60 mg by mouth) or hydrocortisone (100 mg by intravenous push) given 12 hours and immediately before the procedure. Cimetidine (300 mg), a nonselective histamine antagonist, and diphenhydramine (25 to 50 mg) may also be given by intravenous push. A common misconception is that a history of shellfish allergy predisposes the patient to contrast media reactions. The iodine in shellfish is not the allergen. Rather, tropomyosin appears to be the allergen.

CATHETERIZATION PROTOCOL. Each physician should develop a routine for performing diagnostic catheterization to ensure efficient acquisition of all pertinent data. The particular technical approach and necessary procedures should be established individually for each patient so that the specific clinical questions can be addressed. In general, hemodynamic measurements and cardiac output determination should be made before angiography to reflect basal conditions most accurately and to guide angiography. However, in a high-risk case, the approach is to gather the most important diagnostic information first because of the possibility of an adverse event.

Right-heart catheterization should not be performed in all patients undergoing routine coronary angiography. Despite limited risks, right-heart catheterization, including screening oximetric analysis, measurement of pressures, and determination of cardiac output, has a low yield in patients with suspected coronary artery disease without other known cardiac disease. Right-heart catheterization is indicated when a patient has LV dysfunction, congestive heart failure, complicated acute myocardial infarction, valvular heart disease, suspected pulmonary hypertension, congenital disease, intracardiac shunts, or pericardial disease.

Although the use of a temporary pacemaker is not indicated for routine cardiac catheterization, operators should understand the techniques for proper insertion and use when it is needed. Even in patients with isolated left bundle branch block, right-heart catheterization can generally be safely performed with balloon flotation catheters without causing additional conduction disturbance. An example of a balloon flotation catheter (Swan-Ganz) is shown in **Figure 20-1**.

CATHETERS AND ASSOCIATED EQUIPMENT. Catheters used for cardiac catheterization are available in various lengths, sizes, and configurations. Typical catheter lengths vary between 50 and 125 cm; 100 cm is the length most commonly used for adult left-heart catheterization by the femoral approach. The outer diameter of the catheter is specified by French units, where one French unit (F) = 0.33 mm. The inner diameter of the catheter is smaller than the outside diameter because of the thickness of the catheter material. Guidewires used during the procedure must be small enough to pass through the inner diameters of both the introducer needle and the catheter. Guidewires are described by their length in centimeters, diameter in inches, and tip conformation. A commonly used wire is a 150-cm, 0.035-inch J-tip wire. The introducer sheaths are specified by the French number of the largest catheter that passes

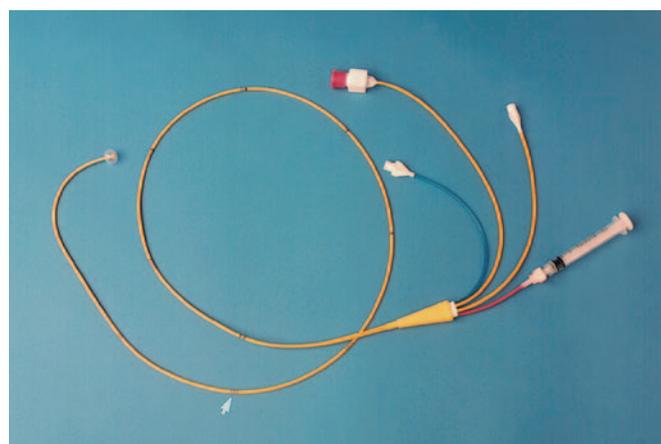


FIGURE 20-1 Typical Swan-Ganz catheter. Proximal ports, left to right, are proximal injection hub, thermistor connector, distal lumen hub, and balloon inflation valve with syringe. The distal end of the catheter has a balloon and a distal end hole. The proximal injectate port exits at 30 cm from the distal lumen (arrow). The thermistor lies just proximal to the balloon.

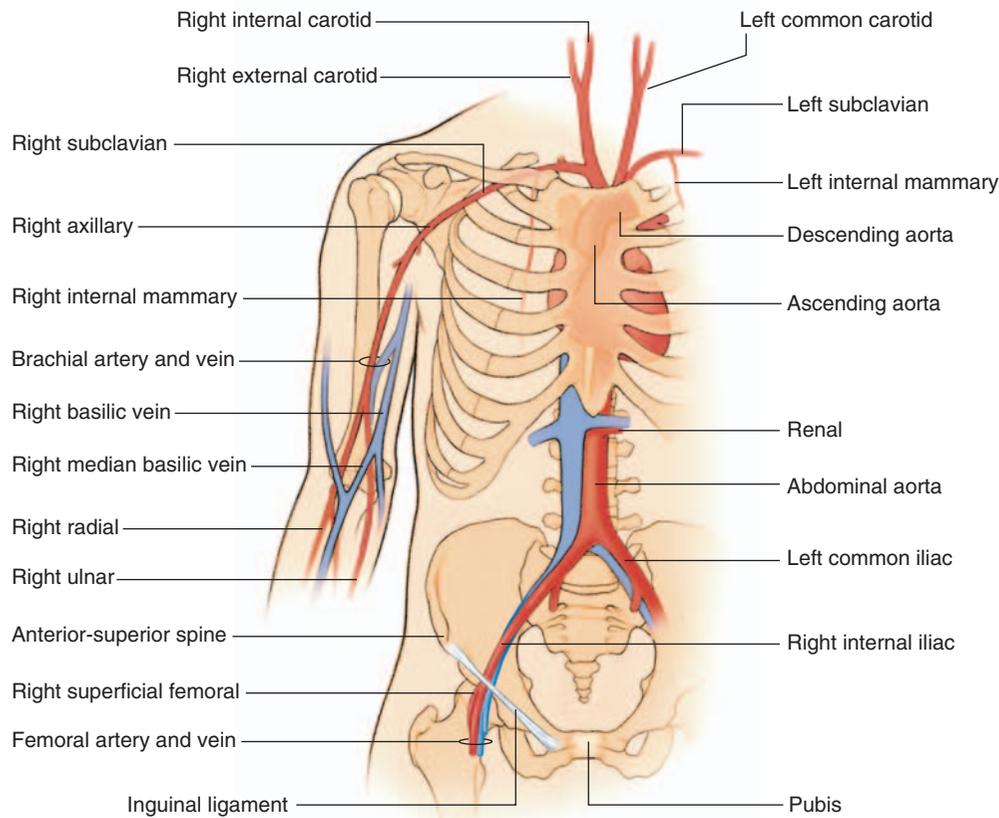


FIGURE 20-2 Principal arteries used for access during cardiac catheterization. Only the superficial veins are shown on the forearm. (Modified from Thibodeau GA, Patton KT [eds]: *Anthony's Textbook of Anatomy and Physiology*. 17th ed. St. Louis, CV Mosby, 2002.)

freely through the inner diameter of the sheath rather than its outer diameter. Therefore, a 7F introducer sheath accepts a 7F catheter (7F = 2.31 mm) but has an outer diameter of more than 2.31 mm.

The choice of the size of the catheters to be used is made by balancing the needs to opacify the coronary arteries and cardiac chambers adequately, to have adequate catheter manipulation, to limit vascular complications, and to permit early ambulation. Although the larger catheters allow greater catheter manipulation and excellent visualization, the most commonly used catheters (4F to 6F) permit earlier ambulation after catheterization and generally provide adequate visualization. Smaller catheters require greater technical skill of manipulation and have lower flow rates. Thus, their use in tortuous anatomy, large body habitus, or high coronary flow states (e.g., aortic regurgitation) may be limited. The relationship between sheath size and vascular complications is not clear. Rather, the arterial puncture technique, anticoagulation status including use of thienopyridines and glycoprotein IIb/IIIa receptor inhibitors, and coagulopathies are more important factors related to vascular complications.¹⁵

Right-Heart Catheterization

Right-heart catheterization allows measurement and analysis of right atrial, right ventricular (RV), pulmonary artery, and pulmonary capillary wedge pressures; determination of cardiac output; and screening for intracardiac shunts. Screening blood samples for oximetry should be obtained from the superior vena cava (SVC) and pulmonary artery in all patients. Right-heart catheterization is performed antegrade through either the inferior vena cava (IVC) or SVC. Percutaneous entry is achieved through the femoral, internal, jugular, subclavian, or antecubital vein. The anatomy of the major arteries and veins used for cardiac catheterization is shown in **Figure 20-2**.

When left-heart catheterization is performed by the Judkins technique (see later), the femoral vein is used most often for access to the

right side of the heart. However, when the right-heart catheter is left indwelling after the procedure, the internal jugular approach is preferable. This approach allows the patient to sit up in bed. The internal jugular approach is preferred to the subclavian to lessen the risk of pneumothorax. The use of a micropuncture kit with a 21-gauge needle and introducer can minimize potential trauma from inadvertent puncture of the carotid artery. When the jugular vein has been entered, the micropuncture assembly can be exchanged for a larger sheath (e.g., 7F) necessary for the right-heart catheterization. Also, the adjunctive use of portable vascular ultrasound probes can help locate and establish the patency of the jugular vein, particularly in patients with short, thick necks or after multiple previous catheterizations.

BALLOON FLOTATION CATHETERS. Balloon flotation catheters are the simplest and most widely used right-heart catheters. If thermodilution cardiac outputs must be determined, catheters that contain thermistors, such as Swan-Ganz catheters, are used (see Fig. 20-1). Intracardiac right-heart pressures and oxygen saturation to evaluate intracardiac shunts can also be obtained. They are both flexible and flow directed, but when the femoral approach is used,

fluoroscopic guidance is almost always necessary to cannulate the pulmonary artery and to obtain pulmonary capillary wedge position. Right-heart catheters have either a J-shaped or S-shaped curvature distally to facilitate passage from the SVC to the pulmonary artery or an S-shaped distal end for femoral insertion. Other right-heart balloon flotation end-hole catheters are more rigid and torqueable and allow passage of conventional 0.035- or 0.038-inch guidewires. Although these lack the ability to obtain thermodilution cardiac outputs, they yield better pressure fidelity because of less catheter whip artifact and a larger end hole.

There are two methods for advancing a balloon flotation catheter from the femoral vein. Often, the catheter can be advanced directly through the right atrium and across the tricuspid valve. Once it is in the right ventricle, the catheter is clockwise rotated to point superiorly and directly into the RV outflow tract. Once it is in the outflow tract, the balloon tip should allow flotation into the pulmonary artery and wedge positions (**Fig. 20-3**). When necessary, deep inspiration or cough can facilitate this maneuver and assist in crossing of the pulmonary valve. If the catheter continues to point inferiorly toward the RV apex, another technique should be used because further advancement can risk perforation of the RV apex.

Another technique for performing right-heart catheterization with a balloon flotation catheter is shown in Figure 20-3. A loop is formed in the right atrium, with the catheter tip directed laterally. The loop can be created by hooking the catheter tip on the hepatic vein or by advancing the catheter while it is directed laterally in the right atrium. Once the loop is formed, the catheter should be advanced farther, which directs the tip inferiorly and then medially across the tricuspid valve. Antegrade blood flow should then direct the catheter into the pulmonary artery. After the catheter is placed into the wedge position, the redundant loop can be removed with the balloon inflated by slow withdrawal.

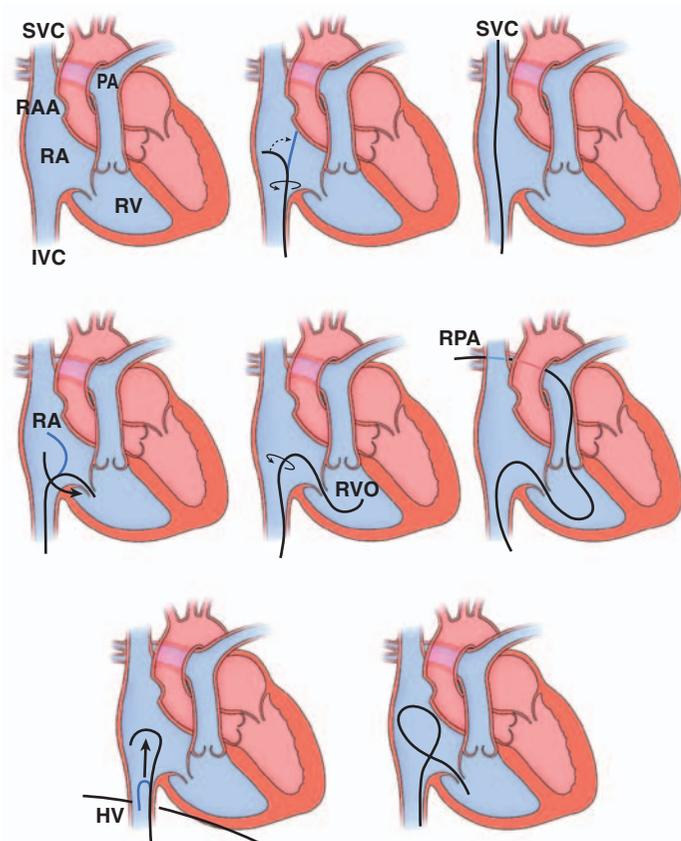


FIGURE 20-3 Right-heart catheterization from the femoral vein, shown in cartoon form. **Top row**, The right-heart catheter is initially placed in the right atrium (RA) aimed at the lateral atrial wall. Counterclockwise rotation aims the catheter posteriorly and allows advancement into the superior vena cava (SVC). Although it is not evident in the figure, clockwise catheter rotation into an anterior orientation would lead to advancement into the right atrial appendage (RAA), precluding SVC catheterization. **Center row**, The catheter is then withdrawn back into the right atrium and aimed laterally. Clockwise rotation causes the catheter tip to sweep anteromedially and to cross the tricuspid valve. With the catheter tip in a horizontal orientation just beyond the spine, it is positioned below the right ventricular outflow (RVO) tract. Additional clockwise rotation causes the catheter to point straight up, allowing advancement into the main pulmonary artery and from there into the right pulmonary artery (RPA). **Bottom row**, Two maneuvers useful in catheterization of a dilated right heart. A larger loop with a downward-directed tip may be required to reach the tricuspid valve and can be formed by catching the catheter tip in the hepatic vein (HV) and advancing the catheter quickly into the right atrium. The reverse loop technique (**bottom right**) gives the catheter tip an upward direction, aimed toward the outflow tract. IVC = inferior vena cava; PA = pulmonary artery; RV = right ventricle. (From Baim DS, Grossman W: *Percutaneous approach, including transseptal and apical puncture*. In Baim DS, Grossman W [eds]: *Cardiac Catheterization, Angiography, and Intervention*. 7th ed. Philadelphia, Lea & Febiger, 2006, p 86.)

NONFLOTATION CATHETERS. When an end-hole catheter (e.g., Courmand or multipurpose) that does not have a balloon tip is used, the technique for cannulation of the pulmonary artery is markedly different. Manipulation and torquing of the nonflotation catheter are necessary to advance it into the pulmonary artery. The catheter should be directed inferiorly across the tricuspid valve and then superiorly into the RV outflow tract. It is generally recommended that one attempt be made to form a loop in the right atrium before advancement into the right ventricle to lessen the risk of perforation.

PATENT FORAMEN OVALE CANNULATION. A probe-patent foramen ovale that allows access to the left atrium is present in 20% to 30% of adult patients. It can be entered by use of a multipurpose catheter with the tip directed medially and slightly posterior. This technique can be used in patients undergoing patent foramen ovale closures. The catheter is withdrawn slowly from the SVC or high right

atrium until a slight forward and medial motion is observed. The catheter then prolapses into the left atrium with gentle pressure against the interatrial septum in patients with a probe-patent foramen ovale. Left atrial position can be verified by the pressure waveform, by blood samples demonstrating arterial saturation, or by hand injection of contrast medium. If left atrial access is necessary and cannot be obtained with this technique, a transseptal catheterization should be undertaken (see Transseptal Catheterization).

Left-Heart Catheterization and Coronary Arteriography

THE JUDKINS TECHNIQUE. Because of its relative ease, speed, reliability, and low complication rate,^{1,10} the Judkins technique has become the most widely used method of left-heart catheterization and coronary arteriography. After local anesthesia with 1% lidocaine (Xylocaine), percutaneous entry of the femoral artery is achieved by puncturing the vessel 1 to 3 cm (or one to two fingerbreadths) below the inguinal ligament (**Fig. 20-4**). The ligament can be palpated as it courses from the anterior superior iliac spine to the superior pubic ramus. This ligament, not the inguinal crease, should be used as the landmark. The inguinal crease can be misleading, particularly in the obese patient. A hemostatic clamp can be used under fluoroscopy to verify that the nick is made over the inferior edge of the femoral head. A transverse skin incision is made over the femoral artery with a scalpel. With a modified Seldinger technique (**Fig. 20-5**), an 18-gauge thin-walled needle (**Fig. 20-6**) is inserted at a 30- to 45-degree angle into the femoral artery, and a 0.035- or 0.038-inch J-tip polytetrafluoroethylene (Teflon)-coated guidewire is advanced through the needle into the artery. The wire should pass freely up the aorta without tactile resistance and feel like a hot knife passing through butter.

After arterial access is obtained, a sheath at least equal in size to the coronary catheter is usually inserted into the femoral artery. The routine use of heparin for diagnostic cardiac catheterization has not been established. However, in prolonged procedures, such as in patients with bypass grafts or stenotic valve disease, it may be administered at 2000 to 3000 units by intravenous push. The routine administration of protamine after the procedure to reverse heparin is not recommended. Although rare, hypotensive reactions to protamine can be severe and are more common in patients with diabetes. In patients receiving heparin before arrival in the laboratory, an activated clotting time should be obtained after access. Sheath removal is usually not recommended until the activated clotting time is less than 170 seconds unless a vascular closure device is being used.

LV systolic and end-diastolic pressures can be obtained by advancing a pigtail catheter into the left ventricle (**Fig. 20-7**). In assessing valvular aortic stenosis, LV and aortic or femoral artery pressures should be recorded simultaneously with two transducers. The aortic catheter should be placed at least into the abdominal aorta rather than into the femoral artery. The attenuation of pressure can be severe in older adults with peripheral arterial disease, and the estimation of aortic pressure from the femoral artery pressure will be inaccurate for determination of valvular severity. Alternatively, pigtail catheters with both a distal and a proximal lumen can be used. These specially designed catheters measure supra- and subvalvular aortic and LV pressure simultaneously when two transducers are used. In suspected mitral stenosis, LV and wedge or left atrial pressures should be obtained simultaneously with two transducers.

Left ventriculography is performed in the 30-degree right anterior oblique and 45- to 50-degree left anterior oblique views. A pigtail catheter is most commonly used for this purpose. Power injection of 30 to 40 mL of contrast medium into the ventricle at 12 to 15 mL/sec is used to assess LV function and the severity of mitral regurgitation. After ventriculography, LV systolic and end-diastolic pressure measurements may be repeated and the systolic pressure recorded as the catheter is withdrawn from the left ventricle into the aorta. If an aortic transvalvular gradient is present, obtaining both of these pressures can detect it. For measurement of suspected intraventricular gradients, a

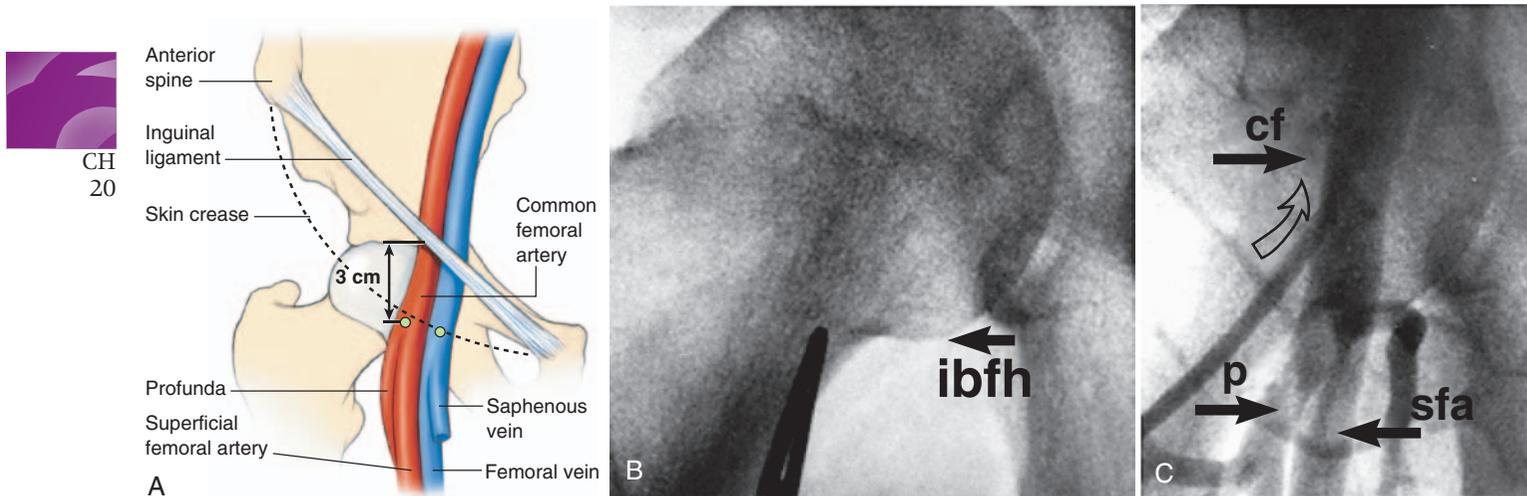


FIGURE 20-4 Regional anatomy relevant to percutaneous femoral arterial and venous catheterization. **A**, Schematic diagram showing the right femoral artery and vein coursing underneath the inguinal ligament, which runs from the anterior superior iliac spine to the pubic tubercle. The arterial skin nick should be placed approximately 3 cm below the ligament and directly over the femoral arterial pulsation; the venous skin nick should be placed at the same level but approximately one fingerbreadth more medial. Although this level corresponds roughly to the skin crease in most patients, anatomic localization relative to the inguinal ligament provides a more constant landmark. **B**, Fluoroscopic localization of skin nick (marked by clamp tip) to the inferior border of the femoral head (ibfh). **C**, Catheter (open arrow) inserted through this skin nick has entered the common femoral artery (cf), above its bifurcation into the superficial femoral artery (sfa) and profunda (p) branches. (From Baim DS, Grossman W: *Percutaneous approach, including transeptal and apical puncture*. In Baim DS, Grossman W [eds]: *Cardiac Catheterization, Angiography, and Intervention*. 7th ed. Philadelphia, Lea & Febiger, 2006, p 81.)

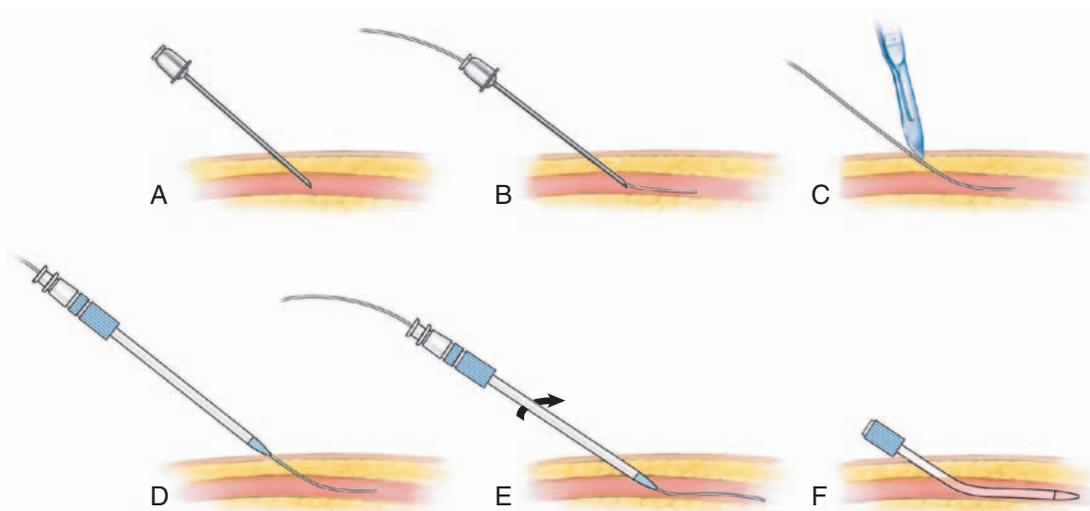


FIGURE 20-5 Modified Seldinger technique for percutaneous catheter sheath introduction. **A**, Vessel punctured by needle. **B**, Flexible guidewire placed into the vessel through the needle. **C**, The needle removed, the guidewire left in place, and the hole in the skin around the wire enlarged with a scalpel. **D**, Sheath and dilator placed over the guidewire. **E**, Sheath and dilator advanced over the guidewire and into the vessel. **F**, Dilator and guidewire removed while the sheath remains in the vessel. (From Hill JA, Lambert CR, Vlietstra RE, Pepine CJ: *Review of general catheterization techniques*. In Pepine CJ, Hill JA, Lambert CR [eds]: *Diagnostic and Therapeutic Cardiac Catheterization*. 3rd ed. Baltimore, Williams & Wilkins, 1998, p 107.)

multipurpose catheter with an end hole is desirable to localize the gradient in the left ventricle. Pigtail catheters contain side holes, which obscure the capacity to define whether the gradient is intraventricular, subvalvular, or transvalvular.

POSTPROCEDURE CARE. After coronary arteriography and left-heart catheterization have been completed, the catheters are removed and firm pressure is applied to the femoral area for 10 minutes by hand. The patient should be instructed to lie in bed for several hours, with the leg remaining straight to prevent hematoma formation. With 4F to 6F catheters, 2 hours of bed rest is usually sufficient, whereas use of catheters larger than 6F usually involves at least 3 to 4 hours.

Alternatively, vascular closure devices may be used. Four types are currently commercially available: collagen plugs, suture closure,

metallic clips, and hemostatic patches. Each allows early ambulation of the patients, within 1 to 2 hours after the procedure, and a shorter time to hemostasis than with manual compression.^{16,17} They also permit early sheath removal in patients receiving anticoagulation. Although one meta-analysis raised concern about the increased risk of pseudoaneurysm and hematoma with arterial puncture closure devices,¹⁸ another study demonstrated reduced vascular complications compared with manual compression.¹⁹ The ultimate success of any means of achieving hemostasis often relies on a single front-wall puncture of the common femoral artery.

The main advantage of the Judkins technique is speed and ease of selective catheterization. These attributes do not, however, preclude the importance of extensive operator experience to ensure quality studies with acceptable safety. The main disadvantage of this technique

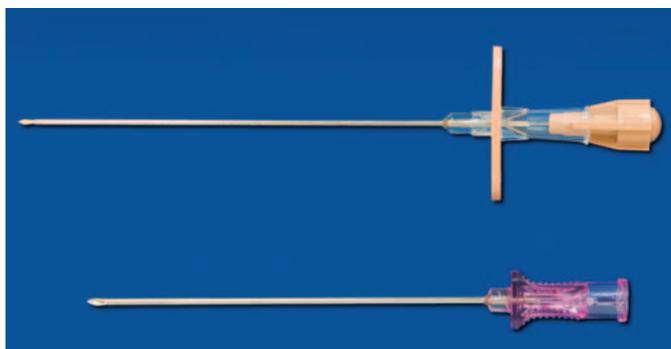


FIGURE 20-6 Two most commonly used needle types for vascular access. **Top**, A two-component, thin-walled Seldinger needle. **Bottom**, A single-piece, thin-walled “front-wall needle.”

is its use in patients with severe iliofemoral atherosclerotic disease, in whom retrograde passage of catheters through areas of extreme narrowing or tortuosity may be difficult or impossible. However, with careful technique, fluoroscopic guidance, and torquable floppy-tipped wires (e.g., Wholey wires), passage through synthetic aortofemoral grafts can be achieved with low complication rates.

BRACHIAL ARTERY TECHNIQUE—SONES TECHNIQUE. Sones and colleagues introduced the first technique for coronary artery catheterization by means of a brachial artery cutdown. The technically demanding Sones technique is still used in some centers and is described in [Chap. 21](#).

PERCUTANEOUS BRACHIAL ARTERY TECHNIQUE. A modification of the Sones technique is the percutaneous brachial artery technique using preformed Judkins catheters. This technique uses the Seldinger method of percutaneous brachial artery entry. A 4F to 6F sheath is placed into the brachial artery, and 3000 to 5000 units of heparin is infused into the side port. A guidewire is then advanced to the ascending aorta under fluoroscopic control. Judkins left, right, and pigtail catheters are passed over the guidewire for routine arteriography and ventriculography. The guidewire may occasionally be necessary to direct the left coronary catheter into the left sinus of Valsalva and the ostium of the left main coronary artery. Alternatively, an Amplatz left or multipurpose catheter is used to intubate the coronary ostium. After removal of the sheath, the arm should be maintained straight with an arm board for 4 to 6 hours with observation of radial and brachial pulses.

The main advantage of the percutaneous brachial technique is that it avoids a brachial artery cutdown and repair. The main disadvantage is that manipulation of catheters can be difficult. Compared with the femoral technique, patients' comfort, hemostasis time, and time to ambulation favor the brachial technique, whereas procedural efficiency, time of radiation exposure, and diagnostic image quality are favorable with the femoral approach. The complication rates appear similar.

PERCUTANEOUS RADIAL ARTERY TECHNIQUE. Left-heart catheterization by the radial artery approach was developed as an alternative to the percutaneous transbrachial approach in an attempt to limit vascular complications. The inherent advantages of the transradial approach are that the hand has a dual arterial supply connected through the palmar arches and that there are no nerves or veins at the site of puncture. In addition, bed rest is unnecessary after the procedure, allowing more efficient outpatient angiography.

The procedure requires a normal Allen test result. The Allen test consists of manual compression of both the radial and ulnar arteries during fist clenching until the hand is blanched. Normal color returns to the opened hand within 10 seconds after release of pressure over the ulnar artery, and significant reactive hyperemia is absent on release of pressure over the radial artery.

In the radial technique, the arm is abducted and the wrist hyperextended over a gauze roll. Routine skin anesthesia is used. A micropuncture needle or a 20-gauge Angiocath is introduced at a 30- to 45-degree angle into the radial artery 2 to 3 cm proximal to the flexor crease of the wrist. A 7- to 16-cm-long 4F or 5F sheath is then introduced over a short 0.025-inch wire. Next, about 10 mL of blood is drawn into a syringe containing heparin (3000 to 5000 units IAO) and vasodilators (e.g., 200 µg of nicardipine plus 100 µg of nitroglycerin) to prevent radial artery spasm. The cocktail is mixed with blood to minimize the burning sensation and is then injected into the side arm of the sheath. Coronary catheters are then advanced over a standard 0.035-inch J-tip exchange

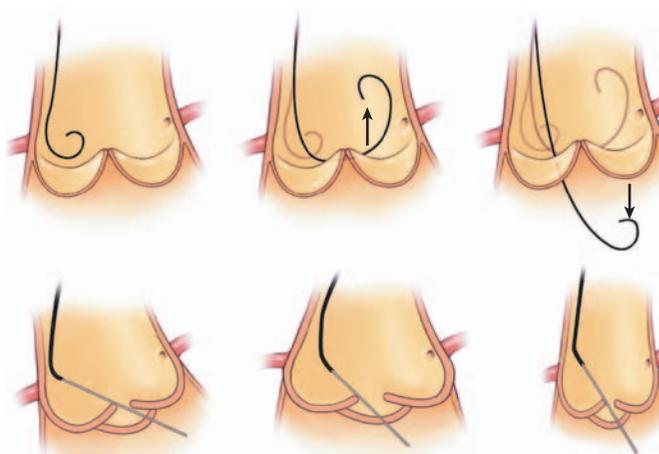


FIGURE 20-7 Technique for retrograde crossing of an aortic valve by a pigtail catheter. The **upper row** shows the technique for crossing of a normal aortic valve. In the **bottom row (left)**, the use of a straight guidewire and pigtail catheter in combination is shown. Increasing the length of protruding guidewire straightens the catheter curve and causes the wire to point more toward the right coronary ostium; reducing the length of protruding wire restores the pigtail contour and deflects the guidewire tip toward the left coronary artery. When the correct length of wire and the correct rotational orientation of the catheter have been found, repeated advancement and withdrawal of catheter and guidewire together allow retrograde passage across the valve. In a dilated aortic root (**bottom row, middle**), the angled pigtail catheter is preferable. In a small aortic root (**bottom row, right**), a right coronary Judkins catheter may have advantages. In patients with bicuspid valves, an Amplatz left catheter is often used as it directs the wire more superiorly. (From Baim DS, Grossman W: *Percutaneous approach including transseptal and apical puncture*. In Baim DS, Grossman W [eds]: *Cardiac Catheterization, Angiography, and Intervention*. 6th ed. Philadelphia, Lea & Febiger, 2006, p 93.)

wire into the ascending aorta. The left and right coronary arteries are intubated in a manner similar to the brachial approach. Hemostasis is obtained at the end of the procedure after sheath removal by use of direct pressure or an inflatable balloon cuff. It is recommended that the arterial puncture site be allowed to bleed for several beats before maintaining direct pressure. The radial pulse should be monitored regularly for several hours after the procedure.

The potential limitations of this procedure include the inability to cannulate the radial artery because of its smaller size and propensity to develop spasm, poor visualization of the coronary arteries resulting from the small-caliber catheters, limited manipulation potential, and risk of radial arterial occlusion caused by dissection or thrombus formation. If intervention is contemplated, device selection may be limited by guide catheter size. Although there is little debate that the femoral approach is the simplest technique for left-heart catheterization, the transradial approach for left-heart catheterization has gained in popularity.²⁰⁻²² A recent registry and meta-analysis have demonstrated improvement in bleeding rates compared with the femoral approach.²¹⁻²² However, large-scale randomized trials are lacking.

TRANSSEPTAL CATHETERIZATION. Transseptal left-heart catheterization has increased in popularity as a result of percutaneous balloon mitral commissurotomy as a preferential option to surgical commissurotomy (see [Chap. 66](#)), electrophysiologic procedures requiring access to pulmonary veins (see [Chap. 40](#)), and use of percutaneous mitral valve repair (see [Chap. 59](#)). Transseptal heart catheterization can be performed with a complication rate of less than 1% in experienced centers.²³⁻²⁵

An 8F Mullins or transseptal sheath and dilator combination is used. The Brockenbrough needle is 18-gauge that tapers to 21-gauge at the distal tip ([Fig. 20-8](#)). The needle is placed in the transseptal sheath. One commonly used approach is to place a 0.032-inch guidewire through the femoral vein, through the right atrium, and into the SVC. The Mullins or transseptal sheath and dilator are then advanced over the wire into the SVC. The guidewire is removed and replaced with a Brockenbrough needle. The distal port is connected to a pressure



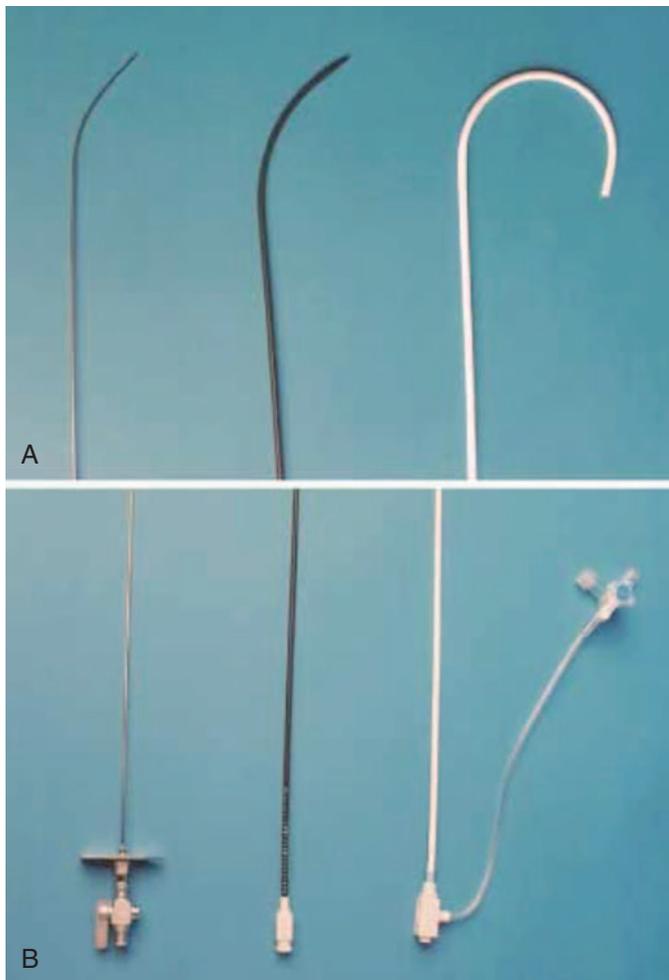


FIGURE 20-8 Transseptal catheters. **A**, Distal catheter. **B**, Proximal catheter. **Right**, Mullins transseptal sheath. **Middle**, Introducer (dilator) that is placed inside sheath to add stiffness to the catheter. **Left**, Brockenbrough transseptal needle that is placed inside the sheath and is used to penetrate the septum.

manifold. With the needle tip just proximal to the Mullins sheath tip, the entire catheter system is withdrawn. The catheter is simultaneously rotated from a 12 o'clock to a 5 o'clock position. The operator observes two abrupt rightward movements. The first occurs as the catheter descends from the SVC to the right atrium. The second occurs as the transseptal dilator tip passes over the limbic edge into the fossa ovalis. The dilator and needle are then advanced gently as a unit. Steady gentle pressure is sometimes adequate to advance the system through the fossa ovalis into the left atrium. If not, the needle should be advanced across the interatrial septum while the sheath is held in place. In cases in which transseptal puncture is technically difficult because of a large right atrium, postsurgical condition, or anatomic variant, intracardiac echocardiography can be useful to localize the fossa ovalis and interatrial septum.²⁶ (See Intracardiac Echocardiography.)

Left atrial position can be confirmed by the overall increase in pressure with left atrial *a* and *v* waveforms, hand injection of contrast medium, or measurement of arterial oxygen saturation. When the position is confirmed, the catheter should be rotated toward 3 o'clock and the dilator and sheath safely advanced 2 to 3 cm into the left atrium. The sheath is held firmly, and the dilator and needle are removed. Left atrial pressure measurements should then be repeated. If measurement of LV pressure or left ventriculography is necessary, the catheter can usually be advanced easily into the left ventricle after slight counterclockwise rotation. The major risk of transseptal catheterization lies in inadvertent puncture of atrial structures, such as the

atrial free wall, left atrial appendage, and coronary sinus, or entry into the aortic root or pulmonary artery.

DIRECT TRANSTHORACIC LEFT VENTRICULAR PUNCTURE.

The sole diagnostic indication for direct LV puncture is to measure LV pressure and to perform ventriculography in patients with mechanical prosthetic valves in both the mitral and aortic positions, preventing retrograde arterial and transseptal catheterization. Crossing of tilting disc valves with catheters should be avoided because catheter entrapment, occlusion of the valve, or possible dislodgment of the disc with embolization may result.

The procedure is performed after localization of the LV apex by palpation or, preferably, by echocardiography.^{27,28} After local anesthesia is administered, an 18- or 21-gauge 6-inch Teflon catheter system is inserted at the upper rib margin and directed slightly posteriorly and toward the right second intercostal space until the impulse is encountered. The needle and sheath are advanced into the left ventricle. The stylet and needle are removed, and the sheath is connected for pressure measurement.

The risks of this procedure include cardiac tamponade, hemothorax, pneumothorax, laceration of the left anterior descending coronary artery, embolism of LV thrombus, vagal reactions, and ventricular arrhythmias. The risk of pericardial tamponade, however, is limited in patients who have undergone prior cardiac surgery because mediastinal fibrosis is present. With current noninvasive imaging techniques including transesophageal echocardiography, this procedure is rarely indicated. Transapical aortic valve implantation uses this technique when femoral access is limited by vessel size (see Chap. 59). Direct visualization of the LV apex is accomplished through intercostal incision followed by apical puncture with the Seldinger technique.

ENDOMYOCARDIAL BIOPSY.

Endomyocardial biopsy is performed most commonly with various disposable or, less frequently, reusable bioptomes. The most popular devices used for the internal jugular vein approach include preshaped 50-cm bioptomes. RV biopsy may be performed with use of the internal jugular vein (see Right-Heart Catheterization for internal jugular technique), the subclavian vein, or the femoral vein. LV biopsy is performed with the femoral arterial approach.

When RV biopsy is performed through the right internal jugular vein, a 7F short straight sheath or long curved sheath is introduced by the usual Seldinger technique. If a short sheath is used, a 7F bioptome is advanced under fluoroscopic guidance to the lateral wall of the right atrium. With counterclockwise rotation, the device is advanced across the tricuspid valve and toward the interventricular septum. When a long preshaped sheath is used, it is positioned against the RV septum. RV pressure should be continuously monitored. The bioptome is passed through the sheath, and samples are obtained. Alternatively, two-dimensional echocardiography rather than fluoroscopy has been used to guide the position of the bioptome.

Contact with the myocardium is confirmed by the presence of premature ventricular contractions, resistance to further advancement, and transmission of the ventricular impulse to the operator. The bioptome is then slightly withdrawn from the septum, the forceps jaws are opened, the bioptome is readvanced to make contact with the myocardium, and the forceps is closed. A slight tug is felt on removal of the device. Four to six samples of myocardium are usually required for adequate pathologic analysis. Preprocedure consultation with a pathologist or transplant cardiologist should be obtained to ensure appropriate specimen collection and processing.

RV biopsy from the femoral vein requires insertion of a long 7F sheath directed toward the portion of the ventricle to be sampled. Various configurations of sheaths are used for RV biopsy. The conventional sheath has a 45-degree angle on its distal end to allow access to the right ventricle. However, specifically designed sheaths have dual curves. These catheters possess the usual 180-degree curve and an additional distal perpendicular septal plane curve of 90 degrees, which allows improved manipulation and positioning toward the interventricular septum. This sheath configuration can also be used from the internal jugular approach.



Whatever access is used, the biptome is advanced through the sheath and should be visualized in both the 30-degree right anterior oblique and 40-degree left anterior oblique views. The right anterior oblique view ensures that the catheter is in the midventricle away from the apex. The left anterior oblique view verifies that the sheath tip is oriented toward the interventricular septum. Contrast medium infusion through the side port of the sheath can be confirmatory. Samples of myocardium are taken in a manner similar to that described earlier.

If LV biopsy is to be performed, the biopsy sheath is generally inserted through the femoral artery and positioned over a multipurpose or pigtail catheter that has been placed in the ventricle. The sheath is advanced below the mitral apparatus and away from the posterobasal wall. The catheter is then withdrawn, and a long LV biptome is inserted. Care must be taken when LV biopsy is performed to prevent air embolism while the biptome is introduced into the sheath. A constant infusion of flush solution through the sheath minimizes the risk of air or thrombus embolism.

Complications of endomyocardial biopsy include cardiac perforation with cardiac tamponade, emboli (air, tissue, or thromboembolus), arrhythmias, electrical conduction disturbances, injury to the tricuspid valve, vasovagal reactions, and pneumothorax. The overall complication rate is between 1% and 3%; the risk of cardiac perforation with tamponade is generally reported as less than 0.05%.^{29,30} Endomyocardial biopsy is the most common cause of severe tricuspid regurgitation after cardiac transplantation.³¹ The use of longer sheaths dramatically decreases the incidence of anatomic disruption of the valve during biopsy.

Systemic embolization and ventricular arrhythmias are more common with LV biopsy. LV biopsy should generally be avoided in patients with right bundle branch block because of the potential for development of complete atrioventricular block as well as in patients with known LV thrombus.

The role of endomyocardial biopsy in the management of cardiovascular disease has been recently defined.³⁰ There are two Class I indications for endomyocardial biopsy for clinical scenarios (**Table 20-2**). The first is new-onset heart failure of less than 2 weeks' duration

associated with either normal or enlarged LV size and hemodynamic compromise. The second is new-onset heart failure of up to 3 months' duration complicated by LV dilation, new ventricular arrhythmias, advanced heart block, or failure to respond to usual care within 2 weeks. The use of biopsy for suspected anthracycline toxicity or restrictive disease is considered a Class IIa indication.³⁰ Cardiac transplant monitoring for rejection is the most common indication for biopsy (see **Chap. 31**).

PERCUTANEOUS INTRA-AORTIC BALLOON PUMP INSERTION. Intra-aortic balloon counterpulsation devices are positioned in the descending thoracic aorta. They have a balloon volume of 30 to 50 mL, use helium as the inflation gas, and are timed to inflate during diastole and to deflate during systole. Balloon size is based on the patient's height. The device is inserted through the femoral artery by the standard Seldinger technique so that the tip is 2 to 3 cm below the level of the left subclavian artery. 7F to 8F systems are used. Optimal positioning requires fluoroscopic guidance. Timing of the balloon is adjusted during 1:2 (one inflation for each two beats) pumping so that inflation of the balloon occurs at the aortic dirotic notch and deflation occurs immediately before systole. This timing ensures maximal augmentation of diastolic flow and maximal systolic unloading. **Figure 20-9** displays the optimal timing of an intra-aortic balloon pump (IABP).³²

Favorable hemodynamic effects include reduction in LV afterload and improvement in myocardial oxygenation.³³ IABP insertion is indicated for patients with angina refractory to medical therapy, cardiogenic shock, or mechanical complications of myocardial infarction (including severe mitral regurgitation and ventricular septal defect) or for those who have severe left main coronary artery stenosis. IABP may also be valuable in patients undergoing high-risk percutaneous coronary intervention or after primary angioplasty in the setting of acute myocardial infarction.³⁴ IABP insertion is contraindicated in patients with moderate or severe aortic regurgitation, aortic dissection, aortic aneurysm, patent ductus arteriosus, severe peripheral vascular disease, bleeding disorders, or sepsis.

Complications of IABP insertion include limb ischemia requiring early balloon removal or vascular surgery, balloon rupture, balloon entrapment, hematomas, and sepsis.^{32,34} The incidence of vascular complications ranges from 12% to greater than 40%. Most patients in whom limb ischemia develops after insertion of a balloon pump device have resolution of the ischemia on balloon removal and do not require surgical

TABLE 20-2 The Role of Endomyocardial Biopsy in 14 Clinical Scenarios

CLINICAL SCENARIO	CLASS OF RECOMMENDATION (I, IIa, IIb, III)	LEVEL OF EVIDENCE (A, B, C)
New-onset heart failure of <2 weeks' duration associated with a normal-sized or dilated left ventricle and hemodynamic compromise	I	B
New-onset heart failure of 2 weeks' to 3 months' duration associated with a dilated left ventricle and new ventricular arrhythmias, second- or third-degree heart block, or failure to respond to usual care within 2 weeks	I	B
Heart failure >3 months' duration associated with a dilated left ventricle and new ventricular arrhythmias, second- or third-degree heart block, or failure to respond to usual care within 2 weeks	IIa	C
Heart failure associated with a dilated cardiomyopathy of any duration associated with suspected allergic reaction and/or eosinophilia	IIa	C
Heart failure associated with suspected anthracycline cardiomyopathy	IIa	C
Heart failure associated with unexplained restrictive cardiomyopathy	IIa	C
Suspected cardiac tumors	IIa	C
Unexplained cardiomyopathy in children	IIa	C
New-onset heart failure of 2 weeks' to 3 months' duration associated with a dilated left ventricle, without new ventricular arrhythmias or second- or third-degree heart block, that responds to usual care within 2 weeks	IIb	B
Heart failure >3 months' duration associated with a dilated left ventricle, without new ventricular arrhythmias or second- or third-degree heart block, that responds to usual care within 2 weeks	IIb	C
Heart failure associated with unexplained hypertrophic cardiomyopathy	IIb	C
Suspected arrhythmogenic RV dysplasia	IIb	C
Unexplained ventricular arrhythmias	IIb	C
Unexplained atrial fibrillation	III	C

From Cooper LT, Baughman K, Feldman AM, et al: The role of endomyocardial biopsy in the management of cardiovascular disease. *J Am Coll Cardiol* 50:1914, 2007.

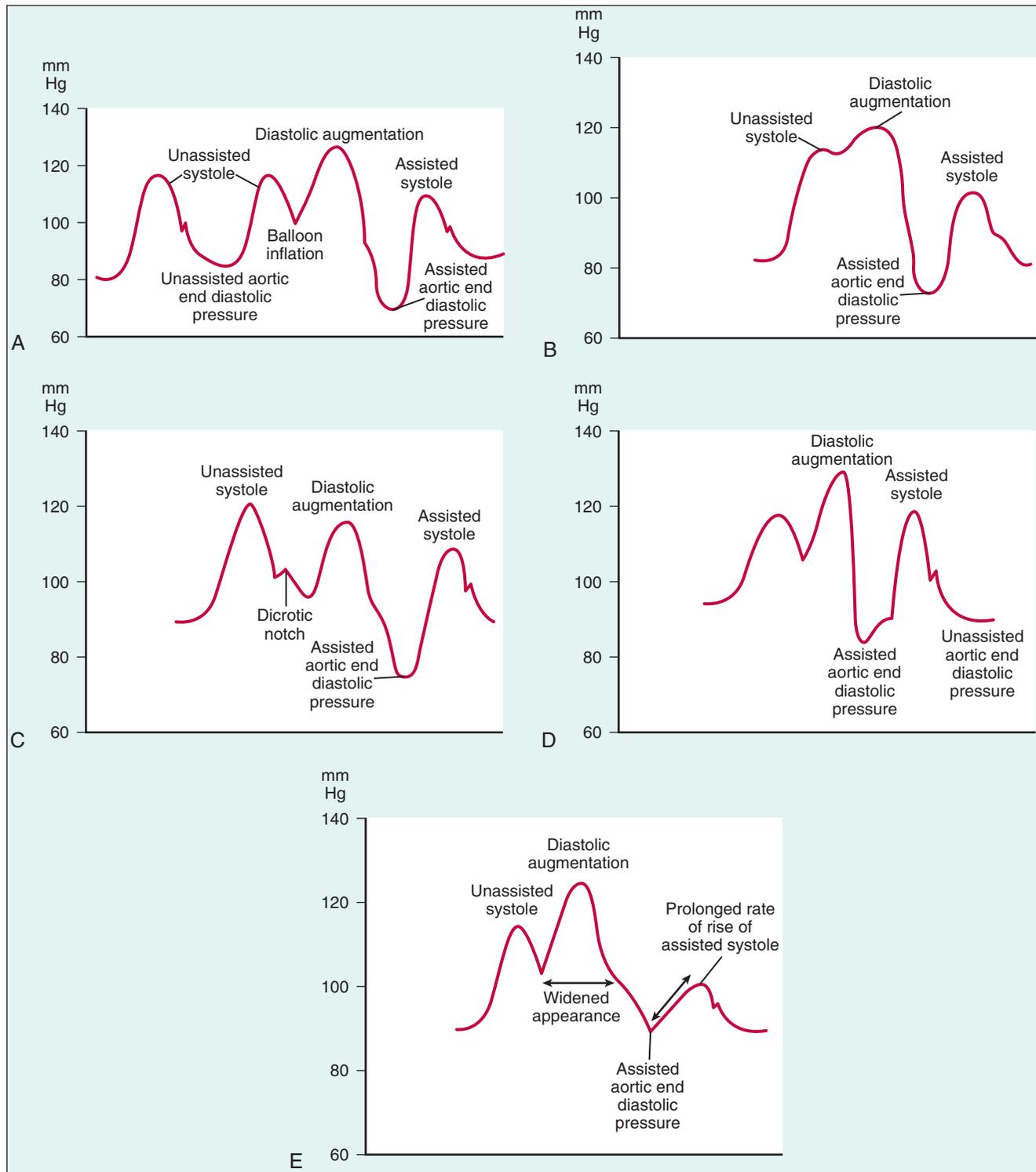


FIGURE 20-9 **A**, Optimal timing and arterial waveforms with an IABP. Systemic arterial pressure waveform from a patient with a normally functioning IABP device in whom the IABP device is programmed to inflate during every other cardiac cycle (commonly referred to as 1:2 inflation). With the first beat, aortic systolic and end-diastolic pressures are shown without IABP support and are therefore unassisted. With the second beat, the balloon inflates with the appearance of the dicrotic notch, and peak-augmented diastolic pressure is inscribed. With balloon deflation, assisted end-diastolic pressure and assisted systolic pressure are observed. To confirm that IABP is producing maximal hemodynamic benefit, the peak diastolic augmentation should be greater than the unassisted systolic pressure, and the two assisted pressures should be less than the unassisted values. **B**, Systemic arterial pressure waveform from a subject in whom balloon inflation occurs too early, before aortic valve closure. Consequently, the left ventricle is forced to empty against an inflated balloon; the corresponding increase in afterload may increase myocardial oxygen demands and worsen systolic function. **C**, Systemic arterial pressure waveform from a patient in whom balloon inflation occurs too late, well after the beginning of diastole, thereby minimizing diastolic pressure augmentation. **D**, Systemic arterial pressure waveform from a patient in whom balloon deflation occurs too early, before the end of diastole. This may shorten the period of diastolic pressure augmentation. A corresponding transient decrease in aortic pressure may promote retrograde arterial flow from the carotid or coronary arteries, possibly inducing cerebral or myocardial ischemia. **E**, Systemic arterial pressure waveform from a subject in whom balloon deflation occurs too late, after the end of diastole, thereby producing the same deleterious consequences as early balloon inflation (increased left ventricular afterload, with a resultant increase in myocardial oxygen demands and a worsening of systolic function). (From Trost JC, Hillis LD: *Intra-aortic balloon counterpulsation*. *Am J Cardiol* 97:1391, 2006.)



intervention (thrombectomy, vascular repair, fasciotomy, or amputation). The risk of limb ischemia is heightened in patients with diabetes or peripheral arterial disease, in women, and in patients with a postinsertion ankle-brachial index of less than 0.8. However, with the use of smaller catheters (7F), vascular complications are reduced.

Hemodynamic Data

The hemodynamic component of the cardiac catheterization procedure focuses on pressure measurements, the measurement of flow (e.g., cardiac output, shunt flows, flow across a stenotic orifice, regurgitant flows, and coronary blood flow), and the determination of vascular resistances. Simply stated, flow through a blood vessel is determined by the pressure difference within the vessel and the vascular resistance as described by Ohm's law: $Q = \Delta P/R$.

Pressure Measurements

Accurate recording of pressure waveforms and correct interpretation of physiologic data derived from these waveforms are major goals of cardiac catheterization. A pressure wave is the cyclical force generated by cardiac muscle contraction, and its amplitude and duration are influenced by various mechanical and physiologic parameters. The pressure waveform from a particular cardiac chamber is influenced by the force of the contracting chamber and its surrounding structures, including the contiguous chambers of the heart, pericardium, lungs, and vasculature. Physiologic variables of heart rate and the respiratory cycle also influence the pressure waveform. An understanding of the components of the cardiac cycle is essential to the correct interpretation of hemodynamic data obtained in the catheterization laboratory.

PRESSURE MEASUREMENT SYSTEMS

Fluid-Filled Systems

Intravascular pressures are typically measured with use of a fluid-filled catheter that is attached to a pressure transducer. The pressure wave is transmitted from the catheter tip to the transducer by the fluid column within the catheter. The majority of pressure transducers used currently are disposable electrical strain gauges. The pressure wave distorts the diaphragm or wire within the transducer. This energy is then converted to an electrical signal proportional to the pressure being applied using the principle of the Wheatstone bridge. This signal is then amplified and recorded as an analog signal.³⁵

There are a number of sources of error when pressures are measured with a fluid-filled catheter-transducer system. Distortion of the output signal occurs as a result of the frequency response characteristics and damping characteristics of the system. The frequency response of the system is the ratio of the output amplitude to input amplitude over a range of frequencies of the input pressure wave. The natural frequency is the frequency at which the system oscillates when it is shock-excited in the absence of friction. Dissipation of the energy of the system, such as by friction, is called damping. To ensure a high-frequency response range, the pressure measurement system should have the highest possible natural frequency and optimal damping. With optimal damping, the energy is dissipated gradually, thus maintaining the frequency response curve as close as possible to an output/input ratio of 1 as it approaches the system's natural frequency. Optimal damping is achieved by use of a short, wide-bore, noncompliant catheter-tubing system that is directly connected to the transducer with use of a low-density liquid from which all air bubbles have been removed.³⁵

The pressure transducer must be calibrated against a known pressure, and the establishment of a zero reference must be undertaken at the start of the catheterization procedure. To "zero" the transducer, the transducer is placed at the level of the atria, which is approximately midchest. If the transducer is attached to the manifold and variable positions during the procedure, a second fluid-filled catheter system should be attached to the transducer and positioned at the level of the midchest. All transducers being used during the procedure

should be zeroed and calibrated simultaneously. Because of possible variable drift during the procedure, all transducers should be rebalanced immediately before simultaneous recordings for transvalvular gradient or simultaneous pressure determinations are obtained.

Potential sources of error include catheter whip artifact (motion of the tip of the catheter within the measured chamber), end-pressure artifact (an end-hole catheter measures an artificially elevated pressure because of streaming or high velocity of the pressure wave), catheter impact artifact (when the catheter is struck by the walls or valves of the cardiac chambers), and catheter tip obstruction within small vessels or valvular orifices occurring because of the size of the catheter itself. The operator must be aware of the many sources of potential error, and when there is a discrepancy between the observed data and the clinical scenario, all components of the system should be examined for errors or artifacts.

Micromanometer Catheters

The use of these catheters, which have the pressure transducer mounted at the tip, greatly reduces many of the errors inherent to fluid-filled systems. However, their utility is limited by the additional cost and time needed for proper calibration and use of the system. These catheters have higher natural frequencies and more optimal damping characteristics because the interposing fluid column is eliminated. In addition, there is a decrease in catheter whip artifact. The pressure waveform is less distorted and is without the 30- to 40-millisecond delay seen in the fluid-filled catheter-transducer system. Commercially available high-fidelity micromanometer systems have both an end hole and side holes to allow over-the-wire insertion into the circulation while also permitting angiography. Catheters that have two transducers separated by a short distance are useful for accurate measurement of gradients across valvular structures and within ventricular chambers. The micromanometer system has been used for research purposes to assess rate of ventricular pressure rise (dP/dt), wall stress, rate of ventricular pressure decay ($-dP/dt$), time constant of relaxation (τ), and ventricular pressure-volume relationships (see Chap. 24).

NORMAL PRESSURE WAVEFORMS. An understanding of the normal pressure waveform morphologies is necessary to comprehend the abnormalities that characterize certain pathologic conditions. Normal pressures in the cardiac chambers and great vessels are listed in **Table 20-3**. Simply stated, whenever fluid is added to a chamber or compressed within a chamber, the pressure usually rises; conversely, whenever fluid exits from a chamber or the chamber relaxes, the pressure usually falls. One exception to this rule is the early phase of ventricular diastolic filling, when ventricular volume increases after mitral valve opening but ventricular pressure continues to decrease because of active relaxation. Examples of normal-pressure waveforms are shown in **Figure 20-10**.

Atrial Pressure

The right atrial pressure waveform has three positive deflections, the *a*, *c*, and *v* waves. The *a* wave is due to atrial systole and follows the P wave of the ECG. The height of the *a* wave depends on atrial contractility and the resistance to RV filling. The *x* descent follows the *a* wave and represents relaxation of the atrium and downward pulling of the tricuspid annulus by RV contraction. The *x* descent is interrupted by the *c* wave, which is a small positive deflection caused by protrusion of the closed tricuspid valve into the right atrium. Pressure in the atrium rises after the *x* descent as a result of passive atrial filling. The atrial pressure then peaks as the *v* wave, which represents RV systole. The height of the *v* wave is related to atrial compliance and the amount of blood returning to the atrium from the periphery. The right atrial *v* wave is generally smaller than the *a* wave. The *y* descent occurs after the *v* wave and reflects tricuspid valve opening and right atrial emptying into the right ventricle. During spontaneous respiration, right atrial pressure declines during inhalation as intrathoracic pressure falls. Right atrial pressure rises during exhalation as intrathoracic pressures increase. The opposite effect is seen when patients are mechanically ventilated.

The left atrial pressure waveform is similar to that of the right atrium, although normal left atrial pressure is higher, reflecting the high-pressure system of the left side of the heart. In the left atrium, as opposed to the right atrium, the *v* wave is generally higher than the *a* wave. This difference occurs because the left atrium is constrained posteriorly by the pulmonary veins, whereas the right atrium can easily decompress throughout the IVC and SVC. The height of the left atrial *v* wave most accurately reflects left atrial compliance.

Pulmonary Capillary Wedge Pressure

The pulmonary capillary wedge pressure waveform is similar to the left atrial pressure waveform but is slightly damped and delayed as a result of transmission through the lungs. The *a* and *v* waves with both

x and *y* descents are visible, but *c* waves may not be seen. In the normal state, the pulmonary artery diastolic pressure is similar to the mean pulmonary capillary wedge pressure because the pulmonary circulation has low resistance. In certain disease states that are associated with elevated pulmonary vascular resistance (hypoxemia, pulmonary embolism, and chronic pulmonary hypertension) and occasionally after mitral valve surgery, the pulmonary capillary wedge pressure may overestimate true left atrial pressure. In this circumstance, accurate measurement of the mitral valve gradient may require the direct left atrial pressure to be obtained.³⁶

Ventricular Pressure

RV and LV waveforms are similar in morphology. They differ mainly with respect to their magnitudes. The durations of systole and isovolumic contraction and relaxation are longer and the ejection period is shorter in the left than in the right ventricle. There may be a small (5 mm Hg) systolic gradient between the right ventricle and the pulmonary artery. Ventricular diastolic pressure is characterized by an early rapid filling wave, during which most of the ventricle fills; a slow filling phase; and the *a* wave, denoting atrial systolic activity. End-diastolic pressure is generally measured at the C point, which is the rise in ventricular pressure at the onset of isovolumic contraction. When the C point is not well seen, a line is drawn from the R wave on the simultaneous ECG to the ventricular pressure waveform, and this is used as the end-diastolic pressure.

Great Vessel Pressures

The contour of the central aortic pressure and the pulmonary artery pressure tracing consists of a systolic wave, the incisura (indicating closure of the semilunar valves), and a gradual decline in pressure until the following systole. The pulse pressure reflects the stroke volume and compliance of the arterial system. The mean aortic pressure more accurately reflects peripheral resistance. As the systemic pressure wave is transmitted through the length of the aorta, the systolic wave increases in amplitude and becomes more triangular, and the diastolic wave decreases until it reaches the midthoracic aorta and then increases. The mean aortic pressures, however, are usually similar; the mean peripheral arterial pressure is typically ≤ 5 mm Hg lower than the mean central aortic pressure.

The difference in systolic pressures between the central aorta and the periphery (femoral, brachial, or radial arteries) is greatest in younger patients because of their increased vascular compliance. These potential differences between proximal aorta and peripheral artery must be considered to measure and to interpret the peak systolic pressure gradient between the left ventricle and the systemic arterial system in patients with suspected aortic stenosis. When a transvalvular gradient is present, the most accurate measure of the aortic pressure is obtained at the level of the coronary arteries. This measurement avoids the effect of pressure recovery, which is defined

TABLE 20-3 Normal Pressures and Vascular Resistances		
PRESSURES	AVERAGE (mm Hg)	RANGE (mm Hg)
Right atrium		
<i>a</i> wave	6	2-7
<i>v</i> wave	5	2-7
Mean	3	1-5
Right ventricle		
Peak systolic	25	15-30
End-diastolic	4	1-7
Pulmonary artery		
Peak systolic	25	15-30
End-diastolic	9	4-12
Mean	15	9-19
Pulmonary capillary wedge		
Mean	9	4-12
Left atrium		
<i>a</i> wave	10	4-16
<i>v</i> wave	12	6-21
Mean	8	2-12
Left ventricle		
Peak systolic	130	90-140
End-diastolic	8	5-12
Central aorta		
Peak systolic	130	90-140
End-diastolic	70	60-90
Mean	85	70-105
VASCULAR RESISTANCES	MEAN (DYNE-SEC · CM ⁻⁵)	RANGE (DYNE-SEC · CM ⁻⁵)
Systemic vascular resistance	1100	700-1600
Total pulmonary resistance	200	100-300
Pulmonary vascular resistance	70	20-130

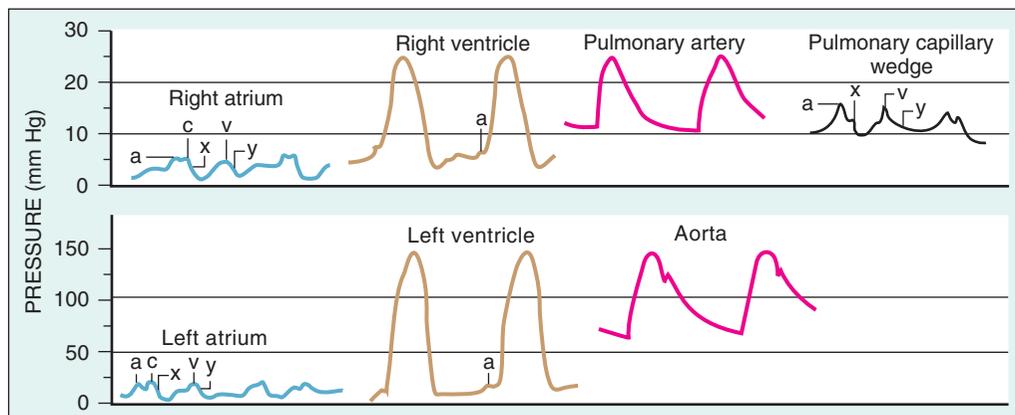


FIGURE 20-10 Normal right- and left-heart pressures recorded from fluid-filled catheter systems in a human. (From Pepine C, Hill JA, Lambert CR [eds]: *Diagnostic and Therapeutic Cardiac Catheterization*. 3rd ed. Baltimore, Williams & Wilkins, 1998.)

as the variable increase in lateral pressure downstream from a stenotic orifice (see Chaps. 15 and 66). This approach can become clinically important in cases of mild to moderate aortic stenosis, particularly when the aorta is small.³⁷ There will be an underestimation of the transvalve gradient and overestimation of aortic valve area because of higher pressure in the femoral artery in younger patients when supra-ventricular pressure is not obtained. This can be avoided with a

dual-lumen pigtail catheter, which measures pressure in the left ventricle and ascending aorta simultaneously.

ABNORMAL PRESSURE CHARACTERISTICS. Abnormal pressure waveforms may be diagnostic of specific pathologic conditions. **Table 20-4** summarizes the more commonly encountered waveforms.

TABLE 20-4 Pathologic Waveforms

<p>I. Right atrial pressure waveforms</p> <p>A. Low mean atrial pressure</p> <ol style="list-style-type: none"> 1. Hypovolemia 2. Improper zeroing of the transducer <p>B. Elevated mean atrial pressure</p> <ol style="list-style-type: none"> 1. Intravascular volume overload states 2. Right ventricular failure due to valvular disease (tricuspid or pulmonic stenosis or regurgitation) 3. Right ventricular failure due to myocardial disease (right ventricular ischemia, cardiomyopathy) 4. Right ventricular failure due to left-sided heart failure (mitral stenosis or regurgitation, aortic stenosis or regurgitation, cardiomyopathy, ischemia) 5. Right ventricular failure due to increased pulmonary vascular resistance (pulmonary embolism, chronic obstructive pulmonary disease, primary pulmonary hypertension) 6. Pericardial effusion with tamponade physiology 7. Obstructive atrial myxoma <p>C. Elevated <i>a</i> wave (any increase to ventricular filling)</p> <ol style="list-style-type: none"> 1. Tricuspid stenosis 2. Decreased ventricular compliance due to ventricular failure, pulmonic valve stenosis, or pulmonary hypertension <p>D. Cannon <i>a</i> wave</p> <ol style="list-style-type: none"> 1. Atrial-ventricular asynchrony (atria contract against a closed tricuspid valve, as during complete heart block, following premature ventricular contraction, during ventricular tachycardia, with ventricular pacemaker) <p>E. Absent <i>a</i> wave</p> <ol style="list-style-type: none"> 1. Atrial fibrillation or atrial standstill 2. Atrial flutter <p>F. Elevated <i>v</i> wave</p> <ol style="list-style-type: none"> 1. Tricuspid regurgitation 2. Right ventricular heart failure 3. Reduced atrial compliance (restrictive myopathy) <p>G. <i>a</i> wave equal to <i>v</i> wave</p> <ol style="list-style-type: none"> 1. Tamponade 2. Constrictive pericardial disease 3. Hypervolemia <p>H. Prominent <i>x</i> descent</p> <ol style="list-style-type: none"> 1. Tamponade 2. Subacute constriction and possibly chronic constriction 3. Right ventricular ischemia with preservation of atrial contractility <p>I. Prominent <i>y</i> descent</p> <ol style="list-style-type: none"> 1. Constrictive pericarditis 2. Restrictive myopathies 3. Tricuspid regurgitation <p>J. Blunted <i>x</i> descent</p> <ol style="list-style-type: none"> 1. Atrial fibrillation 2. Right atrial ischemia <p>K. Blunted <i>y</i> descent</p> <ol style="list-style-type: none"> 1. Tamponade 2. Right ventricular ischemia 3. Tricuspid stenosis <p>L. Miscellaneous abnormalities</p> <ol style="list-style-type: none"> 1. Kussmaul sign (inspiratory rise or lack of decline in right atrial pressure): constrictive pericarditis, right ventricular ischemia 2. Equalization (≤ 5 mm Hg) of mean right atrial ventricular diastolic, pulmonary artery diastolic, pulmonary capillary wedge, and pericardial pressures in tamponade 3. M or W patterns: right ventricular ischemia, pericardial constriction, congestive heart failure 4. Ventricularization of the right atrial pressure: severe tricuspid regurgitation 5. Sawtooth pattern: atrial flutter 6. Dissociation between pressure recording and intracardiac electrocardiogram: Ebstein anomaly 	<p>II. Left atrial pressure–pulmonary capillary wedge pressure waveforms</p> <p>A. Low mean pressure</p> <ol style="list-style-type: none"> 1. Hypovolemia 2. Improper zeroing of the transducer <p>B. Elevated mean pressure</p> <ol style="list-style-type: none"> 1. Intravascular volume overload states 2. Left ventricular failure due to valvular disease (mitral or aortic stenosis or regurgitation) 3. Left ventricular failure due to myocardial disease (ischemia or cardiomyopathy) 4. Left ventricular failure due to systemic hypertension 5. Pericardial effusion with tamponade physiology 6. Obstructive atrial myxoma <p>C. Elevated <i>a</i> wave (any increased resistance to ventricular filling)</p> <ol style="list-style-type: none"> 1. Mitral stenosis 2. Decreased ventricular compliance due to ventricular failure, aortic valve stenosis, or systemic hypertension <p>D. Cannon <i>a</i> wave</p> <ol style="list-style-type: none"> 1. Atrial-ventricular asynchrony (atria contract against a closed mitral valve, as during complete heart block, following premature ventricular contraction, during ventricular tachycardia, or with ventricular pacemaker) <p>E. Absent <i>a</i> wave</p> <ol style="list-style-type: none"> 1. Atrial fibrillation or atrial standstill 2. Atrial flutter <p>F. Elevated <i>v</i> wave</p> <ol style="list-style-type: none"> 1. Mitral regurgitation 2. Left ventricular heart failure 3. Ventricular septal defect <p>G. <i>a</i> wave equal to <i>v</i> wave</p> <ol style="list-style-type: none"> 1. Tamponade 2. Constrictive pericardial disease 3. Hypervolemia <p>H. Prominent <i>x</i> descent</p> <ol style="list-style-type: none"> 1. Tamponade 2. Subacute constriction and possibly chronic constriction <p>I. Prominent <i>y</i> descent</p> <ol style="list-style-type: none"> 1. Constrictive pericarditis 2. Restrictive myopathies 3. Mitral regurgitation <p>J. Blunted <i>x</i> descent</p> <ol style="list-style-type: none"> 1. Atrial fibrillation 2. Atrial ischemia <p>K. Blunted <i>y</i> descent</p> <ol style="list-style-type: none"> 1. Tamponade 2. Ventricular ischemia 3. Mitral stenosis <p>L. Pulmonary capillary wedge pressure not equal to left ventricular end-diastolic pressure</p> <ol style="list-style-type: none"> 1. Mitral stenosis 2. Left atrial myxoma 3. Cor triatriatum 4. Pulmonary venous obstruction 5. Decreased ventricular compliance 6. Increased pleural pressure 7. Placement of catheter in a nondependent zone of the lung
<p>III. Pulmonary artery pressure waveforms</p> <p>A. Elevated systolic pressure</p> <ol style="list-style-type: none"> 1. Primary pulmonary hypertension 2. Mitral stenosis or regurgitation 3. Congestive heart failure 4. Restrictive myopathies 5. Significant left-to-right shunt 6. Pulmonary disease (pulmonary embolism, hypoxemia, chronic obstructive pulmonary disease) 	

Continued



TABLE 20-4 Pathologic Waveforms—cont'd

<p>B. Reduced systolic pressure</p> <ol style="list-style-type: none"> 1. Hypovolemia 2. Pulmonary artery stenosis 3. Subvalvular or supra-ventricular stenosis 4. Ebstein anomaly 5. Tricuspid stenosis 6. Tricuspid atresia <p>C. Reduced pulse pressure</p> <ol style="list-style-type: none"> 1. Right-heart ischemia 2. Right ventricular infarction 3. Pulmonary embolism 4. Tamponade <p>D. Bifid pulmonary artery waveform</p> <ol style="list-style-type: none"> 1. Large left atrial v wave transmitted backward (i.e., mitral regurgitation) <p>E. Pulmonary artery diastolic pressure higher than pulmonary capillary wedge pressure</p> <ol style="list-style-type: none"> 1. Pulmonary disease 2. Pulmonary embolism 3. Tachycardia 	<p>E. Diminished or absent <i>a</i> wave</p> <ol style="list-style-type: none"> 1. Atrial fibrillation or flutter 2. Tricuspid or mitral stenosis 3. Tricuspid or mitral regurgitation when ventricular compliance is increased <p>F. Dip and plateau in diastolic pressure wave</p> <ol style="list-style-type: none"> 1. Constrictive pericarditis 2. Restrictive myopathies 3. Right ventricular ischemia 4. Acute dilation associated with <ol style="list-style-type: none"> a. Tricuspid regurgitation b. Mitral regurgitation <p>G. Left ventricular end-diastolic pressure higher than right ventricular end-diastolic pressure</p> <ol style="list-style-type: none"> 1. Restrictive myopathies
<p>IV. Ventricular pressure waveforms</p> <p>A. Systolic pressure elevated</p> <ol style="list-style-type: none"> 1. Pulmonary or systemic hypertension 2. Pulmonary valve or aortic stenosis 3. Ventricular outflow tract obstruction 4. Supra-ventricular obstruction 5. Right ventricular pressure elevation with significant <ol style="list-style-type: none"> a. Atrial septal defect b. Ventricular septal defect 6. Right ventricular pressure elevation due to factors that increase pulmonary vascular resistance (see factors that increase right atrial pressure) <p>B. Systolic pressure reduced</p> <ol style="list-style-type: none"> 1. Hypovolemia 2. Cardiogenic shock 3. Tamponade <p>C. End-diastolic pressure elevated</p> <ol style="list-style-type: none"> 1. Hypervolemia 2. Congestive heart failure 3. Diminished compliance 4. Hypertrophy 5. Tamponade 6. Regurgitant valvular disease 7. Pericardial constriction <p>D. End-diastolic pressure reduced</p> <ol style="list-style-type: none"> 1. Hypovolemia 2. Tricuspid or mitral stenosis 	<p>V. Aortic pressure waveforms</p> <p>A. Systolic pressure elevated</p> <ol style="list-style-type: none"> 1. Systemic hypertension 2. Arteriosclerosis 3. Aortic insufficiency <p>B. Systolic pressure reduced</p> <ol style="list-style-type: none"> 1. Aortic stenosis 2. Heart failure 3. Hypovolemia <p>C. Widened pulse pressure</p> <ol style="list-style-type: none"> 1. Systemic hypertension 2. Aortic insufficiency 3. Significant patent ductus arteriosus 4. Significant ruptures of sinus of Valsalva aneurysm <p>D. Reduced pulse pressure</p> <ol style="list-style-type: none"> 1. Tamponade 2. Congestive heart failure 3. Cardiogenic shock 4. Aortic stenosis <p>E. Pulsus bisferiens</p> <ol style="list-style-type: none"> 1. Aortic insufficiency 2. Obstructive hypertrophic cardiomyopathy <p>F. Pulsus paradoxus</p> <ol style="list-style-type: none"> 1. Tamponade 2. Obstructive airway disease 3. Pulmonary embolism <p>G. Pulsus alternans</p> <ol style="list-style-type: none"> 1. Congestive heart failure 2. Cardiomyopathy <p>H. Pulsus parvus et tardus</p> <ol style="list-style-type: none"> 1. Aortic stenosis <p>I. Spike-and-dome configuration</p> <ol style="list-style-type: none"> 1. Obstructive hypertrophic cardiomyopathy

Cardiac Output Measurements

There is no completely accurate method of measuring cardiac output in all patients, but it can be estimated on the basis of various assumptions. The two most commonly used methods are the Fick method and the thermodilution method. For comparison among patients, cardiac output is often corrected for the patient's size on the basis of the body surface area and expressed as cardiac index.

THERMODILUTION TECHNIQUES. The thermodilution procedure requires injection of a bolus of liquid (usually normal saline) into the proximal port of the catheter. The resultant change in temperature in the liquid is measured by a thermistor mounted in the distal end of the catheter. The change in temperature versus time is graphed. The cardiac output is then calculated by use of an equation that considers the temperature and specific gravity of the injectate and the temperature and specific gravity of the blood along with the injectate volume. A calibration factor is also used. The cardiac output is inversely related to the area under a thermodilution curve, shown as a function of temperature versus time, with a smaller area under the curve indicative of a higher cardiac output (**Fig. 20-11**). Temperature fluctuation

in the circuit can affect accuracy,³⁸ however, and the use of two thermistors can significantly improve the accuracy of this technique.³⁹

The thermodilution method has several advantages. It obviates the need for withdrawal of blood from an arterial site and is less affected by recirculation. Perhaps its greatest advantage is the rapid display of results with computerized methods. However, a significant error occurs in patients with severe tricuspid regurgitation. Also, in patients with low outputs (especially <2.5 liter/min), thermodilution tends to overestimate the cardiac output.

FICK METHOD. The Fick principle assumes that the rate at which oxygen is consumed is a function of the rate of blood flow times the rate of oxygen pick-up by the red blood cells. The basic assumption is that the flow of blood in a given period is equal to the amount of substance entering the stream of flow in the same period divided by the difference between the concentrations of the substance in the blood upstream and downstream from its point of entry into the circulation (**Fig. 20-12**). The same number of red blood cells that enter the lung must leave the lung if no intracardiac shunt is present. Thus, if certain parameters are known (the number of oxygen molecules

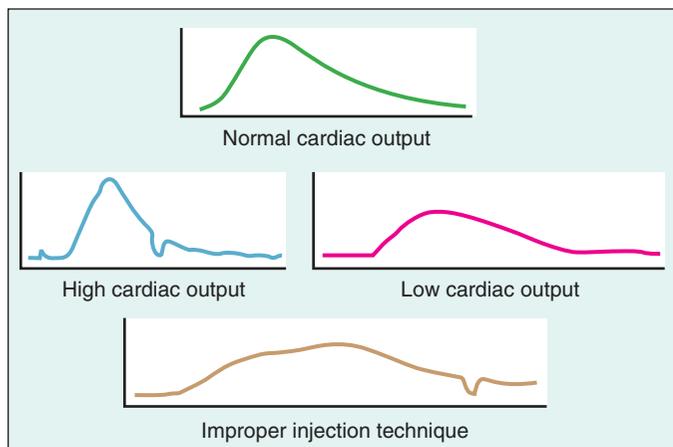


FIGURE 20-11 Thermodilution cardiac output curves. A normal curve has a sharp upstroke after an injection of saline. A smooth curve with a mildly prolonged downslope occurs until it is back to baseline. The area under the curve is inversely related to the cardiac output. At low cardiac output, a prolonged period is required to return to baseline. Therefore, there is a larger area under the curve. In a high cardiac output state, the cooler saline injectate moves faster through the right side of the heart, and temperature returns to baseline more quickly. The area under the curve is smaller and the output is higher.

that are attached to the red blood cells entering the lung, the number of oxygen molecules that are attached to the red blood cells leaving the lung, and the number of oxygen molecules consumed during travel through the lung), the rate of flow of these red blood cells as they pass through the lung can be determined. This can be expressed in the following terms:

$$\text{Fick cardiac output (liter/min)} = \frac{\text{oxygen consumption (mL/min)}}{A\text{-VO}_2 \times 1.36 \times \text{Hgb} \times 10}$$

where $A\text{-VO}_2$ is the arterial-venous oxygen saturation difference, Hgb is the hemoglobin concentration (mg/dL), and the constant 1.36 is the oxygen-carrying capacity of hemoglobin (expressed in mL O_2 /g Hgb).

Measurements must be made in the steady state. Automated methods can accurately determine the oxygen content within the blood samples. Thus, the greatest source of measurement variability is the oxygen consumption. In the original Fick determinations, expiratory gas samples were collected in a large bag during a specified period. By measurement of the expiratory oxygen concentration and knowing the concentration of oxygen in room air, the quantity of oxygen consumed over time could be determined. Currently, measurement of the expired oxygen concentration is quantified by use of a polarograph. This device can be connected to the patient by a plastic hood or by a mouthpiece and tubing.

The advantage of the Fick method is that it is the most accurate method in patients with low cardiac output and thus is preferred to the thermodilution method in these circumstances. It is also independent of the factors that affect curve shape and cause errors in thermodilution cardiac output (e.g., tricuspid regurgitation). The Fick method suffers primarily from the difficulty in obtaining accurate oxygen consumption measurements and the inability to obtain a steady state under certain conditions. Because the method assumes mean flow over time, it is not suitable during rapid changes in flow. Also, the patient cannot be receiving supplemental oxygen during blood sample collection. In patients with significant mitral or aortic regurgitation, the Fick cardiac output should not be used.

Many laboratories use an "assumed" Fick method, in which the oxygen consumption index is assumed on the basis of the patient's age, gender, and body surface area or an estimate is made (125 mL/ m^2) on the basis of body surface area. However, when assumed oxygen consumption rather than measured oxygen consumption is used, large errors can occur.⁴⁰

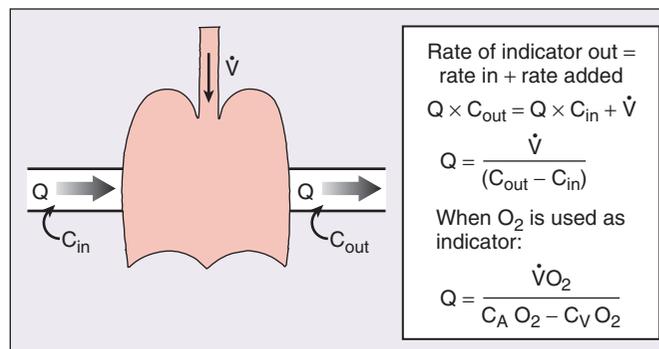


FIGURE 20-12 Schematic illustration of flow measurement by the Fick principle. Fluid containing a known concentration of an indicator (C_{in}) enters a system at flow rate Q . As the fluid passes through the system, indicator is continuously added at rate \dot{V} , raising the concentration in the outflow to C_{out} . In a steady state, the rate of indicator leaving the system (QC_{out}) must equal the rate at which it enters (QC_{in}) plus the rate at which it is added (\dot{V}). When oxygen is used as the indicator, cardiac output can be determined by measuring oxygen consumption ($\dot{V}\text{O}_2$), arterial oxygen content ($C_A\text{O}_2$), and mixed venous oxygen content ($C_V\text{O}_2$). (From Winniford MD, Kern MJ, Lambert CR: *Blood flow measurement*. In Pepine CJ, Hill JA, Lambert CR [eds]: *Diagnostic and Therapeutic Cardiac Catheterization*. 3rd ed. Baltimore, Williams & Wilkins, 1998, p 400.)

ANGIOGRAPHIC CARDIAC OUTPUT. Angiographic stroke volume can be calculated by tracing the LV end-diastolic and end-systolic images. Stroke volume is the quantity of blood ejected with each beat. End-diastolic volume is the maximum LV volume and occurs immediately before the onset of systole. It occurs immediately after atrial contraction in patients in sinus rhythm. End-systolic volume is the minimum LV volume during the cardiac cycle. Calibration of the images with grids or ventricular phantoms is necessary to obtain accurate ventricular volumes. Angiographic cardiac output and stroke volume are derived from the following equations:

$$\text{Stroke volume} = \text{EDV} - \text{ESV}$$

$$\text{Cardiac output} = (\text{EDV} - \text{ESV}) \times \text{heart rate}$$

where EDV is end-diastolic volume and ESV is end-systolic volume.

The inherent inaccuracies of calibrating angiographic volumes often make this method of measurement unreliable. In cases of valvular regurgitation or atrial fibrillation, angiographic cardiac output does not accurately measure true systemic outputs. However, the angiographic cardiac output is preferred to the Fick or thermodilution output for calculation of stenotic valve areas in patients with significant aortic or mitral regurgitation.

DETERMINATION OF VASCULAR RESISTANCE. Vascular resistance calculations are based on hydraulic principles of fluid flow, in which resistance is defined as the ratio of the decrease in pressure between two points in a vascular segment and the blood flow through the segment. Although this straightforward analogy to Ohm's law represents an oversimplification of the complex behavior of pulsatile flow in dynamic and diverse vascular beds, the calculation of vascular resistance based on these principles has proved to be of value in a number of clinical settings.

Determination of the resistance in a vascular bed requires measurement of the mean pressure of the proximal and distal ends of the vascular bed and accurate measurement of cardiac output. Vascular resistance (R) is usually defined in absolute units ($\text{dyne}\cdot\text{sec} \cdot \text{cm}^{-5}$) and is defined as $R = [\text{mean pressure gradient (dyne}/\text{cm}^2)]/[\text{mean flow (cm}^3/\text{sec)}]$. Hybrid units (Wood units) are less often used.⁴¹

Systemic vascular resistance (SVR) in absolute units is calculated by the following equation:

$$\frac{\text{SVR}}{Q_s} = 80(Ao_m - RA_m)$$

where Ao_m and RA_m are the mean pressures (in mm Hg) in the aorta and right atrium, respectively, and Q_s is the systemic cardiac output (in liters/min). The constant 80 is used to convert units from mm Hg/liter/min (Wood units) to the absolute resistance units $\text{dyne}\cdot\text{sec} \cdot \text{cm}^{-5}$. If the right atrial pressure is not known, the term RA_m can be dropped, and the resulting value is called the total peripheral resistance (TPR):



$$\frac{TPR}{Q_s} = 80(Ao_m)$$

Similarly, the pulmonary vascular resistance (PVR) is derived from the following equation:

$$\frac{PVR}{Q_p} = 80(PA_m - LA_m)$$

where PA_m and LA_m are the mean pulmonary artery and left atrial pressures, respectively, and Q_p is the pulmonary blood flow. If the mean left atrial pressure has not been measured directly, mean pulmonary capillary wedge pressure is commonly substituted for it, although errors can occur because of this substitution. In the absence of an intracardiac shunt, Q_p is equal to the systemic cardiac output. Normal values are listed in Table 20-3.

Elevated resistances in the systemic and pulmonary circuits may represent reversible abnormalities or may be permanent because of irreversible anatomic changes. In several clinical situations, such as congestive heart failure, valvular heart disease, primary pulmonary hypertension, and congenital heart disease with intracardiac shunting, determination of whether elevated SVR or PVR can be lowered transiently in the catheterization laboratory may provide important insights into potential management strategies. Interventions that may be used in the laboratory for this purpose include administration of vasodilating drugs (e.g., sodium nitroprusside), exercise, and (in patients with pulmonary hypertension) nitric oxide inhalation or intravenous epoprostenol (Flolan), a pulmonary and systemic vasodilator (see Chap. 78).

Vascular impedance measurements account for blood viscosity, pulsatile flow, reflected waves, and arterial compliance. Hence, vascular impedance has the potential to describe the dynamic relation between pressure and flow more comprehensively than is possible with the simpler calculations of vascular resistance. However, because the simultaneous pressure and flow data required for the calculation of impedance are complex and difficult to obtain, the concept of impedance has failed to gain widespread acceptance, and vascular impedance has not been adopted as a routine clinical index.

Evaluation of Valvular Stenosis

Determination of the severity of valvular stenosis on the basis of the pressure gradient and flow across the valve is one of the most important aspects of evaluation of patients with valvular heart disease (see Chap. 66). In many patients, the magnitude of the pressure gradient alone is sufficient to distinguish clinically significant from insignificant valvular stenosis.

DETERMINATION OF PRESSURE GRADIENTS

Aortic Stenosis

In patients with aortic stenosis, the transvalvular pressure gradient is best measured with a catheter in the left ventricle and another in the proximal aorta. Although it is convenient to measure the gradient between the left ventricle and the femoral artery, downstream augmentation of the pressure signal and delay in pressure transmission between the proximal aorta and femoral artery may alter the pressure waveform substantially and introduce errors into the measured gradient.³⁷

LV–femoral artery pressure gradients may suffice in many patients as an estimate of the severity of aortic stenosis to confirm the presence of a severely stenotic valve. If the side port of the arterial introducing sheath is used to monitor femoral pressure, the inner diameter of the sheath should be at least 1F size larger than the outer diameter of the LV catheter.

The operator should obtain simultaneous ascending aortic and femoral artery pressures to verify similarity between the two sites. The LV–femoral artery pressure gradient may not always be relied on in the calculation of valve orifice area in patients with moderate valve gradients. A careful single catheter pull-back from left ventricle to aorta is often preferable to simultaneous measurement of LV and femoral artery pressures.

A single catheter with a distal and a proximal lumen or a micro-manometer catheter with distal and proximal transducers is preferable for simultaneous measurement of LV pressure and central aortic

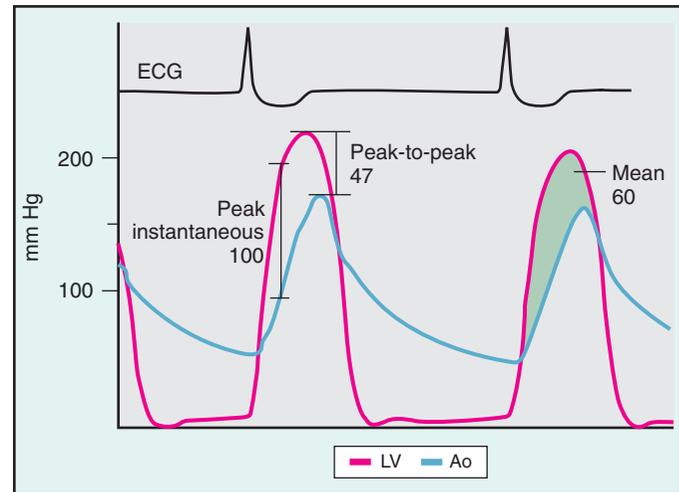


FIGURE 20-13 Various methods of describing an aortic transvalvular gradient. The peak-to-peak gradient (47 mm Hg) is the difference between the maximal pressure in the aorta (Ao) and the maximal left ventricle (LV) pressure. The peak instantaneous gradient (100 mm Hg) is the maximal pressure difference between the Ao and LV when the pressures are measured in the same moment (usually during early systole). The mean gradient (green shaded area) is the integral of the pressure difference between the LV and Ao during systole (60 mm Hg). (From Bashore TM: *Invasive Cardiology: Principles and Techniques*. Philadelphia, BC Decker, 1990.)

pressure. Another method is to use a long arterial catheter in the aorta and the second in the left ventricle.

The mean pressure gradient across the aortic valve is determined by planimetry of the area separating the LV and aortic pressures using multiple beats (Fig. 20-13), and it is this gradient that is applied to calculation of the valve orifice area. The peak-to-peak gradient, measured as the difference between peak LV pressure and peak aortic pressure, is commonly used to quantify the valve gradient because this measurement is rapidly obtained and can be estimated visually. However, there is no physiologic basis for the peak-to-peak gradient because the maximum LV and aortic pressures rarely occur simultaneously. The peak-to-peak gradient measured in the catheterization laboratory is generally lower than the peak instantaneous gradient measured in the echocardiography laboratory. This is because the peak instantaneous gradient represents the maximum pressure difference between the left ventricle and aorta when pressures are measured simultaneously. This maximum pressure difference occurs on the upslope of the aortic pressure tracing (see Fig. 20-13). Mean aortic transvalvular gradient and aortic valve area are well correlated with both techniques.⁴² In patients with low-gradient low-output aortic stenosis, pharmacologic maneuvers can be helpful (see section on pharmacologic maneuvers).

Mitral Stenosis

In patients with mitral stenosis, the most accurate means of determining the mitral valve gradient is measurement of left atrial pressure by the transeptal technique with simultaneous measurement of LV pressure and with planimetry of the area bounded by the LV and left atrial pressures in diastole using several cardiac cycles (Fig. 20-14). The pulmonary capillary wedge pressure is usually substituted for the left atrial pressure, as the pulmonary wedge pressure is more readily obtained. The pulmonary wedge pressure tracing must be realigned with the LV tracing for accurate mean gradient determination. Although it has been generally accepted that pulmonary capillary wedge pressure is a satisfactory estimate of left atrial pressure, studies indicate that the pulmonary wedge pressure may systematically overestimate the left atrial pressure by 2 to 3 mm Hg, thereby increasing the measured mitral valve gradient.³⁶ Improperly wedged catheters, resulting in damped pulmonary artery pressure recordings, further overestimate the severity of mitral stenosis. If there is doubt about accurate positioning of the catheter in the wedge position, the position

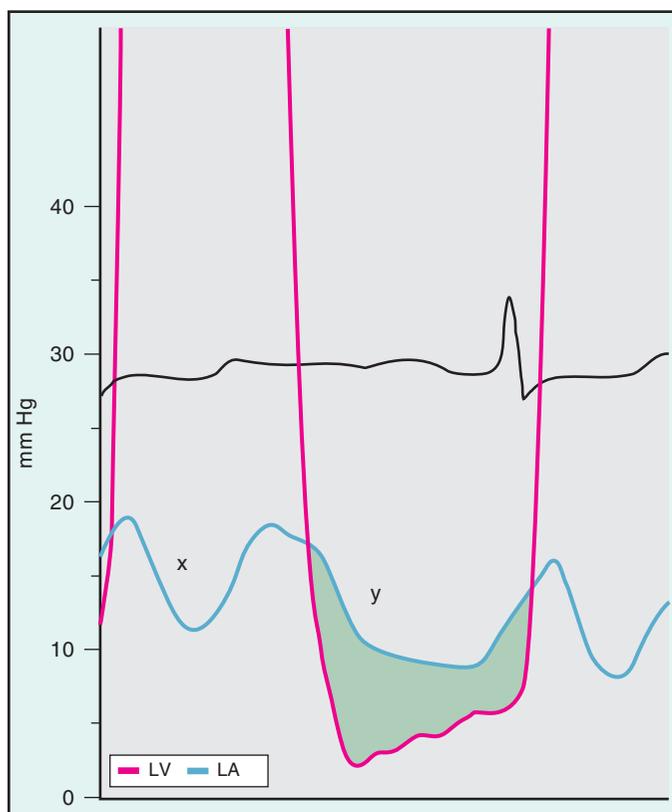


FIGURE 20-14 Pressure gradient in a patient with mitral stenosis. The pressure in the left atrium (LA) exceeds the pressure in the left ventricle (LV) during diastole, producing a diastolic pressure gradient (green shaded area). (From Bashore TM: *Invasive Cardiology: Principles and Techniques*. Philadelphia, BC Decker, 1990.)

can be confirmed by slow withdrawal of blood for oximetric analysis. An oxygen saturation equal to that of the systemic circulation confirms the wedge position.

Right-Sided Valvular Stenosis

In pulmonic stenosis, the valve gradient is obtained by a catheter pull-back from the pulmonary artery to the right ventricle or by placement of separate catheters in the right ventricle and pulmonary artery. Multilumen catheters can also be used for simultaneous pressure recordings. Tricuspid valve gradients should be assessed with simultaneous recording of right atrial and RV pressures.

CALCULATION OF STENOTIC VALVE ORIFICE AREAS. The stenotic orifice area is determined from the pressure gradient and cardiac output with the formula developed by Gorlin and Gorlin from the fundamental hydraulic relationships linking the area of an orifice to the flow and pressure drop across the orifice. Flow (F) and orifice area (A) are related by the fundamental formula

$$F = cAV$$

where V is velocity of flow and c is a constant accounting for central streaming of fluid through an orifice, which tends to reduce the effective orifice size. Hence,

$$A = F/cV$$

Velocity is related to the pressure gradient through the relation $V = k(2g\Delta P)^{1/2}$, where k is a constant accounting for frictional energy loss, g is the acceleration due to gravity (980 cm/sec²), and ΔP is the mean pressure gradient (mm Hg). Substituting for V in the orifice area equation and combining c and k into one constant C,

$$A = \frac{F}{44.3C\sqrt{(\Delta P)}}$$

Gorlin and Gorlin determined the value of the constant C by comparing the calculated valve area with actual valve area measured at autopsy or at surgery in 11 mitral valves. The maximal discrepancy between the actual mitral valve area and calculated values was only 0.2 cm² when the constant 0.85 was used. No data were obtained for aortic valves, a limitation noted by the Gorlins, and a constant of 1.0 was assumed.

Because flow across the aortic valve occurs only in systole, the flow value for calculating aortic valve area is the cardiac output in milliliters per minute divided by the systolic ejection period (SEP) in seconds per beat times the heart rate (HR) in beats per minute. The systolic ejection period is defined from aortic valve opening to closure. Hence, the aortic valve area (AVA) is calculated from the Gorlin formula by the following equation:

$$AVA \text{ (cm}^2\text{)} = \frac{\text{cardiac output (liters/min)} \times 1000}{(44.3)(\text{HR})(\text{SEP})\sqrt{\text{mean gradient}}}$$

Similarly, as mitral flow occurs only in diastole, the cardiac output is corrected for the diastolic filling period (DFP) in seconds per beat in the equation for mitral valve area (MVA), where the diastolic filling period is defined from mitral valve opening to mitral valve closure:

$$MVA \text{ (cm}^2\text{)} = \frac{\text{cardiac output (liters/min)} \times 1000}{(37.7)(\text{HR})(\text{DFP})\sqrt{\text{mean gradient}}}$$

The normal aortic valve area is 2.6 to 3.5 cm² in adults. Valve areas of less than 1.0 cm² represent severe aortic stenosis (see Chap. 66). The normal mitral valve area is 4 to 6 cm², and severe mitral stenosis is present with valve areas of less than 1.0 cm².

The calculated valve area is often crucial in management decisions for patients with aortic stenosis or mitral stenosis. Hence, it is essential that accurate and simultaneous pressure gradient and cardiac output determinations be made, especially in patients with borderline or low-pressure gradients.

There are limitations of the Gorlin-derived orifice area. As the square root of the mean gradient is used in the Gorlin formula, the valve area calculation is more strongly influenced by the cardiac output than the pressure gradient. Thus, errors in measuring cardiac output may have profound effects on the calculated valve area, particularly in patients with low cardiac outputs, in whom the calculated valve area is often of greatest importance.

As noted previously, the thermodilution technique may provide inaccurate cardiac output data when cardiac output is reduced or when concomitant aortic, mitral, or tricuspid regurgitation is present. Thus, the Fick method of determining cardiac output is the most accurate for assessing cardiac output, especially in low-output states. In patients with mixed valvular disease (stenosis and regurgitation) of the same valve, the use of forward flow as determined by the Fick method or thermodilution technique overestimates the severity of the valvular stenosis. This overestimation occurs because the Gorlin formula depends on total forward flow across the stenotic valve, not net forward flow. If valvular regurgitation is present, the angiographic cardiac output is the most appropriate measure of flow. If both aortic and mitral regurgitation are present, flow across a single valve cannot be determined, and neither aortic valve area nor mitral valve area can be assessed accurately.

Other potential errors and limitations are inherent in the use of the Gorlin formula, related both to inaccuracies in measurement of valve gradients and to more fundamental issues regarding the validity of the assumptions underlying the formula. In low-output states, the Gorlin formula may systematically predict smaller valve areas than are actually present. Several lines of evidence indicate that the aortic valve area from the Gorlin formula increases with increases in cardiac output. Although this may represent an actual greater opening of stenotic valves by the higher proximal opening pressures that result from increases in transvalvular flow, the flow dependence of



the calculated valve area may also reflect inherent errors in the assumptions underlying the Gorlin formula, particularly with respect to the aortic valve.

The increase in Gorlin valve area with increases in transvalvular flow is not associated with alterations in direct planimetry of the aortic valve area by transesophageal echocardiography. This suggests that flow-related variation in the Gorlin aortic valve area is due to disproportional flow dependence of the formula and not a true change in the valve area.⁴³

An alternative simplified formula to determine valve areas has been proposed. The effects of the systolic ejection period and the diastolic filling period are relatively constant at normal heart rates, and these terms can be eliminated from the equation. This assumes that $(44.3 \times \text{HR} \times \text{SEP}) \approx 1000$ in most circumstances. In this modified approach, the aortic valve area can be quickly estimated from the following formula:

$$\text{AVA (cm}^2\text{)} = \frac{\text{cardiac output (liters/min)}}{\sqrt{\text{peak to peak or mean gradient (mm Hg)}}}$$

Use of either the mean aortic transvalvular gradient or the peak-to-peak gradient produces similar correlation with the Gorlin formula.

Patients with low-output, low-gradient aortic stenosis remain a challenge for accurate determination of valve area by either cardiac catheterization or echocardiography (see Chaps. 15 and 66). Whether afterload mismatch or intrinsic contractility dysfunction is the primary problem in ventricular impairment can be difficult to ascertain. Thus, the use of pharmacologic stress with low-dose dobutamine infusion has been advocated to distinguish moderate from severe aortic stenosis.^{44,46} The concept is that patients without true anatomic severe aortic stenosis will have an increase in valve areas with little change in transvalvular gradient.² If dobutamine increases the aortic valve area more than 0.2 cm² with no change in gradient, it is likely that the baseline evaluation overestimated the severity of aortic stenosis.² It has also been shown that patients who increase stroke volume by less than 20% lack contractile reserve and have a poor prognosis with either medical or surgical therapy.⁴⁵

Despite theoretical limitations, the Gorlin formula has proved to be a reliable clinical determination for evaluation of patients with suspected aortic stenosis.

Measurement of Intraventricular Pressure Gradients

The demonstration of an intracavitary pressure gradient is among the most interesting yet challenging aspects of diagnostic catheterization (see Chap. 69). Simultaneous pressure measurements are obtained in either the central aorta or femoral artery and from within the ventricular cavity. Pull-back of a multipurpose end-hole catheter from the ventricular apex to a posterior position just beneath the aortic valve demonstrates an intracavitary gradient. An erroneous intracavitary gradient may be seen if the catheter becomes entrapped by the hypertrophic myocardium.

The intracavitary gradient is distinguished from aortic valvular stenosis by the loss of the aortic-LV gradient when the catheter is still within the left ventricle yet proximal to the myocardial obstruction. In addition, careful analysis of the upstroke of the aortic pressure waveform distinguishes a valvular from a subvalvular stenosis, as the aortic pressure waveform demonstrates a slow upstroke in aortic stenosis. Other methods to localize intracavitary gradients include the use of a dual-lumen catheter, use of a double-sensor micromanometer catheter, or placement of an end-hole catheter in the LV outflow tract while a transseptal catheter is advanced into the left ventricle, with pressure measured simultaneously. An intracavitary gradient may be increased by various provocative maneuvers including the Valsalva maneuver, inhalation of amyl nitrate, introduction of a premature ventricular beat, or isoproterenol infusion (see Physiologic and Pharmacologic Maneuvers).

Assessment of Valvular Regurgitation

The severity of valvular regurgitation is generally graded by visual assessment, although calculation of the regurgitant fraction is used occasionally. According to ACC/AHA guidelines, hemodynamic evaluation of either aortic regurgitant or mitral regurgitant lesions is recommended as a Class I indication when pulmonary artery pressure is disproportionate to the severity of regurgitation assessed noninvasively or when there is a discrepancy between clinical and noninvasive findings.² Exercise with right-heart hemodynamic assessment including pulmonary artery pressure, pulmonary capillary wedge pressure, and cardiac output may also provide useful information.

VISUAL ASSESSMENT OF REGURGITATION. Valvular regurgitation may be assessed visually by determination of the relative amount of radiographic contrast medium that opacifies the chamber proximal to its injection. The estimation of regurgitation depends on the regurgitant volume as well as on the size and contractility of the proximal chamber. The original classification scheme devised by Sellers and colleagues remains the standard in most catheterization laboratories:

- + Minimal regurgitant jet seen. Clears rapidly from proximal chamber with each beat.
- ++ Moderate opacification of proximal chamber, clearing with subsequent beats.
- +++ Intense opacification of proximal chamber, becoming equal to that of the distal chamber.
- ++++ Intense opacification of proximal chamber, becoming more dense than that of the distal chamber. Opacification often persists over the entire series of images obtained.

REGURGITANT FRACTION. A gross estimate of the degree of valvular regurgitation may be obtained by determination of the regurgitant fraction (RF). The difference between the angiographic stroke volume and the forward stroke volume can be defined as the regurgitant stroke volume.

$$\text{Regurgitant stroke volume} = \text{angiographic stroke volume} - \text{forward stroke volume}$$

The RF is that portion of the angiographic stroke volume that does not contribute to the net cardiac output. Forward stroke volume is the cardiac output determined by the Fick or thermodilution method divided by the heart rate. Thermodilution cardiac output cannot be used if there is significant concomitant tricuspid regurgitation.

Compared with visual interpretation, 1+ regurgitation is roughly equivalent to an RF of 20% or less, 2+ to an RF of 21% to 40%, 3+ to an RF of 41% to 60%, and 4+ to an RF of more than 60%.

The assumption underlying the determination of RF is that the angiographic and forward cardiac outputs are accurate and comparable, a state requiring similar heart rates, stable hemodynamic states between measurements, and only a single regurgitant valve. Given these conditions, the equation yields only a gross approximation of regurgitant flow.

Shunt Determinations

Normally, pulmonary blood flow and systemic blood flow are equal. With an abnormal communication between intracardiac chambers or great vessels, blood flow is shunted from the systemic circulation to the pulmonary circulation (left-to-right shunt), from the pulmonary circulation to the systemic circulation (right-to-left shunt), or in both directions (bidirectional shunt). The most commonly used method for shunt determination in the cardiac catheterization laboratory is the oximetric method. Although many shunts are suspected before cardiac catheterization, physicians performing the procedure should be vigilant in determining the cause of unexpected findings. For example, an unexplained pulmonary artery oxygen saturation exceeding 80% should raise the operator's suspicion of a left-to-right shunt, whereas unexplained arterial desaturation (<93%) may indicate a right-to-left shunt.⁴⁷ Arterial desaturation commonly results from alveolar hypoventilation and associated "physiologic shunting," the causes of which include oversedation from premedication, pulmonary disease, pulmonary venous congestion, pulmonary edema, and cardiogenic shock. If arterial desaturation persists after the patient takes several deep breaths or after administration of 100% oxygen, a right-to-left shunt is likely.

OXIMETRIC METHOD. The oximetric method is based on blood sampling from various cardiac chambers for the determination of oxygen saturation. A left-to-right shunt is detected when a significant increase in blood oxygen saturation is found between two right-sided vessels or chambers.



A screening oxygen saturation measurement for any left-to-right shunt is often performed with right-heart catheterization by sampling of blood in the SVC and the pulmonary artery. If the difference in oxygen saturation between these samples is 8% or more, a left-to-right shunt may be present, and an oximetry "run" should be performed. This run obtains blood samples from all right-sided locations, including the SVC, IVC, right atrium, right ventricle, and pulmonary artery. In cases of interatrial or interventricular shunts, it is recommended to obtain multiple samples from the high, middle, and low right atrium or the RV inflow tract, apex, and outflow tract to localize the level of the shunt. One may miss a small left-to-right shunt using the right atrium for screening purposes rather than the SVC because of incomplete mixing of blood in the right atrium, which receives blood from the IVC, SVC, and coronary sinus. Oxygen saturation in the IVC is higher than in the SVC because the kidneys use less oxygen relative to their blood flow than do other organs, whereas coronary sinus blood has very low oxygen saturation. Mixed venous saturation is most accurately measured in the pulmonary artery after complete mixing has occurred or can be calculated by IVC + SVC samples (see Shunt Quantification).

A full saturation run obtains samples from the high and low IVC; high and low SVC; high, middle, and low right atrium; RV inflow and outflow tracts and midcavity; main pulmonary artery; left or right pulmonary artery; pulmonary vein and left atrium, if possible; left ventricle; and distal aorta. When a right-to-left shunt must be localized, oxygen saturation samples must be taken from the pulmonary veins, left atrium, left ventricle, and aorta. Although the major weakness of the oxygen step-up method is its lack of sensitivity, clinically significant shunts are generally detected by this technique. Obtaining multiple samples from each chamber can improve sampling error and variability. Another method of oximetric determination of intracardiac shunts uses a balloon-tipped fiberoptic catheter that allows continuous registration of oxygen saturation as it is withdrawn from the pulmonary artery through the right-heart chambers into the SVC and IVC.

SHUNT QUANTIFICATION. The principles used to determine Fick cardiac output are also used to quantify intracardiac shunts. To determine the size of a left-to-right shunt, pulmonary blood flow (PBF) and systemic blood flow (SBF) determinations are required. PBF is simply oxygen consumption divided by the difference in oxygen content across the pulmonary bed, whereas SBF is oxygen consumption divided by the difference in oxygen content across the systemic bed. The effective blood flow (EBF) is the fraction of mixed venous return received by the lungs without contamination by the shunt flow. In the absence of a shunt, PBF, SBF, and EBF all are equal. These equations are as follows:

$$\text{PBF} = \frac{\text{O}_2 \text{ consumption (mL/min)}}{(\text{PVO}_2 - \text{PAO}_2)}$$

$$\text{SBF} = \frac{\text{O}_2 \text{ consumption (mL/min)}}{(\text{SAO}_2 - \text{MVO}_2)}$$

$$\text{EBF} = \frac{\text{O}_2 \text{ consumption (mL/min)}}{(\text{PVO}_2 - \text{MVO}_2)}$$

where PVO_2 , PAO_2 , SAO_2 , and MVO_2 are the oxygen contents (in milliliters of oxygen per liter of blood) of pulmonary venous, pulmonary arterial, systemic arterial, and mixed venous bloods, respectively. The oxygen content is determined as outlined in the section on Fick cardiac output.

If a pulmonary vein is not sampled, systemic arterial oxygen content may be substituted, assuming systemic arterial saturation is 95% or more. As discussed earlier, if systemic arterial saturation is less than 93%, a right-to-left shunt may be present. If arterial desaturation is present but not secondary to a right-to-left shunt, systemic arterial oxygen content is used. If a right-to-left shunt is present, pulmonary venous oxygen content is calculated as 98% of the oxygen capacity.

The mixed venous oxygen content is the average oxygen content of the blood in the chamber proximal to the shunt. When assessing a left-to-right shunt at the level of the right atrium, one must calculate mixed venous oxygen content on the basis of the contributing blood flow from the IVC, SVC, and coronary sinus. The most common method used is the Flamm formula:

$$\text{MVO}_2 = \frac{3(\text{SVC O}_2 \text{ content}) + 1(\text{IVC O}_2 \text{ content})}{4}$$

Assuming conservation of mass, the size of a left-to-right shunt, when there is no associated right-to-left shunt, is simply

$$\text{L} \rightarrow \text{R shunt} = \text{PBF} - \text{SBF}$$

When there is evidence of a right-to-left shunt in addition to a left-to-right shunt, the approximate left-to-right shunt size is

$$\text{L} \rightarrow \text{R shunt} = \text{PBF} - \text{EBF}$$

and the approximate right-to-left shunt size is

$$\text{R} \rightarrow \text{L shunt} = \text{SBF} - \text{EBF}$$

The flow ratio PBF/SBF (or Q_p/Q_s) is used clinically to determine the significance of the shunt. A ratio of less than 1.5 indicates a small left-to-right shunt, and a ratio of 1.5 to 2.0, a moderate-sized shunt. A ratio of 2.0 or more indicates a large left-to-right shunt and generally requires percutaneous or surgical repair to prevent future pulmonary or RV complications. A flow ratio of less than 1.0 indicates a net right-to-left shunt. If oxygen consumption is not measured, the pulmonary-to-systemic blood flow ratio may be calculated as follows:

$$Q_p/Q_s = \text{PBF}/\text{SBF} = \frac{(\text{SAO}_2 - \text{MVO}_2)}{(\text{PVO}_2 - \text{PAO}_2)}$$

where SAO_2 , MVO_2 , PVO_2 , and PAO_2 are systemic arterial, mixed venous, pulmonary venous, and pulmonary arterial blood oxygen saturations, respectively.

INDICATOR-DILUTION METHOD. Although the indicator-dilution method is more sensitive than the oximetric method in detection of small shunts, it cannot be used to localize the level of a left-to-right shunt. An indicator such as indocyanine green dye is injected into a proximal chamber while a sample is taken from a distal chamber with a densitometer, and the density of dye is displayed over time. To detect a left-to-right shunt, dye is injected into the pulmonary artery and sampling is performed in a systemic artery. Presence of a shunt is indicated by early recirculation of the dye on the downslope of the curve.

Physiologic and Pharmacologic Maneuvers

Potentially significant cardiac abnormalities may be absent in the resting condition but may be unmasked by stress. Therefore, if the physician performing a cardiac catheterization procedure cannot elucidate the cause of a patient's symptoms at rest, various physiologic and pharmacologic maneuvers can be considered.

Dynamic Exercise

Dynamic exercise in the catheterization laboratory is performed by supine bicycle ergometry, upright bicycle exercise, or straight arm raises. Upright treadmill exercise can be performed outside the catheterization laboratory by use of a balloon flotation catheter inserted through an antecubital or internal jugular vein to measure pulmonary artery and wedge pressures and cardiac output. The associated changes in the heart rate, cardiac output, oxygen consumption, and intracardiac pressures are monitored at rest and during progressive stages of exercise. Normally, the increased oxygen requirements of exercise are met by an increase in cardiac output and an increase in oxygen extraction from arterial blood. Patients with cardiac dysfunction are unable to increase their cardiac output appropriately in response to exercise and must meet the demands of the exercising muscle groups by increasing the extraction of oxygen from arterial blood, thereby increasing the arteriovenous oxygen difference. The relationship between cardiac output and oxygen consumption is linear, and a regression formula can be used to calculate the predicted cardiac index at a given level of oxygen consumption. The actual cardiac index divided by the predicted cardiac index is defined as the exercise index (see Chap. 14). A value of 0.8 or more indicates a normal cardiac output response to exercise. The exercise factor is another method of describing the same relationship between the cardiac output and oxygen consumption. The exercise factor is the increase in cardiac output divided by the increase in oxygen consumption. Normally, for every 100 mL/min increase in oxygen consumption with exercise, the cardiac output should increase by at least 600 mL/min. Therefore, a normal exercise factor should be 6 or more.⁴⁸

Supine exercise normally causes a rise in mean systemic and pulmonary arterial pressures. There is a proportionately greater decrease

in SVR compared with PVR and an increase in heart rate. Myocardial contractility increases from both increased sympathetic tone and the increase in heart rate. LV ejection fraction rises. During early levels of exercise, increased venous return augments LV end-diastolic volume, leading to an increase in stroke volume. At progressively higher levels of exercise, both LV end-systolic and end-diastolic volumes decrease so that there is a negligible rise in stroke volume. Thus, the augmentation in cardiac output during peak exercise in the catheterization laboratory is generally caused by an increase in heart rate. For this reason, all agents that may impair the chronotropic response should be discontinued before catheterization if exercise is contemplated during the procedure.

Exercise may provoke symptoms in a patient who had been found to have valvular disease of borderline significance in the resting state (see Chap. 66). Exercise increases transvalvular mitral gradient and pulmonary artery pressures in mitral stenosis.

The hemodynamic response to exercise is also useful in evaluating regurgitant valvular lesions. Clinically important valvular regurgitation exists if an increase occurs in LV end-diastolic pressure, pulmonary capillary wedge pressure, and SVR, in conjunction with a reduced exercise index (<0.8) and abnormal exercise factor (<0.6). Simultaneous echocardiographic evaluation of valvular regurgitation is also useful in equivocal cases. Patients with myocardial disease, ischemic or otherwise, may have pronounced increases in LV end-diastolic pressure with exercise.

Pacing Tachycardia

Rapid atrial or ventricular pacing increases myocardial oxygen consumption and myocardial blood flow. With pacing, in contradistinction to dynamic exercise, LV end-diastolic volume decreases, and there is little change in cardiac output.⁴⁹ This method may be used to determine the significance of coronary artery disease or valvular abnormalities. For example, the gradient across the mitral valve increases with rapid atrial pacing because of the increase in heart rate. Pacing has the advantage of allowing greater control and rapid termination of the induced stress.

Physiologic Stress

Various physiologic stresses alter the severity of obstruction in hypertrophic cardiomyopathy (see Chap. 69). The Valsalva maneuver (forcible expiration against a closed glottis) increases the systolic subaortic pressure gradient in the strain phase, during which there is a decrease in venous return and decreased LV volume. This maneuver is also abnormal in patients with congestive heart failure. Another useful maneuver in patients with hypertrophic obstructive cardiomyopathy is the introduction of a premature ventricular beat (Brockenbrough maneuver). Premature ventricular contractions normally increase the pulse pressure of the subsequent ventricular beat. In obstructive hypertrophic cardiomyopathy, the outflow gradient is increased during the post-premature beat with a decrease in the pulse pressure of the aortic contour. A premature ventricular beat may also accentuate the spike-and-dome configuration of the aortic pressure waveform.

Rapid volume loading may reveal occult pericardial constriction (see Chap. 75), when atrial and ventricular filling pressures are relatively normal under baseline conditions as a result of hypovolemia, and can help distinguish pericardial constriction from myocardial restriction. The Kussmaul sign occurs in pericardial constriction. With inspiration, it is demonstrated when mean right atrial pressure fails to decrease or actually increases in relation to impaired RV filling. The ratio of RV to LV systolic pressure-time area during inspiration compared with expiration is called the systolic area index.⁵⁰ This is a measure of enhanced ventricular interdependence⁵¹ (Fig. 20-15). The index is significantly higher in those with proven constrictive pericarditis compared with restrictive cardiomyopathy (1.4 ± 0.2 versus 0.92 ± 0.019 ; $P < 0.0001$), with a sensitivity of 97% and predicted accuracy of 100% for identification of constriction.

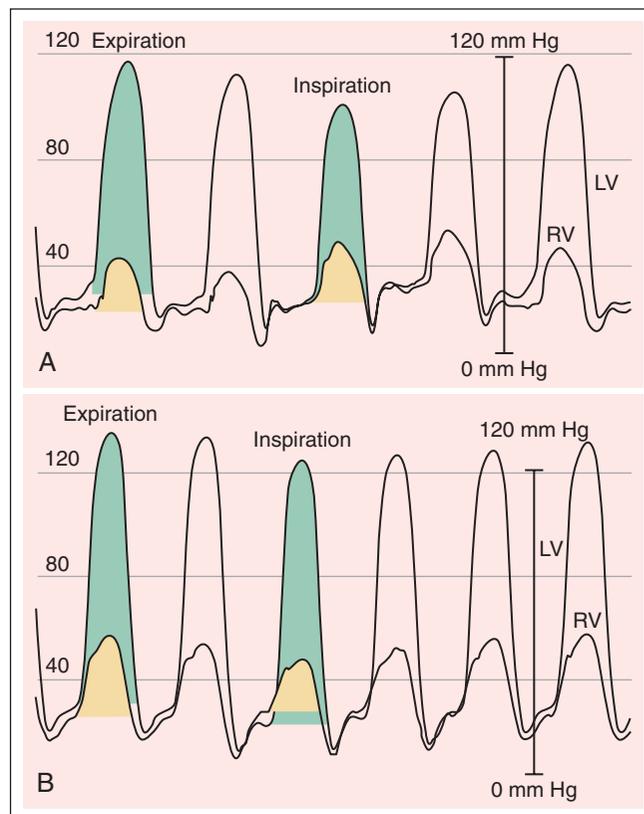


FIGURE 20-15 Left ventricular (LV) and right ventricular (RV) high-fidelity manometer pressure traces from two patients during expiration and inspiration. Both patients have early rapid filling and elevation and end-equalization of the LV and RV pressures at end-expiration. **A**, A patient with surgically documented constrictive pericarditis. During inspiration, there is an increase in the area of the RV pressure curve (yellow shaded area) compared with expiration. The area of the LV pressure curve (green shaded area) decreases during inspiration compared with expiration. **B**, A patient with restrictive myocardial disease documented by endomyocardial biopsy. During inspiration, there is a decrease in the area of the RV pressure curve (yellow shaded area) compared with expiration. The area of the LV pressure curve (green shaded area) is unchanged during inspiration compared with expiration.

Pharmacologic Maneuvers

Dobutamine infusion during cardiac catheterization is indicated in patients with low-flow, low-gradient aortic stenosis (see Chaps. 15 and 66).^{2,46} In patients with a mean gradient below 30 mm Hg, low cardiac output, and low ejection fraction ($<40\%$), the Gorlin formula may not reflect the true valve area. Provocation with dobutamine infusion can assist in distinguishing intrinsic contractile dysfunction versus after-load mismatch from valvular stenosis. Up to one third of patients with low-output severe aortic stenosis as calculated by the Gorlin formula may have pseudosevere aortic stenosis.⁵²

Resting hemodynamics including transvalvular gradient, cardiac output, and aortic valve area should be determined. Dobutamine is infused at $5 \mu\text{g}/\text{kg}/\text{min}$ and increased by 3 to $10 \mu\text{g}/\text{kg}/\text{min}$ every 5 minutes with a maximum of $40 \mu\text{g}/\text{kg}/\text{min}$, mean gradient above 40 mm Hg, 50% increase in the cardiac output, or heart rate above 140 beats per minute. Patients with a final aortic valve area smaller than 1.2 cm^2 and mean gradient above 30 mm Hg are considered to have severe aortic stenosis.⁴⁴

Nitric oxide is an endothelium-derived vasodilator with selective pulmonary vasodilator properties that is useful in evaluating patients with pulmonary hypertension (see Chap. 78). Inhaled nitric oxide is rapidly inactivated, in contrast to intravenous vasodilators, which can cause severe systemic hypotension.⁵³ It has been well established that

lowering of pulmonary artery pressure with vasodilators predicts a favorable clinical outcome.

Inhaled nitric oxide can safely and effectively assess the capacity of a patient for pulmonary vasodilator response without causing systemic hypotension. It can accurately predict a response to subsequent medical therapy.⁵⁴ Doses of 10, 20, 40, or 80 parts per million can be tested during 5- to 10-minute intervals with serial sampling of mean pulmonary artery pressure and calculation of PVR and cardiac output. The definition of an acute response that may warrant initiation of long-term therapy with oral calcium channel blockers is a decrease in the mean pulmonary artery pressure of at least 10 mm Hg to an absolute mean pulmonary artery pressure of less than 40 mm Hg without a decrease in cardiac output.

Sodium nitroprusside infusion may improve the cardiac output and filling pressures in patients with dilated cardiomyopathies and in patients with mitral regurgitation by lowering SVR and PVR. A favorable response to sodium nitroprusside infusion may predict a good clinical outcome.

Agents that increase SVR, such as phenylephrine, reduce the gradient in obstructive hypertrophic cardiomyopathy. This can be used to improve acute systemic hypertension in patients with hypertrophic cardiomyopathy (see Chap. 69).

Isoproterenol infusion may be used to simulate supine dynamic exercise, although untoward side effects limit its applicability. This drug's positive inotropic and chronotropic effects can increase the gradient in obstructive hypertrophic cardiomyopathy and mitral stenosis. Nitroglycerin and amyl nitrate decrease preload and accentuate the systolic gradient in patients with obstructive hypertrophic cardiomyopathy. Amyl nitrate is generally inhaled, and its onset and offset of action are rapid.

Adjunctive Diagnostic Techniques

Left Ventricular Electromechanical Mapping

Advances in catheter design and navigational technology have resulted in catheter-based three-dimensional mapping systems for evaluation of regional and global LV function. The system provides simultaneous electrical, mechanical, and anatomic information.⁵⁵

Electromechanical LV maps can distinguish viable from nonviable myocardium and ischemic from nonischemic myocardium and correlate with thallium uptake.⁵⁶

The mapping system can predict recovery of function after revascularization, providing on-line assessment of viability.⁵⁷ This technique holds promise for guiding local delivery of myocardial regeneration therapies, such as stem cell injection.⁵⁸

Intracardiac Echocardiography

Intracardiac echocardiography (ICE) is used for transvenous imaging within the cardiac chambers. It consists of an 8F or 10F, 90-cm-long catheter that permits two planes of bidirectional steering in the anterior-posterior and left-right direction. The transducer has variable frequencies of 5 to 10 MHz with multiple phased array features, including two-dimensional imaging and color and spectral Doppler analysis.

ICE provides imaging of interatrial or interventricular septum and left-heart structures from either the right atrium or ventricle, with penetration up to 15 cm. Applications include guidance of percutaneous atrial septal defect and patent foramen ovale closures, thus mitigating the need for transesophageal echocardiography and anesthesia (Fig. 20-16). In patients requiring transseptal puncture, ICE can facilitate

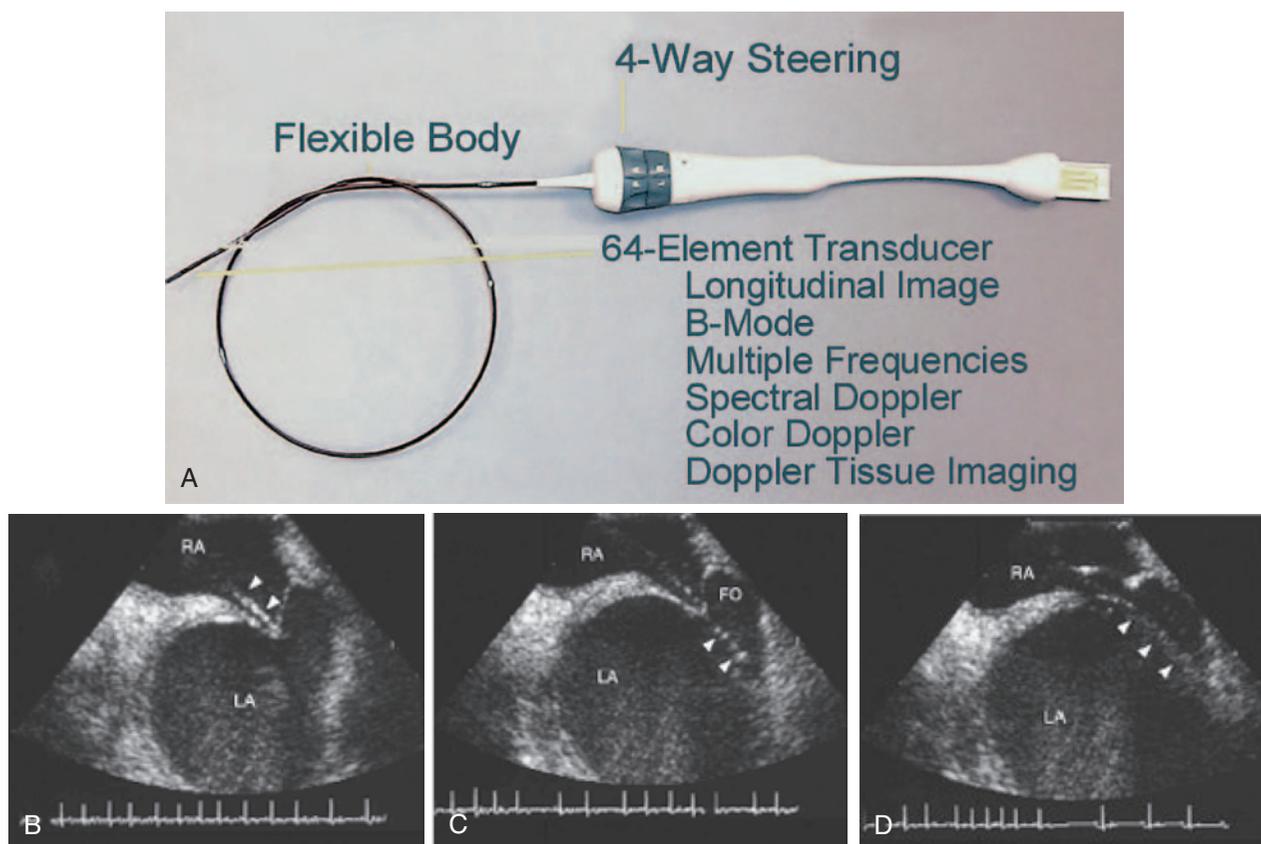


FIGURE 20-16 **A**, Intracardiac echocardiography disposable transducer (Acuson, Inc.), with steering apparatus on proximal end and flexible body with transducer on distal tip of catheter. **B**, Tenting of the membranous fossa by the dilator-needle assembly. The transseptal needle assembly (arrowheads) is advanced to indent the fossa membrane. **C**, Advancement of the transseptal needle across the membranous fossa. Here the needle (arrowheads) is seen near the posterosuperior left atrial wall. The membrane remains tented because the dilator has not yet crossed the septum. **D**, Dilator and sheath passage across the interatrial septum. The dilator and sheath assembly has now advanced into the left atrium, releasing the tenting of the membranous fossa. FO = fossa ovalis; LA = left atrium; RA = right atrium. (From Johnson SB, Seward JB, Packer DL: Phased-array intracardiac echocardiography for guiding transseptal catheter placement: Utility and learning curve. *Pacing Clin Electrophysiol* 25:402, 2002.)



**TABLE 20-5 Risk of Cardiac Catheterization and Coronary Angiography**

COMPLICATION	RISK (%)*
Mortality	0.11
Myocardial infarction	0.05
Cerebrovascular accident	0.07
Arrhythmia	0.38
Vascular complications	0.43
Contrast reaction	0.37
Hemodynamic complications	0.26
Perforation of heart chamber	0.03
Other complications	0.28
Total of major complications	1.70

*No. of patients = 59,792.

From Scanlon PJ, Faxon DP, Audet AM, et al: ACC/AHA guidelines for coronary angiography: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Coronary Angiography). *J Am Coll Cardiol* 33:1760, 1999.

localization of the fossa ovalis. ICE is also used to guide electrophysiologic procedures with identification of anatomic structures difficult to view by fluoroscopy (e.g., pulmonary veins or fossa ovalis for transseptal puncture).²⁶

Complications Associated with Cardiac Catheterization

Cardiac catheterization is a relatively safe procedure but has a well-defined risk of morbidity and mortality (**Table 20-5**). The potential risk of major complications during cardiac catheterization is often related to comorbid disease. The use of low-osmolar and isosmolar contrast media, lower profile diagnostic catheters, and reduced anticoagulation and extensive operator experience have reduced the incidence of complications. Several large studies provide insight into the incidence of major events and delineate cohorts of patients that are at increased risk.^{59,60}

Death related to diagnostic cardiac catheterization occurs in 0.08% to 0.75% of patients, depending on the population studied. Data from the Society for Cardiac Angiography identified subsets of patients with an increased mortality rate.⁶⁰ In an analysis of 58,332 patients, multivariate predictors of significant complications were moribund status, advanced New York Heart Association functional class, hypotension, shock, aortic valve disease, renal insufficiency, unstable angina, mitral valve disease, acute myocardial infarction within 24 hours, congestive heart failure, and cardiomyopathy. The risk of any complication during cardiac catheterization is further increased in octogenarians. Although the overall mortality is approximately 0.8% in this cohort, the risk of nonfatal major complications, which are primarily peripheral vascular, is approximately 5%.

The risk of myocardial infarction varies from 0.03% to 0.06%; of significant bradyarrhythmias or tachyarrhythmias, from 0.56% to 1.3%⁶⁰; and of neurologic complications, from 0.03% to 0.2%.⁵⁶ One study using serial cranial magnetic resonance imaging demonstrated a 22% incidence of focal acute cerebral embolic events after retrograde crossing of stenotic aortic valves, and 3% of patients demonstrated clinically apparent neurologic deficits.⁶¹ However, this study is in contradistinction to previously published large clinical series and requires additional validation.

Stroke can be periprocedural in the laboratory or occur within a few hours after the procedure. Whether the mechanism is different is unclear. Stroke should be distinguished from other conditions, including seizure, migraine, hypoglycemia, and encephalopathy. The standard

stroke management with a multidisciplinary team is important to improve prognosis. Predictors of stroke include diabetes mellitus, hypertension, prior stroke, and renal failure. The procedure length, contrast volume, urgent indications, and use of intra-aortic balloon pumps are known to increase the risk of stroke.⁶²

The most common complication is arterial access site bleeding, which is usually manifested by minor oozing or small hematomas. The incidence of major vascular complications in most series has suggested a slightly higher frequency when the Sones brachial approach is used. The incidence of major vascular complications has decreased during the last decade and is currently reported as approximately 0.20%.^{60,63} Major vascular complications include occlusion requiring arterial repair or thrombectomy, retroperitoneal bleeding, hematoma formation, pseudoaneurysm, arteriovenous fistula formation, and infection. In a patient with unexplained hypotension or back pain, retroperitoneal hematoma should be suspected. Evaluation should include serial complete blood count determinations, evaluation of anticoagulation status, and either CT or ultrasound evaluation of the groin, pelvis, and abdomen. The risk of requiring surgical repair for vascular injury is related to advanced age, congestive heart failure, and larger body surface area. With ultrasound guidance, many pseudoaneurysms can be successfully treated percutaneously with directed infusion of thrombin, and surgical repair can often be avoided.

The proper management of the arterial sheath is important in avoiding complications. Because dwell times correlate with hematoma formation, all sheaths should be removed as soon as possible with an activated clotting time below 170. Frequent blood pressure and pulse monitoring is essential.

Systemic complications can vary from mild vasovagal responses to severe vagal reactions that lead to prolonged hypotension. Minor complications occur in approximately 4% of patients undergoing routine cardiac catheterization.⁶³ The most common untoward effects are transient hypotension and brief episodes of angina lasting less than 10 minutes.

Hives can occur but are less commonly observed with low-osmolar contrast agents and with intra-arterial administration. They are readily treated with intravenous corticosteroids and diphenhydramine. Rarely, anaphylactoid complications are observed. These are also treated with intravenous corticosteroid and diphenhydramine. Epinephrine is administered in severe reactions; the dilution of 0.1 mg/mL is administered at 1.4 µg/min during 5 minutes.

The most common complications of right-heart catheterization are nonsustained atrial and ventricular arrhythmias. Major complications associated with right-heart catheterization are infrequent. These include pulmonary infarction, pulmonary artery or right ventricle perforation, and infection.

Future Perspectives

In many ways, diagnostic cardiac catheterization is at a crossroads. High-resolution coronary CT angiography will likely replace the need for cardiac catheterization in low-risk patients in whom coronary artery disease can be excluded with noninvasive testing. On the other hand, the widespread availability of multiple noninvasive screening modalities will provide early detection of cardiovascular disease that will require cardiac catheterization to precisely define the extent and severity of coronary, vascular, and valvular disease. Transcatheter imaging, diagnostic technologies, and provocative testing expand the catheterization laboratory beyond conventional radiographic imaging and hemodynamic assessment.

The expansion of transcatheter repair of coronary, valvular, and structural heart disease will also stimulate the growth of cardiac catheterization in conjunction with the percutaneous therapeutic procedures (see **Chaps. 58 and 59**). Safety of the technique is well established and continues to improve with experience, standardized quality assurance programs, and advances in imaging technology.

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