Grossman's Cardiac Catheterization, Angiography, and Intervention

SIXTH EDITION

DONALD S. BAIM
WILLIAM GROSSMAN

LIPPINCOTT WILLIAMS & WILKINS

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Sixth Edition

Edited by

Donald S. Baim, M.D.
Professor of Medicine
Harvard Medical School;
Director, Center for Innovative Minimally Invasive Therapy
Brigham and Women's Hospital
Boston, Massachusetts

William Grossman, M.D.
Meyer Friedman Distinguished Professor of Medicine
University of California, San Francisco, School of Medicine;
Chief, Division of Cardiology
University of California, San Francisco Medical Center
San Francisco, California

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Historical Perspective and Present Practice of Cardiac Catheterization

William Grossman

University of California, San Francisco, School of Medicine; Division of Cardiology, University of California, San Francisco Medical Center, San Francisco, California 94143

It is difficult to imagine what our concepts of heart disease might be like today if we had to construct them without the enormous reservoir of physiologic and anatomic knowledge derived from the past 60 years' experience in the cardiac catheterization laboratory. As Andre Cournand remarked in his Nobel lecture of December 11, 1956, "the cardiac catheter was... the key in the lock" (1). By turning this key, Cournand and his colleagues led us into a new era in the understanding of normal and disordered cardiac function in humans.

According to Cournand (2), cardiac catheterization was first performed (and so named) by Claude Bernard in 1844. The subject was a horse, and both the right and left ventricles were entered by a retrograde approach from the jugular vein and carotid artery. In an excellent review of the history of cardiac catheterization, angiography, and interventional cardiology, Mueller and Sanborn (3) describe and cite references for experiments by Stephen Hales and others whose work antedates that of Claude Bernard, and the interested reader is referred to their review for details (3). Although he may not have been the first to perform cardiac catheterization, Claude Bernard's careful application of scientific method to the study of cardiac physiology using the cardiac catheter demonstrated the enormous value of this technical innovation. An era of investigation of cardiovascular physiology in animals then followed, resulting in the development of many important techniques and principles (e.g., pressure manometry, the Fick cardiac output method) which awaited direct application to the patient with heart disease.

Werner Forssmann usually is credited with being the first person to pass a catheter into the heart of a living person—himself (4). At age 25, while receiving clinical instruction in surgery at Eberswalde, near Berlin, he passed a catheter 65 cm through one of his left antecubital veins, guiding it by fluoroscopy until it entered his right atrium. He then walked to the radiology department (which was on a different level, requiring that he climb stairs), where the catheter position was documented by a chest roentgenogram (Fig. 1.1). During the next 2 years, Forssmann continued to perform catheterization studies, including six additional attempts to catheterize himself. Bitter criticism, based on an unsubstantiated belief in the danger of his experiments, caused Forssmann to turn his attention to other concerns, and he eventually pursued a career as a urologist (5). Nevertheless, for his contribution and foresight he shared the Nobel Prize in Medicine with Andre Cournand and Dickinson Richards in 1956.

Forssmann's primary goal in his catheterization studies was to develop a therapeutic technique for the direct delivery of drugs into the heart. He wrote (4):

> If cardiac action ceases suddenly, as is seen in acute shock or in heart disease, or during anesthesia or poisoning, one is forced to deliver drugs locally. In such cases the intracardiac in-

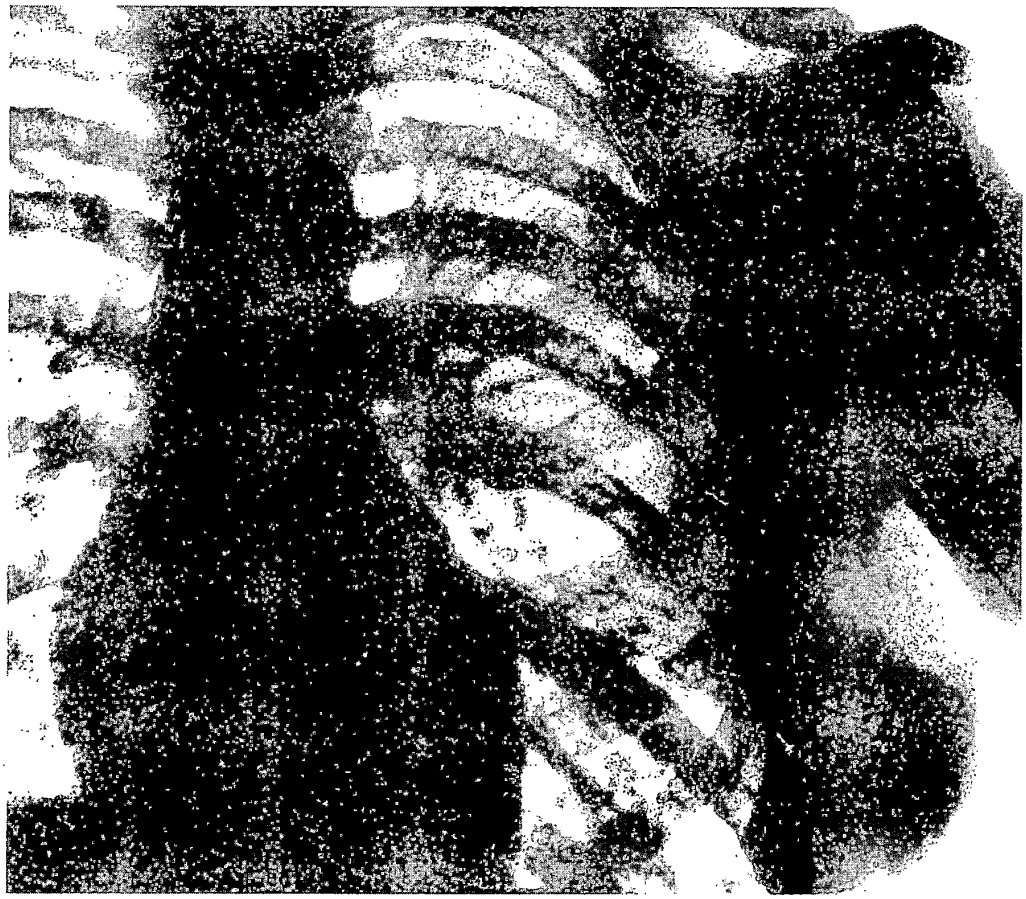

FIG. 1.1. The first documented cardiac catheterization. At age 25, while receiving clinical instruction in surgery at Eberswalde, Werner Forssmann passed a catheter 65 cm through one of his left antecubital veins until its tip entered the right atrium. He than walked to the radiology department where this roentgenogram was taken. (Forssmann W. Die Sondierung des rechten Herzens. *Klin Wochenschr* 1929;8:2085, with permission of Springer-Verlag, Berlin.)

> jection of drugs may be life saving. However, this may be a dangerous procedure because of many incidents of laceration of coronary arteries and their branches leading to cardiac tamponade, and death. . . . Because of such incidents, one often waits until the very last moment and valuable time is wasted. Therefore I started to look for a new way to approach the heart, and I catheterized the right side of the heart through the venous system.

Others appreciated the potential of Forssmann's technique as a diagnostic tool. In 1930, Klein reported 11 right-sided heart catheterizations, including passage to the right ventricle and measurement of cardiac output using the Fick principle (6). The cardiac outputs were 4.5 and 5.6 L/min in two patients without heart disease. In 1932, Padillo and coworkers reported right heart catheterization and measurement of cardiac output in two subjects (2). Except for these few studies, application of cardiac catheterization to study the circulation in normal and disease states was fragmentary until the work of Andre Cournand and Dickinson Richards, who separately and in collaboration produced a remarkable series of investigations of right heart physiology in humans (7–9). In 1947, Dexter reported his studies on congenital heart disease (10). He went further than his predecessors by passing the catheter to the distal pulmonary artery, observing "the oxygen saturation and

source of pulmonary capillary blood'' obtained from the pulmonary artery ''wedge'' position (10). Subsequent studies from Dexter's laboratory (11) and by Werkö (12) elaborated on this pulmonary artery wedge position, and pressure measured at this position was reported to be a good estimate of pulmonary venous and left atrial pressure. During this exciting early period, catheterization was used to investigate problems in cardiovascular physiology by McMichael in England (13), Lenègre in Paris (14), and Warren, Stead, Bing, Dexter, Cournand, and others in the United States (15–23).

Further developments came rapidly, and highlights include the following:

Retrograde left-sided heart catheterization was first reported by Zimmerman (24) and by Limon-Lason (25) in 1950.

The percutaneous technique developed by Seldinger in 1953 was soon applied to cardiac catheterization of both the left and right heart chambers (26).

Transseptal catheterization was first developed in 1959 by Ross (27) and Cope (28) and quickly became accepted as a standard technique.

Selective coronary arteriography was reported by Sones in 1959 and was perfected to a remarkable excellence over the ensuing years (29,30). This technique was modified for a percutaneous approach by Ricketts and Abrams (31) in 1962 and Judkins (32) in 1967.

In 1970, Swan and Ganz introduced a practical balloon-tipped, flow-guided catheter technique that enabled the application of catheterization outside the laboratory (33).

INTERVENTIONAL CARDIOLOGY

In the last 25 years, investigators have focused once again on the therapeutic potential of the cardiac catheter. In 1977, Grüntzig introduced the technique of percutaneous transluminal coronary angioplasty (PTCA) (34,35). In the ensuing years, catheter-based coronary revascularization has been applied widely. With rapidly evolving technology and expanding indications, PTCA and its ''offspring'' (e.g., stents, atherectomy) first rivaled and have now surpassed coronary bypass surgery as the dominant therapeutic modality for coronary artery disease. The development of percutaneous coronary intervention stimulated other innovations such as balloon valvuloplasty and devices to close intracardiac shunts, which together have made ''interventional cardiology'' a new field in cardiovascular medicine. The history of interventional cardiology has been summarized by Spencer King in an excellent review (36), and the interested reader is referred there for further details. In a sense, cardiac catheterization has returned to its roots, because, as mentioned earlier, Werner Forssmann's original intention had been to use the catheter as a tool for therapy, not diagnosis.

At approximately the same time that Grüntzig was developing balloon angioplasty in Zurich, investigators in Germany and Los Angeles were administering the thrombolytic agent streptokinase through catheters placed selectively in the coronary arteries of patients early in the acute phase of transmural myocardial infarction. This new catheter-based therapy, which was viewed as radical at the time, produced angiographic findings that confirmed beyond any doubt the role of acute coronary thrombosis in the genesis of myocardial infarction. When investigators found that similar therapeutic benefit could be achieved by intravenous administration of the thrombolytic agent, the intracoronary direct-infusion technique all but died out except for a few special indications. However, catheter-based therapy for acute coronary thrombosis has undergone a renaissance in the last 10 years with the demonstration that PTCA/stenting in this setting produces results that are comparable or superior to those achieved with thrombolytic therapy (37,38).

It is clear as we enter the 21st century that interventional cardiology—by virtue of its new technologies, potent adjunctive drug therapies (e.g., blockers of the platelet IIb/IIIa receptor), expanding indications, and improving results—has blossomed. In many ways, interventional cardiology, rather than purely diagnostic techniques, has become the dominant discipline within the broad field of cardiac catheterization.

Although the emphasis in the field (and in this textbook) is now appropriately on the dynamic field of catheter-based intervention, the basic principles of catheter insertion, hemodynamic measurement, high-quality angiography, and integration of catheterization findings with both the clinical scenario and the findings of noninvasive tests are not just historical curiosities: they are the foundations on which all current interventional techniques are built, and from which future evolution of cardiac catheterization will proceed.

INDICATIONS FOR CARDIAC CATHETERIZATION

As performed today, cardiac catheterization is a combined hemodynamic and angiographic procedure undertaken for diagnostic and often therapeutic purposes. As with any invasive procedure, the decision to perform cardiac catheterization must be based on a careful balance of the risk of the procedure against the anticipated benefit to the patient. A summary of the indications for cardiac catheterization is given in Table 1.1 and discussed in the following paragraphs.

Cardiac catheterization usually is recommended to confirm the presence of a clinically suspected condition, define its anatomic and physiologic severity, and determine the presence or absence of associated conditions when a therapeutic intervention is planned in a symptomatic patient. The most common indication for cardiac catheterization today arises in the patient with an acute coronary ischemic syndrome in whom an invasive therapeutic intervention (PTCA, stent, or coronary artery bypass graft surgery) is contemplated. The patient with an acute coronary ischemic syndrome has most commonly experienced recent rupture of an atherosclerotic plaque within a coronary artery. Exposure of plaque contents to flowing blood leads to platelet deposition and coronary thrombosis, which, in turn, leads to transmural ischemia if the thrombus is completely obstructing, or to unstable angina if it causes only partial or intermittent occlusion. The goal of cardiac catheterization in such patients is to identify the culprit artery and restore vessel patency by PTCA/stent placement. The diagnostic part of the catheterization procedure may reveal other features (e.g., multivessel or left main coronary artery disease, severe associated valvular disease) that provide critical information for the decision to proceed with open-heart surgery.

Is cardiac catheterization necessary in all patients being considered for cardiac surgery? Al-

TABLE 1.1. *Indications for cardiac catheterization*

I. Need to choose among therapeutic options
  A. Revascularization (catheter-based or coronary artery bypass grafting) versus continued medical therapy—Perform coronary angiography and (in selected patients) left ventriculography in patients with
    1. Acute coronary syndrome
    2. Incapacitating or progressive angina
    3. Coronary artery disease with high-risk stress test
  B. Valve replacement/repair versus continued medical therapy—Assess coronary anatomy, and associated conditions (e.g., pulmonary hypertension) that may increase risk in patients with
    1. Aortic stenosis with angina, syncope, and/or heart failure
    2. Aortic, mitral, or tricuspid regurgitation with increasing congestive heart failure or evidence of decreasing ventricular function
    3. Mitral, pulmonic, or tricuspid stenosis and progressive symptoms who are candidates for balloon valvuloplasty
  C. Anticoagulation, thrombolytic therapy versus none—in patients with suspected acute pulmonary embolism if lung scan is nondiagnostic or if there is acute congestive heart failure or hypotension
  D. Repair of congenital heart or vascular defect—Assess anatomy and physiology in preparation for catheter-based therapy (e.g., atrial septal plug, balloon valvuloplasty) or whenever uncertainties remain despite thorough nonhuman research
II. Obscure or confusing clinical picture, even when there is no immediate therapeutic question
  A. Coronary angiography in patients with chest pain of uncertain origin
  B. Endomyocardial biopsy in patients with idiopathic cardiomyopathy
III. Research studies—Cardiac catheterization for pure research purposes (e.g., follow-up coronary angiography to assess regression of atherosclerosis after lipid-lowering therapy or collateral development after treatment with growth factor) should be done only if the patient has given informed consent and there is a protocol that has been approved by the institution's Committee on Human Research.

though few would disagree that consideration of heart surgery is an adequate reason for the performance of catheterization, clinicians differ about whether all patients being considered for heart surgery should undergo preoperative cardiac catheterization. In this regard, it should be emphasized that the risks of catheterization are small compared with those of embarking upon cardiac surgery in a patient for whom an incorrect clinical diagnosis or the presence of an unsuspected additional condition greatly prolongs and complicates the planned surgical approach. The operating room is not a good place for surprises; by providing the surgical team with a precise and complete road map of the course ahead, cardiac catheterization can permit a carefully reasoned and maximally efficient operative procedure. Furthermore, information obtained by cardiac catheterization may be invaluable in the assessment of crucial determinants of prognosis, such as left ventricular function, status of the pulmonary vasculature, and patency of the coronary arteries. For these reasons, my colleagues and I recommend cardiac catheterization for almost all patients for whom heart surgery is contemplated.

Other major therapeutic considerations besides heart surgery may depend on the information afforded by cardiac catheterization and angiography. For example, the decision for pharmacologic intervention with heparin and/or a thrombolytic agent in suspected acute pulmonary embolism, or with high doses of a β-blocker and/or calcium antagonists in suspected hypertrophic subaortic stenosis, might well be considered of sufficient magnitude to warrant confirmation of the diagnoses by angiographic and hemodynamic investigation before the initiation of therapy. A clinical diagnosis of primary pulmonary hypertension made by echocardiography usually requires cardiac catheterization (a) to confirm the diagnosis and (b) to assess potential responsiveness to pharmacologic agents, such as epoprostenol (39).

A second broad indication for performing cardiac catheterization is diagnosis of obscure or confusing problems in heart disease, even when a major therapeutic decision is not imminent. A common instance of this indication is presented by the patient with chest pain of uncertain cause, about whom there is confusion regarding the presence of obstructive coronary artery disease. Both management and prognosis of this difficult problem are greatly simplified when it is known, for example, that the coronary arteries are widely patent. Another example within this category might be the symptomatic patient with a suspected diagnosis of cardiomyopathy. Although some may feel satisfied with a clinical diagnosis of this condition, the implications of such a diagnosis in terms of prognosis and therapy (e.g., long-term bed rest, chronic anticoagulant therapy) are so important that I believe it is worthwhile to be aggressive in ruling out potentially correctable conditions (e.g., hemochromatosis, pericardial effusive-constrictive disease) with certainty, even though the likelihood of their presence may appear to be remote on clinical grounds.

Research

On occasion, cardiac catheterization is performed primarily as a research procedure. Although research is conducted to some degree in many of the diagnostic and therapeutic studies performed at major medical centers, this is quite different from catheterization for the sole purpose of a research investigation. Such studies should be carried out only under the direct supervision of an experienced investigator who is an expert in cardiac catheterization, using a protocol that has been carefully scrutinized and approved by the Committee on Human Research at the investigator's institution, and after a thorough explanation has been made to the patient detailing the risks of the procedure and the fact that the purpose of the investigation is to gather research information.

Contraindications

If it is important to carefully consider the indications for cardiac catheterization in each patient, it is equally important to discover any contraindications. Over the years, the concept of contraindications has been modified by the fact that patients with acute myocardial infarction,

TABLE 1.2. *Relative contraindications to cardiac catheterization and angiography*

1. Uncontrolled ventricular irritability: the risk of ventricular tachycardia/fibrillation during catheterization is increased if ventricular irritability is uncontrolled
2. Uncorrected hypokalemia or digitalis toxicity
3. Uncorrected hypertension: predisposes to myocardial ischemia and/or heart failure during angiography
4. Intercurrent febrile illness
5. Decompensated heart failure: especially acute pulmonary edema, unless catheterization can be done with the patient sitting up
6. Anticoagulated state: prothrombin time longer than 18 seconds
7. Severe allergy to radiographic contrast agent
8. Severe renal insufficiency and/or anuria: unless dialysis is planned to remove fluid and radiographic contrast load

cardiogenic shock, intractable ventricular tachycardia, and other extreme conditions have tolerated catheterization and coronary angiography surprisingly well. At present the only absolute contraindication to cardiac catheterization is the refusal of a mentally competent patient to consent to the procedure.

A long list of relative contraindications must be kept in mind, however, and these include all intercurrent conditions that can be corrected and whose correction would improve the safety of the procedure. Table 1.2 lists these relative contraindications. For example, ventricular irritability can increase the risk and difficulty of left heart catheterization and can greatly interfere with interpretation of ventriculography (see Chapter 12); if possible, ventricular irritability should be suppressed medically before or during catheterization. Hypertension increases predisposition to ischemia and/or pulmonary edema and should be controlled before and during catheterization. Other conditions that should be controlled before elective catheterization include intercurrent febrile illness, decompensated left-sided heart failure, correctable anemia, digitalis toxicity, and hypokalemia. Allergy to a radiographic contrast agent is a relative contraindication to cardiac angiography, but proper premedication can substantially reduce the risks of a major adverse reaction, as discussed in Chapter 3.

Anticoagulant therapy is more controversial as a contraindication. As pointed out in Chapters 4 and 11, heparin may lower the incidence of thromboembolic complications during coronary angiography (40). It is important to distinguish anticoagulation with oral anticoagulants (e.g., warfarin) from that with heparin. Heparin anticoagulation can be reversed rapidly during catheterization if necessary (e.g., in the case of perforation of the heart or great vessels or uncontrolled bleeding from femoral or brachial sites). Reversal of the prolonged prothrombin time of oral anticoagulation before or during cardiac catheterization represents a more complex problem. I strongly oppose acute reversal of oral anticoagulation with parenteral vitamin K because of the occasional induction of a hypercoagulable state, which has been known to result in thrombosis of prosthetic valves or thrombus formation within cardiac chambers, arteries, or veins. If reversal of oral anticoagulation is required, we favor administration of fresh-frozen plasma. For patients chronically anticoagulated with an oral agent, we routinely recommend discontinuation of the oral anticoagulants 48 hours before cardiac catheterization, with heparin given during these 48 hours for patients who have a strong indication for continuous anticoagulation (e.g., mechanical cardiac valve prosthesis). I prefer to have the international normalized ratio (INR) less than 2.0, or the prothrombin time less than 18 seconds, and no heparin administration for 4 hours before the catheterization. If anticoagulant therapy cannot be interrupted at all, we prefer heparin for the reasons just mentioned.

FACTORS INFLUENCING CHOICE OF APPROACH

Of the various approaches to cardiac catheterization, certain ones have only historical interest (transbronchial approach, posterior transthoracic left atrial puncture, and suprasternal puncture of the left atrium). In this book only the following are discussed in detail: (a) catheterization by percutaneous approach from various sites (including femoral or radial arteries, transseptal catheterization, and apical left ven-

tricular puncture) and (b) catheterization by direct surgical exposure of the brachial artery and vein.

The great vessels and all cardiac chambers can be entered in almost all cases by either the direct exposure or percutaneous approaches (or a combination of both). Each method has its advantages and disadvantages, its adherents and detractors. In reality, the methods are not mutually exclusive but rather complementary; ideally, the physician performing cardiac catheterization should be well versed in both methods.

Advantages of the Percutaneous Femoral Approach

The percutaneous femoral approach is clearly the dominant technique in cardiac catheterization today, presenting a broad set of advantages and indications. The femoral approach does not require arteriotomy and arterial repair and can be performed repeatedly in the same patient at intervals, whereas the brachial arteriotomy approach can rarely be repeated safely more than two or three times; infection and thrombophlebitis at the catheterization site are rare; surgical (suture) closure of the skin is not necessary; and the approach is readily adaptable to a variety of other entry vessels (e.g., internal jugular vein, axillary artery, radial artery). Larger-caliber devices (i.e., valvuloplasty balloons or intraaortic counterpulsation catheters) can be introduced into the femoral artery but not usually into the smaller brachial artery. The femoral approach is clearly the method of choice in a patient with absent or diminished radial and brachial pulsations or when the direct brachial approach has been unsuccessful. In the occasional patient with tight aortic stenosis in whom retrograde catheterization has proved impossible, percutaneous transseptal catheterization of the left atrium and ventricle is helpful; in the rare instance in which retrograde arterial and transseptal catheterization have not succeeded in gaining entry into the left ventricle (or are contraindicated by the presence of disc mitral and/or aortic prostheses or left atrial thrombus), percutaneous transthoracic puncture of the left ventricle may be considered (see Chapter 4).

Advantages of the Percutaneous Radial Approach

In recent years, percutaneous technique using the radial, brachial, or ulnar arteries as entry sites (41–46) has been applied to retrograde left heart catheterization, coronary angiography, PTCA, and even stent placement. This approach is becoming increasingly popular and has been demonstrated to have advantages of cost and patient comfort. A study by Mann and colleagues (41) reported on the use of percutaneous transradial catheterization for stent placement in patients with acute coronary syndromes. A total of 144 patients with acute coronary syndromes were randomly assigned to either a femoral or a radial approach. Stenting from the radial approach allowed earlier hospital discharge and was associated with decreased hospital charges and fewer bleeding complications. Not all patients assigned to the radial approach strategy were able to have radial artery catheterization. Six of the 74 had a negative Allen test or Doppler examination, or both, suggesting an incomplete palmar arch, so that radial catheterization was thought to be contraindicated; accordingly, they were included in the femoral approach group. In three additional patients, the radial artery was not successfully cannulated, and these patients also crossed over to the femoral approach group.

Advantages of the Brachial Approach

Much less common today, the direct exposure approach usually utilizes cutdown on the brachial artery and basilic vein at the elbow (see Chapter 5). In general, the percutaneous radial approach has all the advantages of the direct brachial approach and few of its disadvantages. Nevertheless, the brachial cutdown approach is still used by a few centers and is worthy of some comment. The brachial approach may have advantages in a patient with severe peripheral vascular disease involving the abdominal aorta, iliac, or femoral arteries; suspected femoral vein or inferior vena caval thrombosis; or coarctation of the aorta. The brachial or radial approach may also have advantages in the very obese patient, in whom the percutaneous femoral technique

may be technically difficult and where hard to control breathing occurs after catheter removal. Another advantage occasionally cited for the direct brachial approach is use of a single left heart catheter (Sones catheter) for left ventriculography and coronary angiography.

DESIGN OF THE CATHETERIZATION PROTOCOL

Every cardiac catheterization should have a protocol—a carefully reasoned, sequential plan designed specifically for the individual patient. Although this protocol may exist only in the mind of the operator, it is often helpful to prepare a written protocol and post it in the catheterization suite so that all personnel in the laboratory understand exactly what is planned and can anticipate the needs of the operator.

Certain general principles should be considered in the design of a protocol. First, hemodynamic measurements should precede angiographic studies whenever possible, so that crucial pressure and flow measurements may be made as close as possible to the basal state. A separate arterial monitor line (which may be just the sidearm of the arterial sheath) can be helpful; when complications develop (and they do, no matter how skilled the operator), this second transducer allows continuous monitoring of arterial pressure. Second, pressures and selected oxygen saturation values should be measured and recorded in each chamber "on the way in," that is, immediately after the catheter enters and before it is directed toward the next chamber. If problems should develop during the later stages of a catheterization procedure (atrial fibrillation or other arrhythmia, pyrogen reaction, hypotension, or reaction to contrast material), the investigator will be glad to have measured pressures and saturations this way, rather than waiting until the time of catheter pullback. Third, measurements of pressure and cardiac output should be made as simultaneously as possible. A simple routine for recording pressure during the cardiac output measurement can be learned by the laboratory personnel and performed efficiently in every case.

Beyond these general guidelines, the protocol reflects individual patient differences. With regard to angiography, it is important to keep in mind Sutton's law (when asked why he robbed banks, Willie Sutton is reported to have replied, "Because that's where the money is") and order the contrast injections in relation to the most important diagnostic considerations in a given patient.

PREPARATION AND PREMEDICATION OF THE PATIENT

It goes without saying that the emotional as well as the "medical" preparation of the patient for cardiac catheterization is the responsibility of the operator. It is our firm obligation to fully explain the proposed procedure in such terms that the patient can give truly informed consent. We always tell the patient and his or her family that there is risk involved and the extent of the risk, depending on the specific procedure and the patient's clinical situation. If appropriate, we reassure patient and family that we do not anticipate any special problems. Our consent form lists the specific risks and informs the patient that "There is a less than 1% risk of serious complications (stroke, heart attack, or death)." If the patient and family want to know more about these risks, they will ask for details. We do not understate the discomfort involved or the duration of the procedure—doing so risks one's credibility. We have been satisfied with this overall approach and can heartily recommend it. A study of psychologic preparation for cardiac catheterization (47) found that patients who received careful psychologic preparation had lower levels of autonomic arousal both during and after cardiac catheterization than did control subjects.

Once the question of indications and contraindications has been dealt with and the patient's consent obtained, attention can be directed toward the matter of medications. As mentioned earlier, we prefer to have the INR less than 2.0 (prothrombin time less than 18 seconds) and no heparin administered for 4 hours. One exception is the patient with unstable angina, in whom a therapeutic heparin infusion may be continued until arterial entry and then supplemented by

3,000 to 5,000 additional units. For these patients, "front-wall" arterial puncture is particularly important, as discussed in Chapter 4. For patients receiving chronic anticoagulation therapy, we discontinue oral anticoagulants the day before hospitalization (or 48 hours before study for outpatient catheterizations), and on admission we begin intravenous heparin, which is stopped within 4 hours of the catheterization. Heparin and oral anticoagulants are reinstituted after the catheterization, and heparin is stopped once adequate prolongation of prothrombin time has been achieved.

The question of administering antibiotics prophylactically is raised occasionally, and some laboratories administer them routinely before catheterization. We do not administer antibiotics prophylactically before cardiac catheterization, and we know of no controlled studies to support their use.

A wide variety of sedatives has been employed for premedication. We no longer routinely order premedication to be given before the patient is sent to the catheterization laboratory. Instead, we assess the patient's state of alertness and need for sedation once he or she is on the catheterization table. At that time, we usually administer midazolam (Versed, Roche Laboratories, Nutley, NJ) 1 mg IV and/or fentanyl 25 to 50 mg IV.

It is our practice to have the patient fasting (except for oral medications) after midnight, but some laboratories allow a light tea and toast breakfast without ill effects. Complete vital signs should be recorded before the patient leaves the floor (for inpatients), or shortly after arrival at the ambulatory center (for outpatients), so that the procedure may be aborted if a change has occurred since the patient was last seen.

For a typical inpatient, our precatheterization orders might be the following:

1. To Cardiac Catheterization Laboratory at 7:30 a.m. tomorrow by stretcher; patient to be in hospital gown.
2. Fasting after midnight except for regularly scheduled oral medications.
3. Have patient urinate before leaving for Cardiac Catheterization Laboratory.
4. Record complete vital signs before patient leaves for Cardiac Catheterization Laboratory.

This list is only a general procedure guide and obviously would be modified as the details of specific situations require.

THE CARDIAC CATHETERIZATION FACILITY

A modern cardiac catheterization laboratory requires an area of 500 to 700 ft^2, within which is housed a conglomeration of highly sophisticated electronic and radiographic equipment. Reports of the Inter-Society Commission for Heart Disease Resources on optimal resources for cardiac catheterization facilities appeared in 1971, 1976, and 1983. The most recent American College of Cardiology/American Heart Association (ACC/AHA) Guidelines for Cardiac Catheterization Laboratories (48) were published in 1991. In this report, a variety of issues were dealt with, including the following:

1. Traditional versus nontraditional settings for a cardiac catheterization laboratory; location within a hospital versus a freestanding facility
2. Ambulatory cardiac catheterization: indications and contraindications
3. Ethical issues related to self-ownership of laboratories, self-referral of patients, and advertising
4. Optimal annual caseload for physicians and for the laboratory
5. Safety issues during conduct of the procedure (e.g., sterile technique, heparin)
6. Physical arrangements and space requirements
7. Radiation safety and radiologic techniques.

The report (48) provides detailed discussion of these issues. Certain points, however, are worth discussing here.

Location Within a Hospital Versus a Freestanding Facility

The issue of whether cardiac catheterization laboratories should be hospital-based, freestand-

ing, or mobile has been the subject of much debate (48–50). There are many potential concerns about performance of cardiac catheterization in a freestanding facility, and the available data from such facilities are limited. Mobile cardiac catheterization laboratories may be either freestanding or hospital-based (as is the case for mobile magnetic resonance imaging and other mobile diagnostic units), so mobile and freestanding units should not be equated automatically. In its 1991 report, the ACC/AHA Task Force "generally found that in freestanding catheterization laboratories, access to emergency hospitalization may be delayed, and appropriate oversight may be lacking. Additionally, opportunities for self-referral may be fostered and the perception of commercialism and entrepreneurial excess in practice created" (48). The report concluded that "in view of the lack of appropriately controlled safety and need data for hospital-based, mobile or freestanding laboratories operating without on-site (accessible by gurney) cardiac surgical facilities, the Task Force reaffirms the position that further development of these facilities cannot be endorsed at this time" (48).

Outpatient Cardiac Catheterization

Outpatient cardiac catheterization has been demonstrated by a variety of groups to be safe, practical, and highly cost-efficient, and it is widely practiced throughout the world. Outpatient catheterization can be accomplished by the radial or brachial approach, which allows the patient to be ambulatory within minutes after completion of the catheterization study (42,44–46,51). However, outpatient catheterization also can be accomplished safely by the percutaneous femoral technique (52–54). In one study (52), 2,207 patients underwent elective outpatient cardiac catheterization at the Kaiser Permamente Regional Cardiac Catheterization Laboratory in Los Angeles, California. Ninety-seven percent of the procedures were done by the percutaneous femoral approach, using 7F catheters without sheaths. Heparin was given intraarterially in a relatively low dose (2,000 to 3,000 units) and was not reversed with protamine at the end of the procedure. Hemostasis was obtained by manual compression for 10 minutes over the femoral artery, followed by placement of a pressure dressing and sandbag for 4 hours. Patients were checked at 15-minute intervals during the 4-hour surveillance period and discharged after they had become ambulatory. Each patient was contacted at home by telephone on the following day by a nurse from the outpatient observation area, and patients were seen after 1 to 2 weeks by their referring cardiologist for follow-up consultation and discussion of results. Complication rates were extremely low, lower than rates generally reported for inpatient diagnostic catheterization (see Chapter 3). More recently, 5F catheters (55,56) and radial artery catheterization techniques (42,44–46) have been used for outpatient cardiac catheterization, substantially reducing the potential risks.

On-site Cardiac Surgery

Another issue addressed in the ACC/AHA Guidelines is the question of proximity and availability of cardiac surgical facilities. The report emphasized that laboratories without in-house cardiovascular surgery must have formal arrangements with a hospital that has on-site cardiovascular surgery facilities and that regulatory and third-party reimbursement agencies should review these arrangements on a regular basis (48). Immediately available cardiac surgical backup is particularly critical for laboratories performing diagnostic catheterization on unstable, acutely ill, or high-risk patients and for those performing coronary angioplasty, endomyocardial biopsy, or transseptal catheterization.

Physician and Laboratory Caseload

Utilization levels and optimal physician caseload represent a third issue of general interest addressed in the ACC/AHA Guidelines (48). The report recommended certain levels of utilization for cost-effectiveness and maintenance of skills (Table 1.3). Note that the optimal caseload has an upper limit as well as a lower limit. A cardiologist should not have such an excessive caseload that it interferes with proper precathe-

TABLE 1.3. *ACC/AHA task force guidelines for catheterization laboratory and physician caseloads*

Category	Cases per year
Adult catheterization laboratories	300
Pediatric catheterization laboratories	150
Physician caseload*	
Adult diagnostic catheterizations	≥150 but ≤1,000
Adult PTCA procedures	75
Pediatric catheterizations	50
Electrophysiology procedures	100

* The report indicates that physicians with extensive experience (e.g., more than 1,000 independently performed catheterizations) can perform fewer catheterizations to maintain their skill levels.

Pepine CJ, Allen HD, Bashore TM, et al. ACC/AHA Guidelines for Cardiac Catheterization and Cardiac Catheterization Laboratories. American College of Cardiology/American Heart Association Ad Hoc Task Force on Cardiac Catheterization. *Circulation* 1991;84: 2213, and updated in 1999.

terization evaluation of the patient or with adequate postcatheterization interpretation of the data, report preparation, patient follow-up, and continuing medical education.

Performing the Procedure

Having carefully considered indications and contraindications, chosen a method of approach, designed the catheterization protocol, and prepared the patient, the physician's next step is to perform the cardiac catheterization itself and thereby gain the anatomic and physiologic information needed in the individual case. The individual cardiac catheterization selectively draws on the procedures that are described throughout this text. Detailed descriptions of catheter insertion and hemodynamic measurements are contained in Section II (Chapters 4–6) and Section III (Chapters 7–10), respectively. Descriptions of angiographic and interventional techniques are given in Section IV (Chapters 11–14) and Section VII (Chapters 23–28). Methods for evaluation of cardiac function and special techniques used only in selected situations are described in Section V (Chapters 15–17) and Section VI (Chapters 18–22).

These descriptions are not proposed as the only correct approaches to cardiac catheterization (many laboratories and operators take different approaches, and yet obtain excellent results). Rather, they are the methods that we have consistently found to be safe, successful, and practical. Their strengths and weaknesses are well characterized, and I believe that they constitute an excellent point of reference as one's practice continues to evolve based on new data and personal preference.

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4

Percutaneous Approach, Including Transseptal and Apical Puncture

Donald S. Baim

Harvard Medical School; Center for Innovative Minimally Invasive Therapy, Brigham and Women's Hospital, Boston, Massachusetts 02115

In contrast with the direct brachial technique (see Chapter 5), the percutaneous approach to left and right heart catheterization achieves vascular access by needle puncture (1), and thus obviates surgical isolation of the vessel during either the insertion or the subsequent withdrawal of the cardiac catheter. Once the needle has been positioned within the vessel lumen, a flexible guidewire is advanced through the needle and well into the vessel being accessed (2). This guidewire remains in an intravascular position as the needle is withdrawn and provides the means for introducing the desired catheter. Although most operators once inserted end-hole catheters directly over the guidewire, current practice is to first place an introducing sheath over the guidewire, and then to advance the catheter through this sheath (3,4). This modification reduces patient discomfort and eliminates repetitive local arterial trauma during catheter exchanges, although it does increase the size of the puncture slightly (the outer diameter of the sheath is 1F size or 0.33 mm larger than the corresponding bare catheter). At the termination of the percutaneous catheterization procedure, the catheters and introducing sheaths are withdrawn, and bleeding from the puncture sites is controlled by the application of direct pressure.

Percutaneous entry via the femoral approach has become the dominant approach to cardiac catheterization. More than 85% of the procedures contained in the 1990 registry of the Society for Cardiac Angiography and Intervention were performed via this route (5). With appropriate skill and knowledge of regional anatomy, moreover, the same percutaneous techniques used for femoral artery and vein cannulation can be adapted to allow catheter insertion from a variety of other entry sites. Venous catheterization can thus be performed via the internal jugular, subclavian, or median antecubital vein, whereas arterial catheterization can be performed via the brachial, axillary, or radial arteries, or even the lumbar aorta.

CATHETERIZATION VIA THE FEMORAL ARTERY AND VEIN

Patient Preparation

After palpation of the femoral arterial pulse within the inguinal skin crease, a safety razor is used to shave an area approximately 10 cm in diameter surrounding this point. Although most catheterizations can be performed quickly and easily from a single groin, we have found it expedient to prepare both groins routinely. The right groin is generally used, since it is more easily accessed by the operator standing on that side of the table. If difficulties in catheter advancement force a switch to the other groin once the procedure has begun, however, having the left groin already prepared saves time and inconvenience. The shaved areas are scrubbed with a

povidone-iodine/detergent mixture and then painted with povidone-iodine solution. The latter is blotted dry using a sterile towel and the patient is draped from clavicles to below the feet, leaving exposed only the sterile prepared groin areas. Most laboratories now use disposable paper drapes with adhesive-bordered apertures for this purpose, frequently packaged together with other disposable supplies (syringes, needles, bowls, and so on) in a custom kit available from any of several vendors.

Selection of Puncture Site

The adjacent femoral artery and vein (Fig. 4.1*A,B*) are the most commonly used vessels for percutaneous diagnostic cardiac catheterization (5). It is important to perform the punctures at the correct level (1 or 2 cm below the inguinal ligament) to facilitate vessel entry and avoid local vascular complications. To do so, some operators rely on the location of the inguinal skin crease to position the skin nicks through which puncture will be attempted (see later). We prefer locating the skin nicks in reference to the inguinal *ligament* (which runs from the anterior superior iliac spine to the pubic tubercle), since the position of the skin crease can be misleading in obese patients. More recently, we have begun to confirm the appropriate localization of the skin nick by fluoroscopy, which should show the nick to overlie the inferior border of the femoral head (6) (Fig. 4.1*C,D*). Making the skin nicks at this level increases the chance that needle puncture will take place in the common femoral segment, rather than too high (above the inguinal ligament) or too low (in the superficial femoral or profunda branches of the common femoral artery). The femoral artery should be easily palpable over a several-centimeter span above and below the skin nick site. The femoral vein will lie approximately one fingerbreadth medial to the artery, along a parallel course.

Most difficulties in entering the femoral artery and vein—and most vascular complications—arise as the result of inadequate identification of these landmarks prior to attempted vessel puncture. Puncture of the artery at or above the inguinal ligament makes catheter advancement difficult and predisposes to inadequate compression, hematoma formation, and/or retroperitoneal bleeding following catheter removal. Puncture of the artery more than 3 cm below the inguinal ligament increases the chance that the femoral artery will have divided into its profunda and superficial femoral branches. Puncture in the crotch between these two branches fails to enter the arterial lumen, while puncture of either one of the branches increases the risk of false aneurysm formation or thrombotic occlusion due to smaller vessel caliber. Because the superficial femoral artery frequently overlies the femoral vein, low venous punctures may pass inadvertently through the superficial femoral artery, leading to excessive bleeding and possible arteriovenous fistula formation (6) (see Chapter 3).

Local Anesthesia

Adequate local anesthesia is essential for a successful catheterization. Inadequate anesthesia leads to poor patient cooperation and makes the time in the catheterization laboratory unpleasant for both patient and operator. Once the inguinal ligament and femoral artery have been identified, the femoral artery is palpated along its course using the three middle fingers of the left hand, with the uppermost finger positioned just below the inguinal ligament. Without moving the left hand, a linear intradermal wheal of 1% or 2% lidocaine is raised slowly by tangential insertion of a 25- or 27-gauge needle along a course overlying both the femoral artery and vein at the desired level of entry.

With the left hand remaining in place, transverse skin punctures are made over the femoral artery and vein, using the tip of a No. 11 scalpel blade. The smaller needle is then replaced by a 22-gauge 1.5-inch needle, which is used to infiltrate the deeper tissues along the intended trajectory for arterial and venous entry. As this needle is advanced, small additional volumes of lidocaine are infiltrated by slow injection. Each incremental infiltration should be preceded by aspiration so that intravascular boluses can be

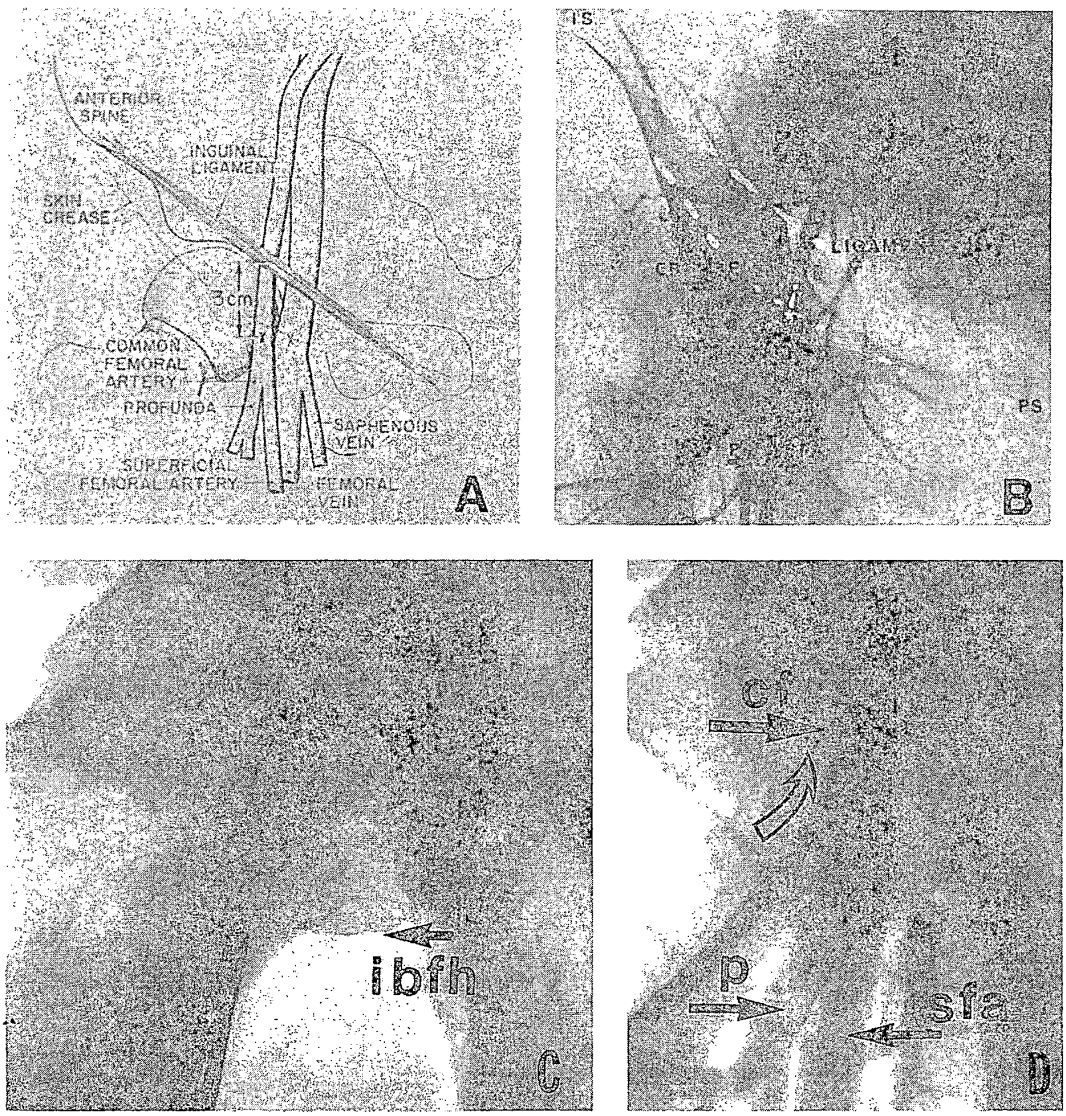


FIG. 4.1. Regional anatomy relevant to percutaneous femoral arterial and venous catheterization. **A:** Schematic diagram showing the right femoral artery and vein coursing underneath the inguinal ligament, which runs from the anterior superior iliac spine to the pubic tubercle. The arterial skin nick (indicated by X) should be placed approximately 3 cm below the ligament and directly over the femoral arterial pulsation, and 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 (see text for details). **B:** Corresponding radiographic anatomy as seen during abdominal aortography. **C:** Fluoroscopic localization of skin nick (marked by clamp tip) to the inferior border of the femoral head (ibfh). **D:** Catheter *(open arrow)* inserted via this skin nick has entered the common femoral artery (cf), safely above its bifurcation into the superficial femoral (sfa) and profunda branches. (For further details see Kim D, Orron DE, Skillman JJ, et al. Role of superficial femoral artery puncture in the development of pseudoaneurysm and arteriovenous fistula complicating percutaneous transfemoral cardiac catheterization. *Cathet Cardiovasc Diagn* 1992;25:91.)

avoided. If the anesthetic track passes through the artery or vein, infiltration should be suspended until the tip of the needle has passed out of the back wall of the vessel and then continued to the full length of the needle or to the point where the needle tip contacts the periosteum. Approximately 10 to 15 mL of 1% xylocaine administered in this fashion usually provides adequate local anesthesia. The patient should be warned that he may experience some burning as the anesthetic is injected, but that the medication will abolish any subsequent sharp sensations.

Once local anesthesia has been achieved, the small skin nicks can be enlarged and deepened, using the tips of a curved "mosquito" forceps. This procedure decreases the resistance that is encountered during subsequent advancement of the needle and subsequent vascular sheath, and increases the likelihood that any vascular bleeding will become manifest as oozing through the puncture rather than hidden in the formation of a deep hematoma.

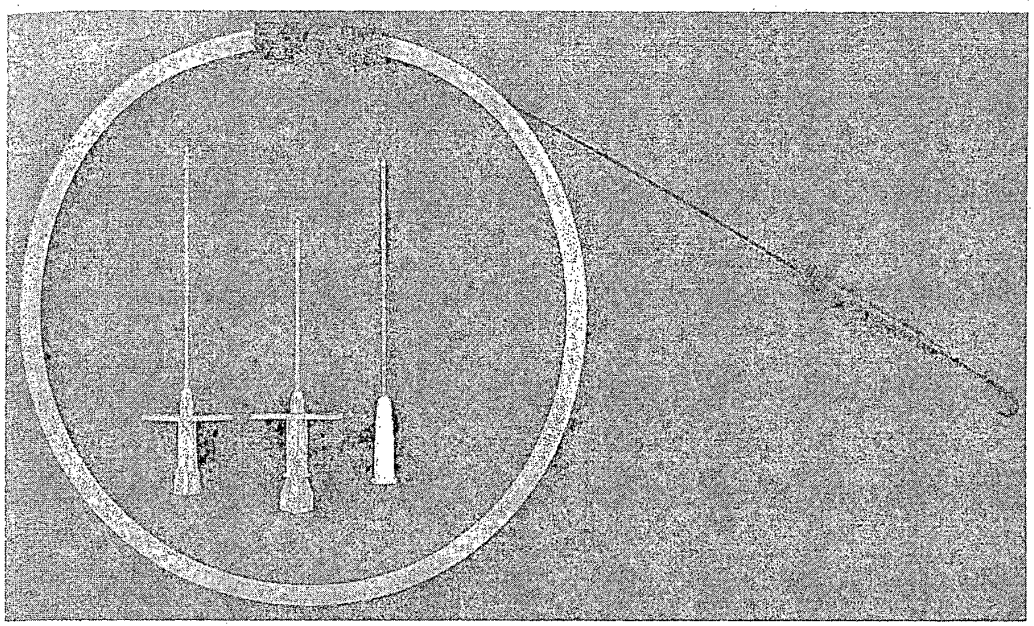
Femoral Vein Puncture

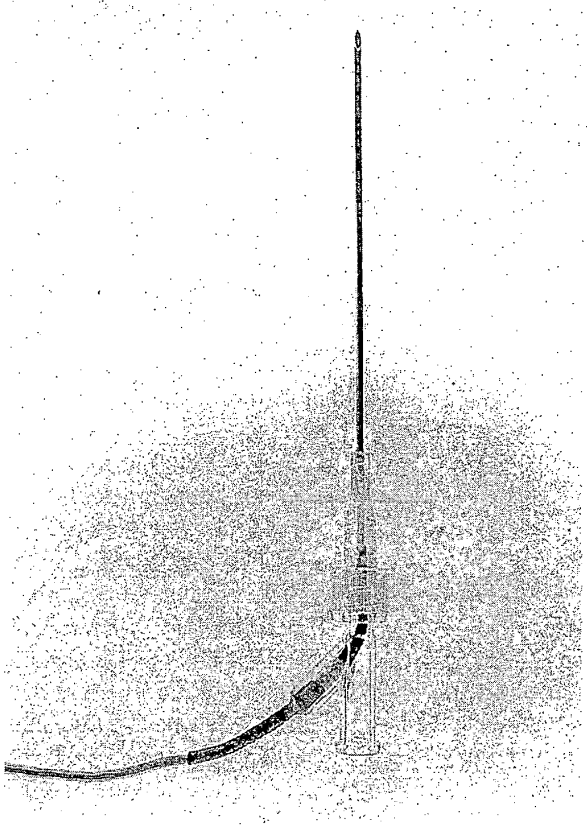
If right heart catheterization is to be performed or secure venous access is desired (for administration of fluids and medications, or rapid placement of a temporary pacing catheter), the femoral venous puncture is usually performed prior to arterial puncture. With the left hand palpating the femoral artery along its course below the inguinal ligament, the needle is introduced through the more medial skin nick. Classically, the 18-gauge thin-walled Seldinger needle, which consists of a blunt, tapered external cannula through which a sharp solid obturator projects (Fig. 4.2) was used for both arterial and venous access. The needle should be grasped so that the index and middle fingers lie below the lateral flanges of the needle and the thumb rests on the top of the solid obturator as the needle is advanced along the sagittal plane angled approximately 45° cephalad. Although this needle can occasionally be advanced up to its hub, the tip of the needle will usually stop more superficially as it encounters the periosteum of the underlying pelvic bones. The periosteum is well innervated and may be quite tender if the initial lidocaine infiltration failed to reach this level. Accordingly, forceful contact with the periosteum is neither necessary nor desirable. If the patient experiences significant discomfort, some operators will remove the obturator from the Seldinger needle and infiltrate additional lidocaine into the deep tissues through the outer cannula.

At this point, it is hoped that the Seldinger needle has transfixed the femoral vein. The obturator is removed, and a 10-mL syringe is attached to the hub of the cannula. The syringe and cannula are then depressed so that the syringe lies closer to the anterior surface of the thigh (Fig. 4.3) and the needle is more parallel (rather than perpendicular) to the vein. Gentle suction is applied to the syringe, and the whole assembly is slowly withdrawn toward the skin surface. In doing so, it is helpful to control the needle with both the left hand (which also rests on the patient's leg for support) and the right hand (which also controls the aspirating syringe). As the tip of the cannula is withdrawn into the lumen, venous blood will flow freely into the syringe.

We and most laboratories, however, have switched away from the Seldinger needle, in favor of an 18-gauge "single-wall-puncture" needle that has a sharpened tip and lacks the inner obturator. Placement of a fluid-filled syringe on the needle's hub allows direct entry into the lumen of the vein, without the need to first exit the back wall and then pull back. Otherwise,

FIG. 4.2. Percutaneous needles and guidewire. **Top panel** shows a Seldinger needle (left) with its sharp solid obturator in place, a Potts-Cournand needle (center), which differs in the fact that its obturator is hollow and therefore allows the operator to see blood flashback as the artery is punctured, and an 18-gauge thin-wall needle (right) used for internal jugular vein puncture and now frequently also for arterial entry. These percutaneous needles are surrounded by an 0.038-inch, 145-cm J guidewire. **Bottom panel:** A Doppler-guided Smart Needle.





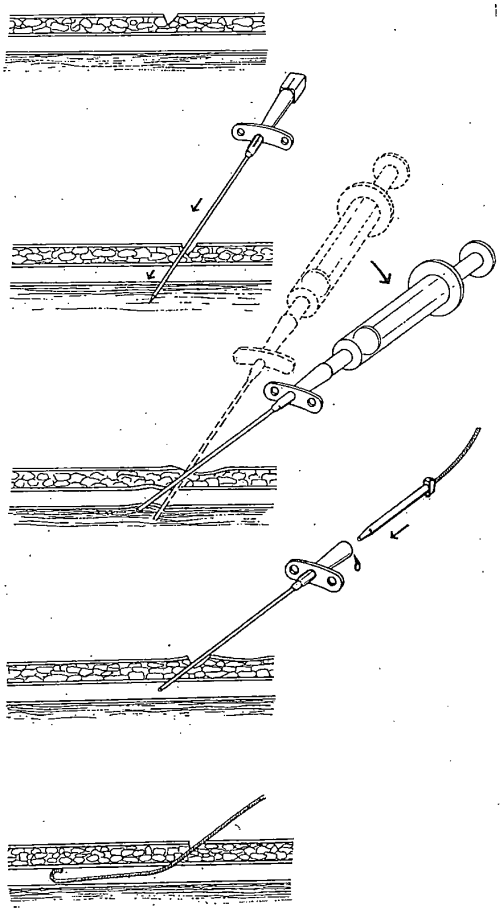


FIG. 4.3. Seldinger technique for venous puncture. A skin nick has been created overlying the desired vein, which is punctured through and through by a Seldinger needle with its solid obturator in place. In the center panel, the obturator is removed and the needle cannula is attached to a syringe. Depression of the syringe toward the surface of the skin tents the vessel slightly and facilitates axial alignment of the cannula at the moment that slow withdrawal brings the tip of the cannula back into the vessel lumen. This is recognized by the sudden ability to withdraw venous blood freely into the syringe, which is then removed from the needle cannula to permit advancement of the J guidewire (shown here with a plastic straightener in place). Once the guidewire has been advanced safely into the vessel, the needle cannula can be removed.

the technique used after entry of the venous lumen has been achieved is identical. With the left hand stabilizing the needle, the right hand is used to remove the syringe and to advance a 0.035- or 0.038-inch J guidewire into the hub of the needle. The wire tip may be straightened by hyperextension of the wire shaft in the right hand or by leaving the tip of the wire within the plastic introducer supplied by the manufacturer. The wire should slide through the needle and 30 cm into the vessel with no perceptible resistance. Fluoroscopy should then show the tip of the guidewire just to the left (patient's right) of the spine.

If difficulty is encountered in advancing the guidewire, it should never be overcome by force. Fluoroscopy may simply reveal that the tip of the wire has lodged in a small lumbar branch; it can be drawn back slightly and redirected or gently prolapsed up the iliac vein. When resistance to advancement is encountered at or just beyond the tip of the needle, however, even greater care is required. This resistance may simply be caused by apposition of the tip of the needle to the back wall of the vein, which can be corrected by further depression of the needle hub, with or without slight withdrawal of the needle shaft. If this maneuver fails to allow free advancement of the wire, however, the wire should be removed, and the syringe should be reattached to the needle hub to ensure that free flow of venous blood is still present before additional wire manipulation is attempted; the wire should not be reintroduced unless free flow is obtained. If it is necessary to withdraw the wire, this should always be done gently, since it is theoretically possible for the wire to "snag" on the tip of the needle. Were this to occur, the needle and wire should be removed as a unit. If the wire still cannot be advanced after these manuevers, the needle should be withdrawn, and the puncture site should be compressed for 1 to 3 minutes. The anatomic landmarks should be reconfirmed and puncture reattempted. In some cases, puncturing the vein during a Valsalva maneuver may help by distending the femoral vein and making clean puncture more likely.

After the wire has freely entered the vein, the needle is removed, leaving the wire well within the vein and secured at the skin entry site by the

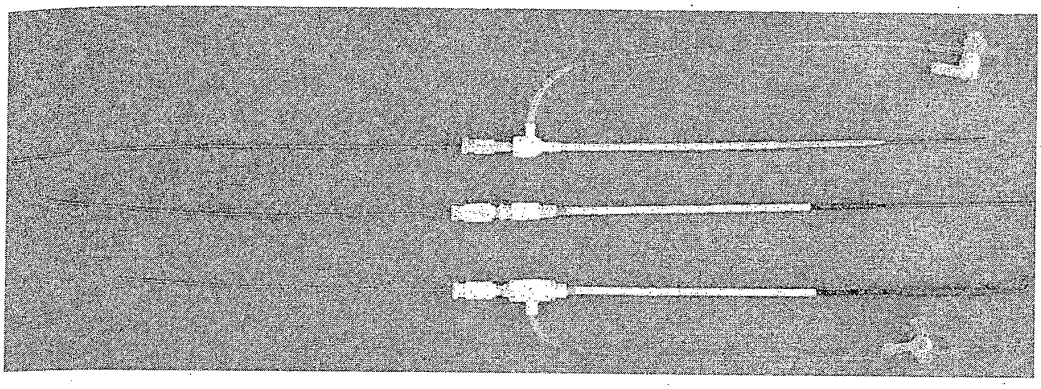


FIG. 4.4. Vascular sheaths. **Center:** An original sheath and dilator assembly (USCI "888"). In contrast to the original design, modern arteriovenous introducers are equipped with backbleed valves and sidearm attachment. **Top:** A Cordis sheath. **Bottom:** A USCI Hemaquet. Each device is inserted over a conventional guidewire as a unit, following which the inner Teflon dilator is removed to permit catheter introduction. The sidearm sheaths also permit fluid infusion and an additional site for pressure monitoring with the catheter in place.

left hand. The protruding wire is wiped with a moistened gauze pad, and its free end is threaded into the lumen of a sheath and dilator combination adequate to accept the intended right-sided heart catheter. All current sheaths are equipped with a backbleed valve and sidearm connector (Fig. 4.4) to control bleeding around the catheter shaft and to provide a means of administering drugs or extra intravenous fluids during the right-sided heart catheterization. The operator must make sure that he has control of the proximal end of the guidewire and that it is held in a fixed position as the sheath and dilator are introduced through the skin. Insertion is eased if the sheath and dilator are rotated as a unit while they are advanced progressively through the soft tissues. If excessive resistance is encountered, it may be necessary to remove the dilator from the sheath and to introduce the dilator alone before attempting to introduce the combination. If inspection shows that initial attempts have created significant burring at the end of the sheath, a new sheath should be obtained.

Once the sheath is in place, the wire and dilator are removed, and the sheath is flushed by withdrawal of blood and injection of heparinized saline solution. In our laboratory we usually infuse the sidearm of the venous sheath from a 1-L bag of normal saline solution, connected via a sterile length of intravenous extension tubing, to maintain sheath patency and provide a carrier for drug administration by the nurse. Although drug administration can also take place via a peripheral intravenous line, the side arm of the sheath avoids any concerns about how quickly volume can be administered or whether infiltration of the peripheral line might jeopardize drug delivery in an emergency. Even if right heart catheterization is not planned, the femoral sheath makes it easy to place a right heart catheter or a temporary transvenous pacemaker lead if hemodynamic instability or bradyarrhythmia ensue.

Catheterizing the Right Heart from the Femoral Vein

A right (as well as a left) heart catheterization is needed to obtain a "full" profile of the hemodynamic state. Only the right heart catheterization can provide data regarding mean left heart filling pressure (the pulmonary capillary wedge, rather than just the post-a wave left ventricular end-diastolic pressure), detect pulmonary arterial hypertension, measure the cardiac output, and detect left-to-right intracardiac shunts. Leaving the right heart catheter in the pulmonary artery during the procedure also gives an ongoing measure of changes in the hemodynamic state as fluid and contrast loading take place, various medications (nitrates, diuretics, etc.) are given, and episodes of ischemia develop and are

treated. For these reasons, our practice was once to perform a right heart catheterization in every patient who came to the cardiac catheterization laboratory.

In contrast, the 1990 Society for Cardiac Angiography and Intervention (SCA&I) survey showed that the practice was to perform right heart catheterization in only 28% of procedures (5). This practice has likely fallen further, after several standard-setting and regulatory agencies ruled that a left heart catheterization alone is adequate for most patients undergoing evaluation for coronary artery disease. The time (<5 minutes), added expense (<$100), and added risk (<1/10,000) of right heart catheterization are small, but so is the added information. We now skip the right heart catheterization in patients with a primary diagnosis of coronary artery disease, unless they have symptoms of congestive heart failure, noninvasive evidence of depressed left ventricular function or associated valvular disease, or recent myocardial infarction. In such patients, however, we still believe that the quantitation of overall hemodynamic function provided by right heart catheterization justifies performance of this low-risk adjunctive part of the overall catheterization evaluation.

If right heart catheterization *is* to be performed, the desired right heart catheter (Fig. 4.5) is flushed, attached to the venous manifold, introduced into the sheath, and advanced up the inferior vena cava. Although conventional woven Dacron (Goodale-Lubin or Cournand) catheters provide excellent torque control, their inherent stiffness makes them poorly suited for

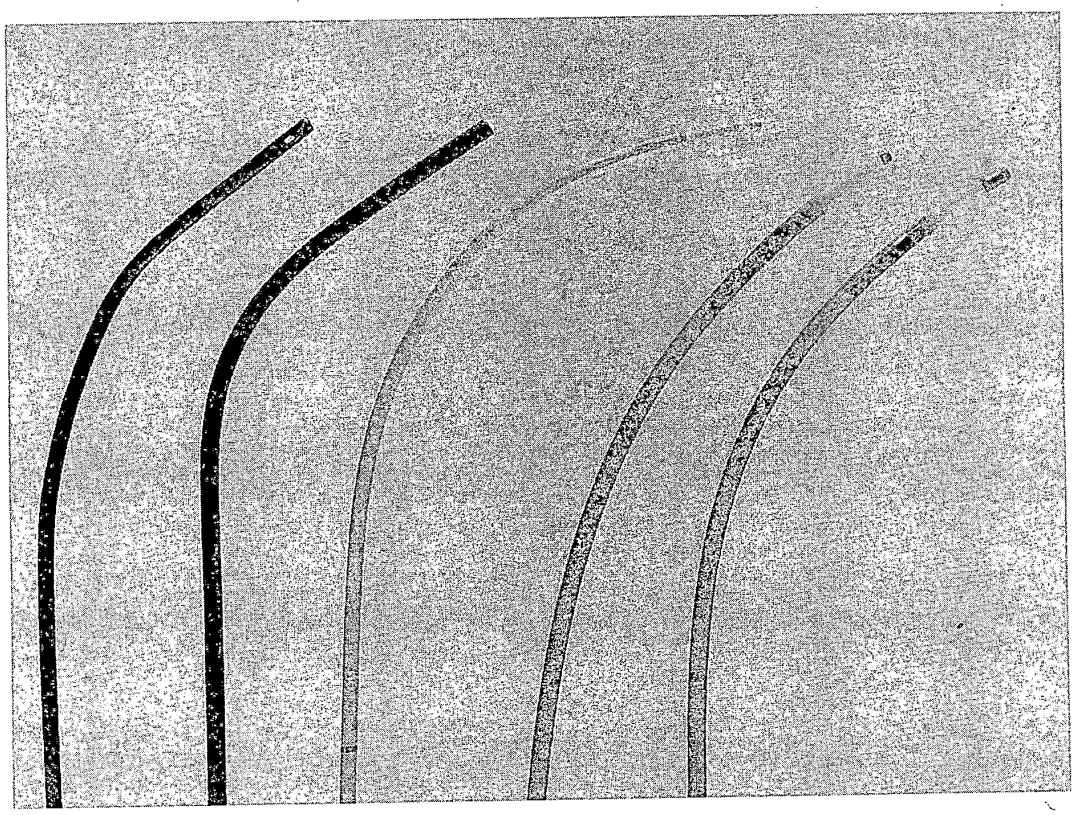


FIG. 4.5. Right-sided heart catheters used from the femoral approach. **Left:** Woven Dacron Goodale-Lubin, Cournand catheters. **Center:** Swan-Ganz catheter. **Right:** Newer balloon catheters, including the PWP pressure measurement catheter and the Baim-Turi catheter with bipolar pacing electrodes (USCI).

routine use in a training laboratory. We therefore for a time used 7F Swan-Ganz catheters to exploit their ease of passage, low risk of injury to the right-sided heart chambers, and ability to perform thermodilution measurements of cardiac output. Unfortunately, such soft catheters have poor frequency response (see Chapter 7), do not adequately transmit the torque required for easy catheterization of the right-sided heart from the femoral approach, and accept only 0.021-inch guidewires. To bridge this gap, we have begun using a stiffer, balloon-tipped catheter [PWP monitoring catheter (USCI, Billerica, MA)] that combines the safety of the Swan-Ganz catheter with the catheter control and frequency response previously found only in the woven Dacron catheters. The larger lumen diameter and stiffer wall of this catheter (compared with the traditional Swan-Ganz design) improve frequency response and allow the passage of conventional 0.035- and 0.038-inch-diameter guidewires when necessary. When temporary pacing is desired, this catheter is also available with bipolar pacing capacity (Baim-Turi, USCI).

Deviation of the catheter tip from its parasspinous position during advancement from the leg suggests entry into a renal or hepatic vein, which can be corrected by slight withdrawal and rotation of the catheter. Once the catheter is above the diaphragm and within the right atrium, it is rotated counterclockwise to face the lateral wall of the right atrium (Fig. 4.6). Additional counterclockwise rotation and gentle advancement allow passage of the catheter tip into the superior vena cava, which is contiguous with the posterolateral wall of the right atrium. In contrast, anterior orientation of the catheter tip at this point may result in its entrapment in the right atrial appendage and inability to reach the superior vena cava. Alternatively, the tip of the catheter can be withdrawn to the inferior vena cava, and a 0.035-inch J guidewire can be introduced to bridge the straight line path from the inferior to the superior vena cava, along the back wall of the right atrium. Once in position, a baseline superior vena caval blood sample is obtained for measurement of oxygen saturation and comparison with the subsequently measured pulmonary arterial blood oxygen saturation, to screen for unsuspected left-to-right shunts. The catheter is then flushed with heparinized saline solution and withdrawn to the right atrium for pressure measurement.

To advance a catheter from the femoral vein to the pulmonary artery, the tip of the catheter is positioned in the lower portion of the right atrium, directed toward its lateral border. If a balloon flotation catheter is being used, the balloon is inflated at this point. Clockwise rotation is applied, which causes the catheter tip to sweep the anterior and anteromedial wall of the right atrium, along which the tricuspid valve is located (see Fig. 4.6). As the catheter tip passes over the tricuspid orifice, slight advancement causes it to enter the right ventricle, where pressure is again recorded. If the right atrium is enlarged, greater curvature of the catheter may be necessary (i.e., a large J loop). Such a loop may be formed by bending the tip of the catheter against the lateral right atrial wall or by engaging in the ostium of the hepatic vein (just below the diaphragm). This larger loop can then be rotated clockwise in the atrium as described earlier, causing the tip of the catheter to enter the right ventricle. Right ventricular pressure is then recorded.

Simple advancement of the catheter in the right ventricle causes the tip to move toward the apex of that chamber and usually does not result in catheterization of the pulmonary artery. To achieve this latter end, the catheter must be withdrawn slightly so that its tip lies horizontally and just to the right (patient's left) of the spine. In this position, clockwise rotation causes the tip of the catheter to point upward (and slightly posteriorly) in the direction of the right ventricular outflow tract (Fig. 4.6). The catheter should be advanced only when it is in this orientation to minimize the risk of ventricular arrhythmias or injury to the right ventricle. Advancement may be facilitated if performed as the patient takes a deep breath. If these maneuvers fail to achieve access to the pulmonary artery due to enlargement of the right atrial and ventricular chambers, the catheter may be withdrawn to the right atrium and formed into a large "reverse loop," which allows the tip of the catheter to cross the tricuspid valve in an upward orientation more

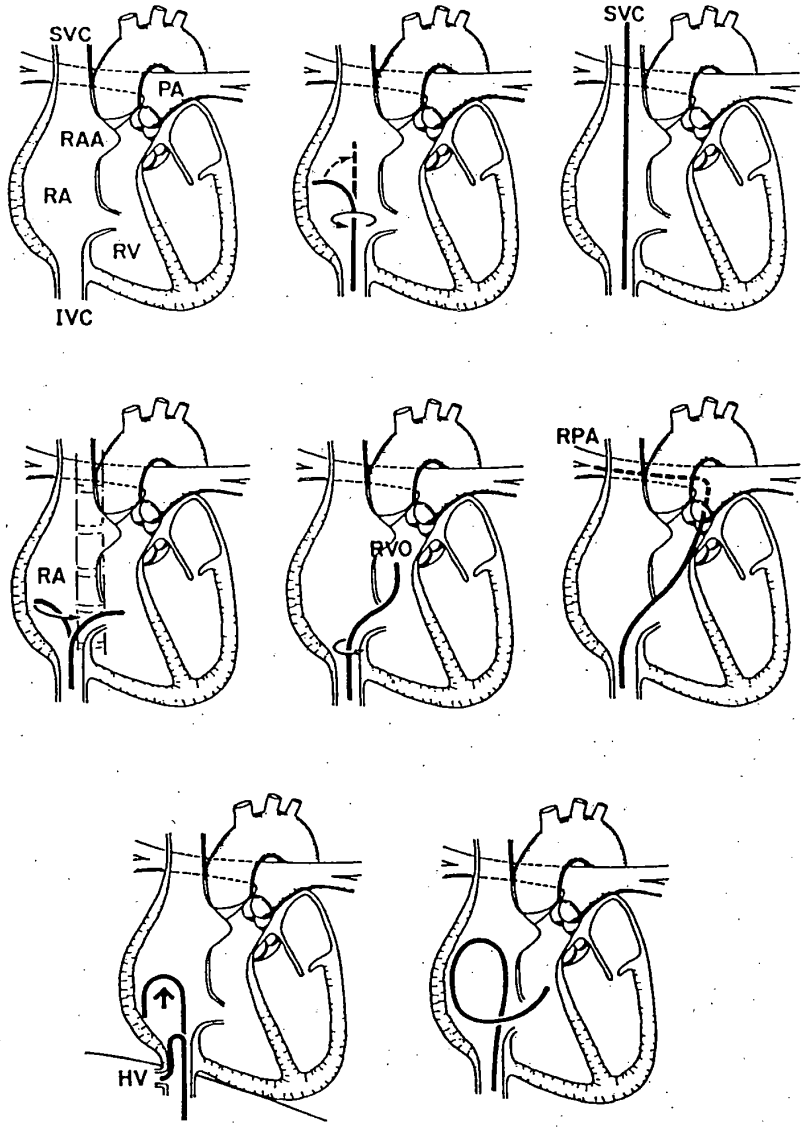


FIG. 4.6. Right heart catheterization from the femoral vein, shown in cartoon form. **Top Row:** The right heart catheter is initially placed in the right atrium (RA) aimed at the lateral atrial wall. Counterclockwise rotation aims the catheter posteriorly and allows advancement into the superior vena cava (SVC). Although 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 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 for 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.

likely to enter the outflow tract (Fig. 4.6, bottom right). When manipulated appropriately, the catheter tip should cross the pulmonic valve and advance to a wedge position without difficulty. Having the patient take a deep breath and cough during advancement is often of assistance in achieving a wedge position. Alternatively, a small amount of air may be released from the balloon to decrease its size and facilitate wedging in a smaller, more distal branch of the pulmonary artery. Catheters advanced from the leg are more likely to seek the left pulmonary artery, whereas catheters advanced from above tend to seek the right pulmonary artery as they make a continuous counterclockwise curve through the right heart chambers. If needed, either pulmonary artery can be catheterized by appropriate manipulation or careful introduction of a curved J guidewire, although we generally do not like to extend guidewires into the thin-walled pulmonary arteries unless absolutely necessary. Following measurement of the wedge pressure, the balloon (if a balloon-tip catheter is being used) is deflated, and the catheter is withdrawn into the more proximal left or right pulmonary artery. There, pulmonary arterial pressure is measured and another blood sample for measurement of oxygen saturation is obtained. If a more simultaneous "snapshot" of the hemodynamic state is desired, these "entry" pressures can be rerecorded during a right-sided heart pullback. For practical reasons, we now tend to rerecord only the wedge pressure (simultaneous with the left ventricular pressure) and pulmonary artery pressure, coincident with the measurement of the cardiac output. We then leave the right heart catheter in the proximal pulmonary artery for the duration of the case, allowing continuous monitoring of the pulmonary artery diastolic pressure as an index of volume status and ischemic left ventricular dysfunction.

Attempts to perform right heart catheterization occasionally result in entry into other structures. If a woven Dacron catheter is advanced in the right atrium with a posteromedial orientation, it may cross a patent foramen ovale and enter the left atrium. This is sometimes hard to detect by catheter position alone because the catheter appearance in the left atrium or ventricle may be indistinguishable (in the anteroposterior view) from its course during usual right heart catheterization. It can, however, be recognized by a change in the pressure waveform, position of the catheter tip across the spine, and ability to withdraw fully oxygenated blood from the catheter tip. Although more unusual, a woven Dacron catheter can also enter the ostium of the coronary sinus, located inferiorly and posteriorly to the tricuspid orifice. There will be continued presence of a right atrial waveform, but blood sampling will disclose a far lower oxygen saturation (20% to 30%) than was present in the superior vena cava. Anatomic abnormalities can also be suspected when the catheter takes an unusual position or course during attempted right heart catheterization. Figure 4.7 depicts the appearance of the right-sided heart catheter course in three such congenital abnormalities (persistent left superior vena cava, patent ductus arteriosus, and anomalous pulmonary venous return). The most important points about these side trips off the beaten path to the right ventricle are that the operator should recognize that the tip of the catheter is not in the right ventricle (i.e., one should not attempt to get to the pulmonary artery) and should decide where the catheter is (by pressure monitoring, saturation analysis, or hand injection of a small amount of contrast agent) before withdrawing the catheter to the right atrium and proceeding with the right heart catheterization.

In patients with elevated right heart pressures, prior placement of an inferior vena caval filter or umbrella, those undergoing specialized procedures (endomyocardial biopsy, coronary sinus catheterization), or those in whom prolonged postprocedure monitoring with a balloon-flotation catheter is desired, the *right internal jugular vein* offers an excellent alternative to the femoral vein. The technique for jugular puncture is described in Chapter 20, and the method of advancing the right-sided heart catheter to the pulmonary artery is identical to that described for the brachial approach in Chapter 5. On occasion, percutaneous right heart catheterization is performed from the subclavian or median basilic vein using a similar technique.

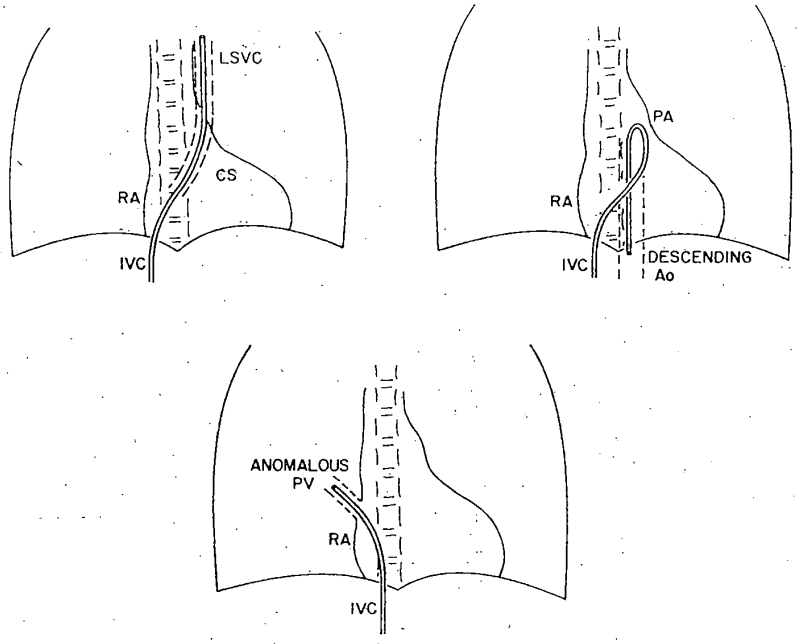


FIG. 4.7. Unsuspected anatomic abnormalities frequently can be detected by an unusual catheter course or position. **Upper left panel:** The course of a catheter passed from the femoral vein to the inferior vena cava (IVC), right atrium (RA), coronary sinus (CS), and up into an anomalous left superior vena cava (LSVC). **Upper right panel:** The catheter crossing from the pulmonary artery (PA) to the descending aorta (Ao) by way of a patent ductus arteriosus. **Bottom panel:** The catheter entering an anomalous pulmonary vein draining into the right atrium.

Femoral Artery Puncture

The common femoral artery is punctured by inserting the Seldinger or single-wall-puncture needle through the more lateral skin nick. Again, the needle is inserted at approximately 45° along the axis of the femoral artery as palpated by the three middle fingers of the left hand. The experienced operator may feel the transmitted pulsations as the tip of the needle contacts the wall of the femoral artery. With the Seldinger needle it is customary to advance the needle completely through the artery until the periosteum is encountered. The obturator is then removed, and the hub of the needle is depressed slightly toward the anterior surface of the thigh. Arterial pressure makes it unnecessary to attach a syringe to the cannula, so that both hands can be used to stabilize the needle as it is slowly withdrawn. When the needle comes back into the lumen of the femoral artery as evidenced by vigorous pulsatile flow of arterial blood, a 0.035- or 0.038-inch J guidewire should then be advanced carefully into the needle.

If a single wall puncture is desired, the operator may prefer a Potts-Cournand needle (Fig. 4.2), in which the obturator has a small lumen that transmits a flashback of arterial blood as the vessel is entered, or the same single-wall-puncture needle described for venous entry. When the femoral pulse is difficult to palpate or numerous needle insertions have been fruitless, it may be easiest to utilize the 18-gauge Smart-Needle (CardioVascular Dynamics, Irvine CA; see Fig. 4.2, bottom panel). The obturator of this device contains a Doppler crystal that helps aim the needle tip toward the center of the arterial lumen. Pulsatile arterial flow has auditory characteristics that distinguish it clearly from the more continuous venous flow signals detected from the adjacent femoral vein.

Whichever needle is used to enter the arterial lumen, the guidewire introduced through the

needle should move freely up the aorta [located to the right (patient's left) side of the spine on fluoroscopy] up to the level of the diaphragm. When difficulty in advancing the guidewire is encountered at or just beyond the tip of the needle and is not corrected by slight depression or slight withdrawal of the needle, the guidewire should be withdrawn to ensure that vigorous arterial flow is still present before any further wire manipulation is attempted. If flow is not brisk or if the wire still cannot be advanced, the needle should be removed and the groin should be compressed for 5 minutes. The operator should verify the correctness of the anatomic landmarks and attempt repuncture of the femoral artery. If the second attempt is unsuccessful in allowing wire advancement, a third attempt on the same vessel is unwise, and an alternative access site should generally be selected.

If wire motion is initially free but resistance is encountered after several centimeters (particularly if the patient complains of any discomfort during wire advancement), extensive iliac disease or subintimal position of the wire is possible. The wire should be pulled back slightly under fluoroscopic control and the needle should be removed as the left hand is used to stabilize the wire and control arterial bleeding. After the wire is wiped with a moist gauze pad, a small (5F) dilator can be cautiously introduced to a point just below where wire movement became difficult. The wire is then withdrawn from the dilator, which is aspirated to ensure free flow of blood and flushed carefully. A small bolus of contrast medium (either a low-osmolar agent or ionic contrast diluted to half strength, to avoid local discomfort) is then injected gently under fluoroscopic monitoring. This should disclose the anatomic reason for difficult wire advancement—generally either iliac tortuosity, stenosis, or dissection. Problems advancing the wire above the aortic bifurcation may also suggest the presence of an abdominal aortic aneurysm (7), which warrants use of soft-tip guidewires and extreme care to avoid perforation or dislodgment of cavitary thrombus or debris. If contrast injection through the small dilator reveals that subintimal wire passage has occurred, retrograde left heart catheterization should be relocated to the other femoral artery or to the brachial or radial artery and the patient should be observed for signs of progressive dissection or arterial compromise, both of which are fortunately rare with retrograde guidewire dissections. If the problem turns out to be more tortuosity or stenosis (Fig. 4.8*A*), a more specialized guidewire (e.g., a steerable peripheral guidewire such as a Wholey, or a hydrophyllic-coated guidewire such as the Terumo Glidewire) may be carefully reintroduced through the dilator in an attempt to reach the descending aorta. In an era when the obstructing lesion can be quickly and effectively treated by angioplasty or stent placement (see Chapter 27), iliac stenosis is no longer a firm indication to abandon retrograde left heart catheterization!

In an aging population with diffuse atherosclerotic disease, the question of performing left heart catheterization via a prosthetic (e.g., aortobifemoral) graft arises frequently (8,9). This is not an ideal approach because the graft wall is tough (making sheath insertion difficult), such grafts may contain diffuse atherosclerotic or thrombotic debris, and graft closure or serious graft infection may occur. The graft should be identified as a separate structure from the adjacent native femoral artery and punctured using a front-wall approach. Even if the graft hood is punctured correctly, the guidewire may pass through the anastomosis and into the native femoral artery rather than proximally up the graft (8). In that event, contrast injections through a small dilator in a right anterior oblique (RAO) projection (right leg) and the use of special steerable guidewires may be required to remain within or cross into the graft lumen, and thereby reach the descending aorta (Fig. 4.8*B*). A vascular introducing sheath should always be used to avoid excessive friction during catheter movement, or excessive traction on catheter tips during withdrawal, but may require the use of serial dilators for passage of the sheath through the tough graft wall. This approach via a vascular graft can thus be used with care, particularly when other alternatives (e.g., brachial, axillary, or radial artery) are themselves less than desirable. Some operators choose to administer prophylactic antibiotics [Kefzol (Eli Lilly, Indian-

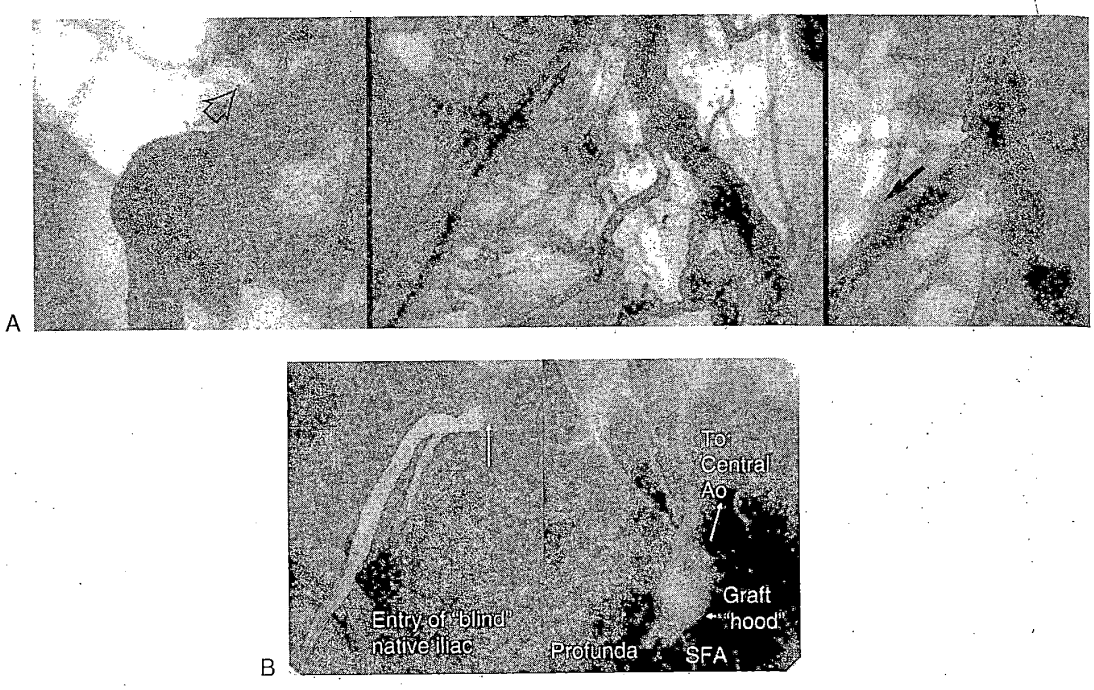


FIG. 4.8. A: Entry of the right femoral artery was straightforward, but guidewire advancement stopped in the iliac system. **Left:** Contrast injection through a 5F dilator shows severe iliac stenosis with extensive cross-pelvic collaterals. This was crossed with a Terumo Glidewire to allow completion of the diagnostic angiography and a right coronary artery angioplasty (not shown). **Center:** Injection in the abdominal aorta shows the proximal extent of the iliac stenosis. **Right:** Iliac stenosis then dilated and treated by placement of a Palmaz-Schatz iliac stent, with restored antegrade iliac flow. **B:** Retrograde left heart catheterization in a patient with previous aortic-bifemoral grafting (left) entry of the graft hood has resulted in passage of the wire into the "blind" native iliac. In a RAO projection, the more anterior pathway to the central aorta (Ao) via the graft can be seen overlying the native iliac, with the bifurcation of the common femoral artery into the profunda and superficial femoral artery (SFA) just below.

apolis, IN) 1 g every 8 hours for 24 hours] when accessing a prosthetic graft.

Catheterizing the Left Heart from the Femoral Artery

Once the guidewire has been advanced to the level of the diaphragm and the needle has been removed, the operator's left hand is used to stabilize the wire and control arterial bleeding while the wire is wiped with a moistened gauze pad to remove any adherent blood. If the catheter is to be introduced directly into the artery, the soft tissues are predilated by brief introduction of a Teflon arterial dilator one F-size smaller than the intended catheter, before inserting the left heart catheter itself. Essentially all left heart catheterizations from the femoral approach, however, are now performed using an appropriate-sized vascular sheath (e.g., a 7F sheath for a 7F catheter) that is equipped with a backbleed valve and sidearm tubing as described earlier. The 15-cm length sheath is commonly used for diagnostic catheterization but can only reach the mid-iliac. In the presence of severe tortuosity, it may be preferable to use the 23-cm-length sheath designed for interventional procedures, which is sufficiently long to enter the distal aorta above the bifurcation. This helps to improve the torque responsiveness of diagnostic catheters under those circumstances.

The chosen sheath is introduced over the

guidewire (the proximal end of which is held in a straightened, fixed position) with a rotational motion, following which the guidewire and dilator are removed and the sheath is aspirated, flushed, and connected to a pressurized flush system [Intraflo II (30 mL/hr), Abbot Critical Care, North Chicago, IL] to avoid clot formation in the sheath. Alternatively, this sidearm can be connected to a manifold for monitoring arterial pressure at a separate site (e.g., during passage of a pigtail catheter across a stenotic aortic valve). This sheath should be "power" flushed immediately after each catheter is introduced or withdrawn, by briefly activating the Intraflow device.

Classically, once the sheath had been inserted, the guidewire was removed. The desired left heart catheter was then flushed and loaded with a 145-cm J guidewire and its nose was introduced into the backbleed valve of the sheath. The soft end of the guidewire was then advanced carefully through the catheter, out the end of the sheath, and to the level of the diaphragm before the catheter itself was advanced. One concern, however, is that readvancement of the guidewire out the end of the sheath can cause vascular injury if severe iliac tortuosity or disease is present. We therefore adopted a modified technique in which a short-exchange length (175 cm) Newton J (Cook, Bloomington, IN) is placed through the access needle and its tip is left at the diaphragm as the dilator is removed from the sheath and the left heart catheter is inserted. This obviates the need to renegotiate complex iliofemoral anatomy with the guidewire.

Once the catheter has been advanced to the desired level (either above the diaphragm or into the ascending aorta), the guidewire is removed, so that the catheter can be connected to the arterial manifold and double-flushed (withdrawal and discarding of 10 mL of blood, followed by injection of heparinized saline solution). All subsequent left heart catheters are then introduced by reinserting this wire to the level of the diaphragm (allowing one catheter to be removed and the second to be reintroduced safely), rather than withdrawing the first catheter completely and then inserting the second catheter and wire through the sheath *de novo*. Of course if the left heart catheterization is being performed without the aid of a sheath, the operator must leave the tip of the wire in the abdominal aorta during the removal of the first catheter and the introduction of a second catheter to retain access to the vessel. These "over-the- wire" catheter exchanges are facilitated by extending the back end of the wire straight down the patient's leg and holding it fixed there to ensure that the wire remains in constant position within the aorta as the newer catheter is advanced.

A Word About Heparin

As described in Chapter 3, early catheterizations from the femoral artery had a higher incidence of major complications than catheterization from the brachial artery. One difference was that brachial catheterization utilized systemic heparinization to avoid thrombosis in the smaller brachial artery with a potentially occlusive catheter in its lumen. When systemic heparinization was adopted in femoral procedures, the rates of complications became equivalent. On this basis, the practice of full intravenous heparinization (5,000 U) immediately after the left-sided sheath was inserted, became a standard way to provide therapeutic anticoagulation that lasted at least 40 minutes in most patients. Lesser amounts of heparin (2,500 to 3,000) may also be used, particularly in smaller patients. If it is decided to perform catheter-based coronary intervention, larger heparin doses (usually 10,000 U, or 70 to 100 U/kg) are required, which will require further supplementation if a smaller heparin dose has been administered for the immediately preceding diagnostic procedure. This type of higher heparin dosing is routinely monitored by an activated clotting time (ACT) machine in the cardiac catheterization laboratory, and titrated to an ACT of roughly 300 seconds (10). If it is planned to use an intravenous IIb/IIIa receptor blocker, lower levels of heparin anticoagulation (ACT 250 to 275) may be desired to prevent excessive bleeding risk. Given the limitations of heparin (see Chapter 3), other anticoagulant agents, including low-molecular-weight heparins and other direct-acting thrombin antagonists, are

being explored for cardiac catheterization procedures.

While the use of heparin is mandatory for interventional or prolonged diagnostic procedures, many laboratories have abandoned the use of systemic heparinization for simple diagnostic catheterizations, where the complications are extremely low with or without heparin (11). For this issue to be decided scientifically, more than 100,000 patients would have to be randomized to undergo diagnostic catheterization with and without systemic heparinization. Absent such trial data, we are now less likely to use systemic heparinization for simple procedures but still feel that systemic heparinization is appropriate for more prolonged or complex diagnostic catheterizations, cases where a guidewire will be required to cross a stenotic aortic valve, and (absolutely) for all percutaneous coronary interventions.

If systemic heparinization is used, its effects must be reversed at the termination of the left heart catheterization and associated angiography. This is usually accomplished by the administration of protamine (1 mL = 10 mg of protamine for every 1,000 IU of heparin) (12). The operator should be watchful for potential adverse reactions to protamine, characterized by hypotension and vascular collapse, as discussed in Chapter 3. Protamine reactions appear to be more common in insulin-dependent diabetics and patients with previous protamine exposure, who are more likely to have elevated levels of IgG or IgE antiprotamine antibodies (13). Although severe protamine reactions in these patients are uncommon, we prefer delaying sheath removal for approximately 1 hour in insulin-dependent diabetics to allow heparin to wear off without protamine administration. This is also our practice in patients with unstable symptoms, threatening anatomy, where there is a concern that abrupt reversal of the heparin effect may trigger thrombosis.

Catheter Selection

The initial left heart catheter in most cases is a pigtail catheter with multiple side holes (Fig. 4.9). This catheter usually can be flushed in the descending aorta and then advanced to the ascending aorta without difficulty. If left ventricular and femoral arterial (sheath side arm) pressures are being monitored (as in catheterization to evaluate aortic stenosis), the rough equality of central aortic and femoral arterial pressure should be confirmed at this time (Fig. 4.10) (3,4,14). The systolic peak in the femoral waveform may be slightly delayed and accentuated compared with the ascending aortic pressure trace, but the diastolic and mean pressures should be virtually identical. A greater difference in mean pressure between the catheter and the sheath may be seen in a patient with an extensively diseased iliac artery and may require the use of a longer sheath, as described earlier. For the highest-pressure fidelity, the sheath size should be one F larger than the intended left heart catheter (e.g., a 6F pigtail advanced through a 7F sheath). Alternatively, catheters can be advanced from separate arterial entry sites to record left ventricular and ascending aor-

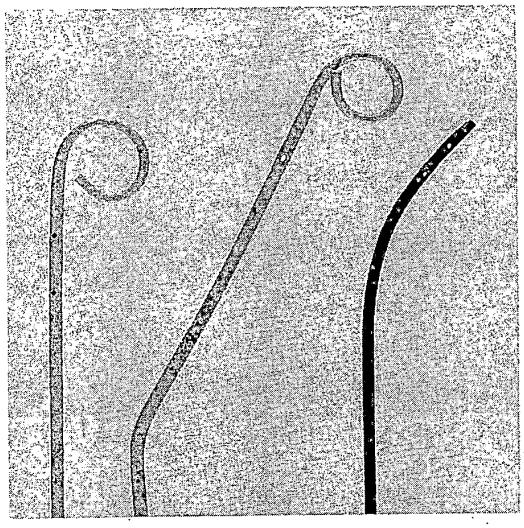


FIG. 4.9. Left heart catheters used from the femoral approach. **Left to right:** Pigtail, 145° angled pigtail, and Teflon Gensini catheter (no longer in common use). All three catheters have an end hole to allow placement over a guidewire and multiple side holes to minimize the tendency for catheter whipping or intramyocardial injection during power injection of contrast.

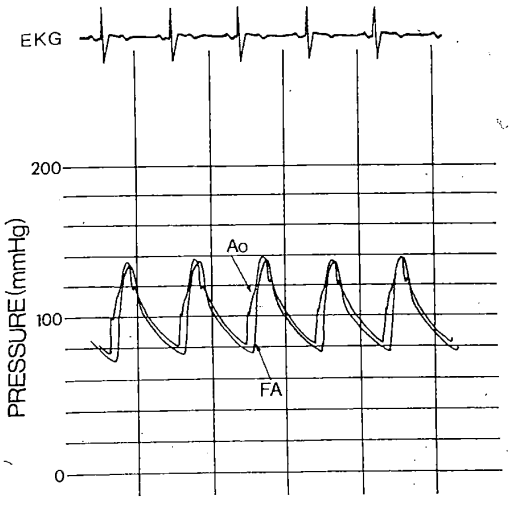


FIG. 4.10. Central aortic pressure (Ao) measured through a 7.3F pigtail catheter (Cook) and femoral artery (FA) pressure measured from the sidearm of an 8F arterial sheath (Cordis, Miami Lakes, FL). Only minimal damping of the femoral artery pressure is seen, blunting its systolic overshoot, which frequently exceeds central aortic systolic pressure (see Chapter 9). With larger (7.5F and 8F) catheters, more damping may occur in the sidearm pressure. Catheter and sidearm are connected to small-volume-displacement transducers without intervening tubing.

tic pressure, or a specially designed 8F pigtail with separate end lumens and a side-hole lumen may be used to perform such pressure recordings (15).

Crossing the Aortic Valve

After measurement of the ascending aortic pressure, the pigtail catheter is advanced across the aortic valve and into the left ventricle. If the aortic valve is normal and the pigtail is oriented correctly, it will usually cross the valve directly. In many cases, however, it may be necessary to advance the pigtail down into one of the sinuses of Valsalva to form a secondary loop (Fig. 4.11). As the catheter is withdrawn slowly, this loop will open to span the full diameter of the aorta, at which point a very subtle further withdrawal will often cause the pigtail to fall across the valve.

If significant aortic stenosis is present, the pigtail must be advanced across the valve with the aid of a straight 0.038-inch guidewire. Approximately 6 cm of the guidewire is advanced beyond the end of the pigtail catheter, and the catheter is withdrawn slightly until the tip of the guidewire is leading (Fig. 4.11). The position of the tip of the guidewire within the aortic root can then be controlled by rotation of the pigtail catheter and adjustment of the amount of wire that protrudes. Less wire protruding directs the wire tip more toward the left coronary ostium, whereas more wire protruding directs the wire more toward the right coronary ostium. With the wire tip positioned so that it is directed toward the aortic orifice, the tip of the wire usually quivers in the systolic jet. Wire and catheter are then advanced as a unit until the wire crosses into the left ventricle. If the wire buckles in the sinus of Valsalva instead of crossing the valve, the catheter-wire system is withdrawn slightly and readvanced with or without subtle change in the length of protruding wire or the orientation of the pigtail catheter. Alternatively, some operators prefer to leave the pigtail catheter fixed and move the guidewire independently in attempts to cross stenotic aortic valves. In either case, the wire should be withdrawn and cleaned and the catheter should be double-flushed vigorously every 3 minutes despite systemic heparinization. If promising wire positions are not obtained, the process should be repeated using a different catheter: an angled pigtail or left Amplatz catheter if the aortic root is dilated or a Judkins right coronary catheter if the aortic root is unusually narrow (16). Other catheters have been proposed for this purpose (17), but we have found these standard catheters to suffice in virtually all cases.

When the tip of the guidewire is across the aortic valve, additional wire should be inserted before any attempt is made to advance the catheter itself. Otherwise the catheter may be diverted into a sinus of Valsalva, causing the wire to flip out of the left ventricle. The straight wire should be advanced carefully, since there is a potential (admittedly small in the hypertrophic left ventricle of a patient with aortic stenosis) to perforate the left ventricular wall if the guidewire is ad-

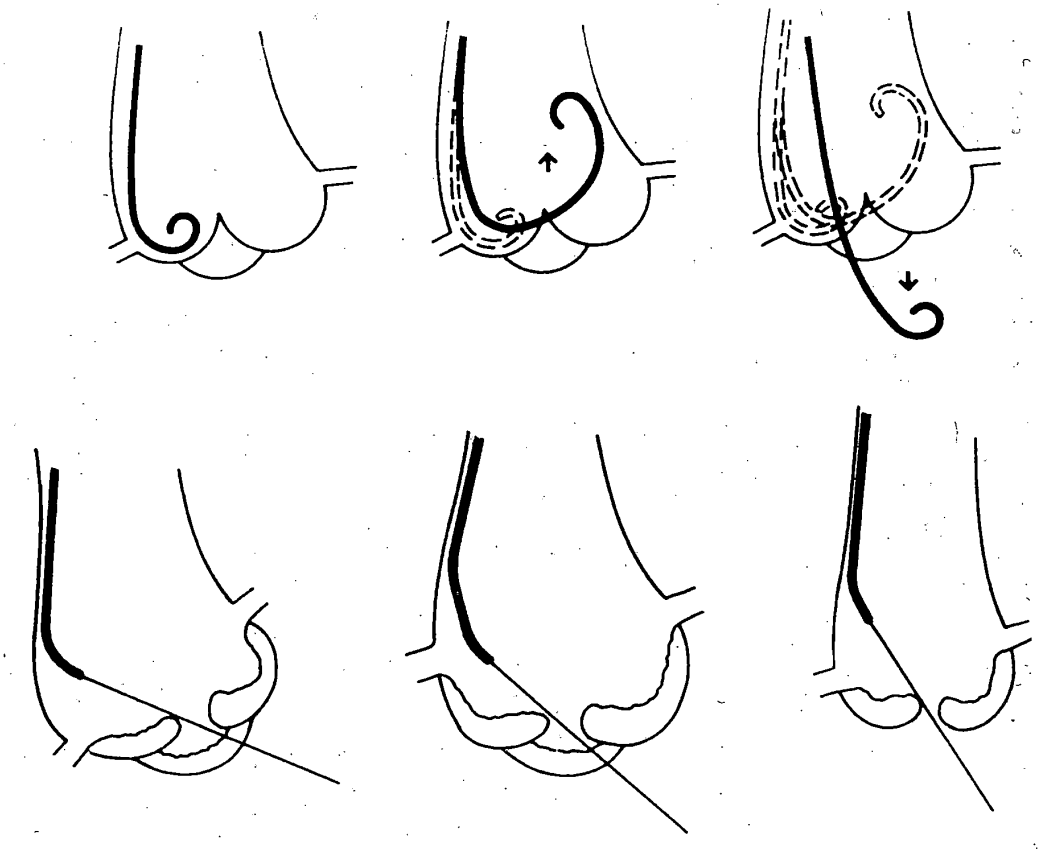


FIG. 4.11. Crossing the aortic valve with a pigtail catheter. **Top row, left:** Although a correctly oriented pigtail catheter will frequently cross a normal aortic valve directly, it may also come to rest in the right or noncoronary sinus of Valsalva. **Top row, center:** Further advancement of the catheter enlarges the loop to span the aortic root and positions the catheter. **Top row, right:** Slow withdrawal causes the catheter to sweep across the aortic orifice and fall into the left ventricle. **Bottom row, left:** To cross a stenotic aortic valve, the pigtail catheter must be led by a segment of straight guidewire. 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 catheter curve and causes the wire to point more toward the left coronary. Once the correct length of wire and the correct rotational orientation of the pigtail catheter have been found, repeated advancement and withdrawal of both the catheter and guidewire as a unit will allow the wire to cross the valve. **Bottom row, center:** In a dilated aortic root, an angled pigtail provides more favorable wire positions. **Bottom row, right:** In a small aortic root, a Judkins right coronary catheter may be preferable.

vanced further when it has become trapped in an endocardial surface feature. Once the catheter is in the left ventricle, the wire is immediately withdrawn and the catheter is aspirated vigorously, flushed, and hooked up for pressure monitoring, so that a gradient can be measured even if the catheter is rapidly ejected from the left ventricle or must be withdrawn because of arrhythmia. When using a left Amplatz catheter to cross a stenotic valve, however, we prefer to cross the valve with a full-exchange-length (260-cm) guidewire. Once the tip of this wire has entered the left ventricle, it is left in position as the Amplatz catheter is removed, and a con-

ventional pigtail catheter is substituted before an attempt is made to measure left ventricular (LV) pressure.

The same approach applies to retrograde catheterization across a porcine aortic valve prosthesis, although it is more common to use a J-tip guidewire to help avoid the area between the support struts and the aortic wall. Ball valves (Starr-Edwards, Baxter-Healthcare, Santa Ana, CA) can be crossed retrograde with this approach, but use of a small (4F or 5F) catheter will minimize the amount of aortic regurgitation resulting from catheter interference with diastolic ball seating. Tilting disc valves (Bjork-Shiley, Shiley, Inc., Irvine, CA; St. Jude Medical, St. Paul, MN; Sulzer Carbomedics, Austin, TX), however, should not be crossed retrograde because of the potential for producing torrential aortic regurgitation, catheter entrapment, or even disc dislodgement, if the catheter passes across the smaller (minor) orifice. Although safe passage through the major orifice may be possible under careful fluoroscopic control (18), we still prefer a transseptal or even apical puncture approach (see later) when it is necessary to enter the left ventricle in a patient who has a tilting disc valve in the aortic position.

Control of the Puncture Site Following Sheath Removal

After the effect of heparin (if used) has been reversed by Protamine or has been allowed to wear off (to an ACT < 160 seconds), the arterial catheter and sheath are removed. The standard way to control the puncture site and promote the formation of a hemostatic plug is to apply firm manual pressure. This is best done using three fingers of the left hand that are positioned sequentially up the femoral artery beginning at the skin puncture. With the fingers in this position, there should be no ongoing bleeding into the soft tissues or through the skin puncture. It should be possible to apply sufficient pressure to obliterate the pedal pulses and then release just enough pressure to allow them to barely return. This pressure is then gradually reduced over the next 10 to 15 minutes, at the end of which time pressure is removed completely. The venous sheath is usually removed 5 minutes after compression of the arterial puncture has begun, with gentle pressure applied over the venous puncture using the right hand. To avoid tying up the catheterization laboratory during this period, the patient is usually taken to a special "holding room" in the catheterization laboratory or back to his hospital bed before the sheaths are removed. If such relocation is to be performed prior to sheath removal, it is important that the sheaths be secured in place (suture, or at least tape) to prevent them from being pulled out during transport.

After procedures using larger arterial sheaths (i.e., PTCA or balloon valvuloplasty), or performed in the setting of thrombolytic agents or IIb/IIIa receptor blockers, more prolonged compression (30 to 45 minutes) is typically required. To avoid fatigue of the operator or other laboratory personnel performing compression, we typically use a mechanical device such as the Compressar (Instromedix, Beaverton, OR) or FemoStop (USCI, Billerica, MA)] to apply similar local pressure. These devices can be equally or even more effective in prolonged holds (19), but we still prefer manual compression for removal of smaller (6F and 7F) sheaths or in patients with peripheral vascular disease or prior peripheral grafting surgery that makes it important to avoid compressive occlusion or flow restriction that may cause arterial occlusion. In every case, however, it should be emphasized that a trained person must be in attendance throughout the compression to ensure that the device is providing adequate control of puncture site bleeding and is not compromising distal perfusion.

After compression has been completed, the puncture site and surrounding area are inspected for hematoma formation and active oozing, and the quality of the distal pulse is assessed before application of a bandage.

It is our policy to keep the patient at bedrest with the leg straight for 4 to 6 hours following percutaneous femoral catheterization (20), with a sandbag in place over the puncture site for the first few hours after catheter removal. In patients

at higher risk for rebleeding (those with hypertension, obesity, or aortic regurgitation), application of a pressure bandage in addition to the sandbag may be of value. Although the patient should be instructed not to move the leg for several hours following the catheterization procedure, the patient does not have to lie flat during this time. Elevation of the head and chest to 30° to 45° by the electrical or manual bed control, without muscular effort by the patient, will greatly increase the patient's comfort and will not increase the risk of local bleeding. The only reason to insist that the patient lie completely flat is if there is significant orthostatic hypotension. Before ambulation and again before discharge, the puncture site should be reinspected for recurrent bleeding, hematoma formation, development of a bruit suggestive of pseudoaneurysm or A-V fistula formation, or loss of distal pulses.

Puncture Closure Devices

The technique described earlier relies on manual or mechanical pressure for initial control of arterial bleeding and then on local hemostasis for ongoing plugging of the arterial puncture site. The potential for ongoing bleeding (with formation of hematoma, false aneurysm, or arteriovenous fistula) has already been described in Chapter 2 and tends to be more common with interventional procedures that require larger sheath size or more aggressive postprocedure antithrombotic therapy. This has prompted the development of a variety of new devices that seek to provide more positive closure of the arterial puncture site (Fig. 4.12). The simplest device (Vasoseal, Datascope, Paramus, NJ) applies a collagen plug in the skin track apposed to outer wall of the femoral artery (21). In randomized

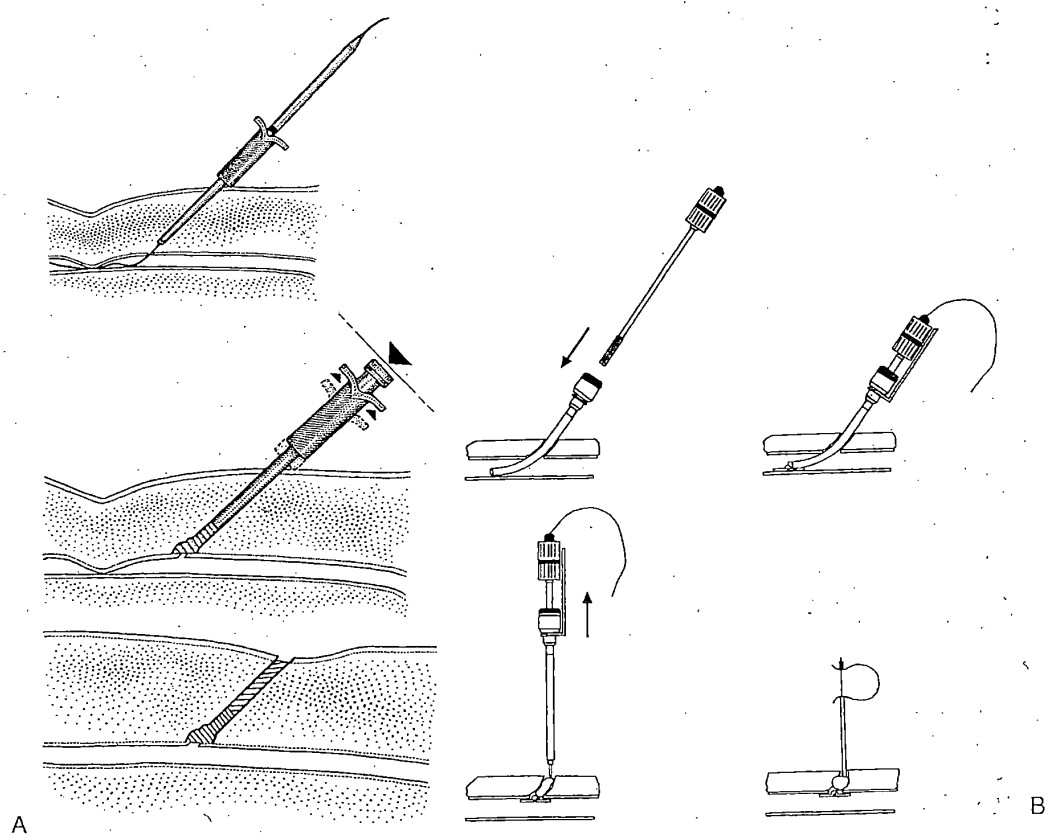


FIG. 4.12. Schematic diagrams of various new devices for the closure of femoral arterial punctures. **A:** The Vasoseal. **B:** The AngioSeal (Kensey-Nash) device. **C:** The Prostar suture device. **D:** The Duet device. (See the text for details.)

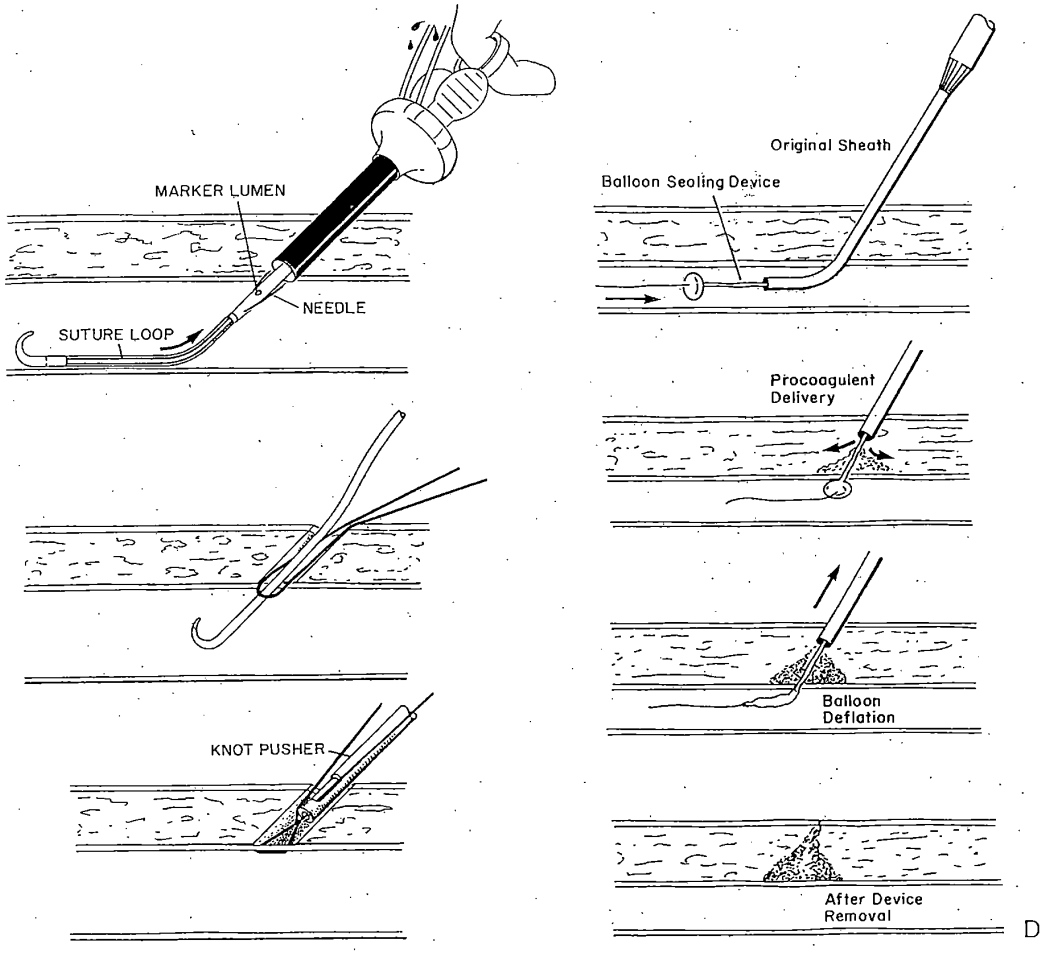


FIG. 4.12. (*continued*)

trials, this device shortens the time to hemostasis (from 17 to 4 minutes) and ambulation (from 19 to 13 hours), without clear benefit in terms of hematoma formation or the need for vascular surgery compared with manual compression. In diagnostic catheterization, it can also accelerate time to ambulation to 1 to 2 hours (22). Next in complexity is the AngioSeal hemostatic puncture closure device (Sherwood, Davis & Geck), which positions a rectangular absorbable "anchor" made of absorbable suture material against the inside wall of the artery and uses an attached suture to winch a small collagen plug down against the outside of the artery (23). In a randomized trial of mostly diagnostic procedures (24), the AngioSeal reduced the time to hemostasis (2.6 versus 15.3 minutes) and ambulation (1 hour versus 4 to 6 hours) compared with manual compression, with a modest decrease in hematoma formation. The Duett device (25) differs in that it uses a liquid procoagulant mixture (thrombin and collagen) that is injected into the soft-tissue track leading from the outside of the artery to the skin. A compliant balloon-on-a-wire is first positioned and inflated within the artery, pulled into contact with the end of the same sheath that was used for the catheterization, and pulled back against the inside of the puncture site to tamponade bleeding. The sheath is then withdrawn roughly 1 cm further so that its end lies outside the vessel lumen, and the sheath sidearm is used to inject the procoagulant

into the soft-tissue tract leading to the outside of the artery. While each of these devices places a great deal of faith in the pro-coagulant properties of its collagen component, the approach of Perclose (Redwood City, CA) relies on the use of a sheathlike device to perform suture-mediated closure of the arterial puncture site. This device has undergone several design changes to improve the ease of delivery, but it still relies on the passage of fine nitinol needles through the margins of the arterial puncture and out through the skin tunnel, where they can be tied to provide surgical hemostasis (26). It shortens the time from the end of the procedure to hemostasis (19 minutes versus 243 minutes) and ambulation (106 minutes for diagnostic and 232 minutes for interventional procedures, versus 4 to 6 hours and 6 to 12 hours, respectively), with a comparably low incidence of major complications (27). If no venous sheath has been placed, some laboratories even allow immediate ambulation after a successful suture-mediated closure.

Given this array of new devices, groin closure devices are being used in most cases in some laboratories. Others, under less pressure to provide early ambulation and same-day discharge, restrict it to patients with an increased risk of bleeding with manual compression or other conditions (back pain, trouble voiding) that make prolonged bedrest undesirable. As these devices continue to evolve and the demand for early ambulation offsets the moderate cost ($100 to $300) of a closure device, they may ultimately replace prolonged local manual or mechanical pressure in the control of postprocedure bleeding from the femoral artery. The conversion to puncture-sealing devices will be accelerated if they can consistently reduce the 1% to 2% incidence of hemorrhagic complications at the arterial puncture site that constitute one of the most common morbidities associated with catheterization from this route. Of course, the success of these puncture-sealing approaches rests on the premise that a single, accurate, front-wall puncture of the common femoral artery has been performed and that favorable conditions prevail within the vessel and the surrounding soft tissue. Each also requires a modest level of skill and training on the part of the operator, and the realization that difficulties encountered in performing a clean closure once the sheath has been removed and wire access has been given up, may increase rather than decrease the incidence of complications requiring vascular surgery or transfusion. In an era of increasingly sophisticated catheter-based therapies, it seems likely that an effective device for definitive closure of the femoral artery puncture site will replace the 50-year-old practice of pressing on the puncture site until the bleeding stops!

Contraindications to Femoral Approach to Left Heart Catheterization

As discussed in Chapter 1, the choice of catheterization approach (femoral or brachial) is usually a function of operator, institution, and patient preference. Because of technical ease, however, data from the 1990 SCA&I registry shows that 83% of diagnostic (and 96% of interventional) catheterizations are performed via the femoral approach (5). In patients with peripheral vascular disease (femoral bruits or diminished lower extremity pulses), abdominal aortic aneurysm, marked iliac tortuosity, prior femoral arterial graft surgery, or gross obesity, however, catheter insertion and manipulation may present technical challenges even for experienced operators. Recognition of these relative contraindications may favor the use of the percutaneous axillary, brachial, radial, or even translumbar aortic approaches (see later). Each laboratory should thus have one or more operators skilled in these alternative percutaneous routes, particularly if no operators skilled in the brachial cutdown approach (see Chapter 5) are available.

Beyond the limitations of access to the central arterial circulation, one important parameter in the selection of a percutaneous access site is the ability to obtain hemostasis after catheter removal. In the femoral arterial entry technique, this is usually obtained easily after removal of a percutaneous arterial catheter, but patients with a wide pulse pressure (e.g., severe aortic incompetence or systemic hypertension), gross obesity, or ongoing anticoagulation have more problems with bleeding after femoral catheterization than do patients without these factors,

particularly if a groin closure device is not used. The vascular complications of percutaneous retrograde arterial catheterization are usually not life-threatening (28–31) and have already been discussed in Chapter 3. In the final analysis, however, there are relatively few patients who absolutely cannot be catheterized from the femoral approach.

ALTERNATIVE SITES FOR LEFT HEART CATHETERIZATION

The techniques described earlier for percutaneous insertion of a femoral catheter also can be used successfully from the axillary, brachial, or radial arteries, or even the lumbar aorta, with the use of an introducing sheath. In certain cases, access to the left heart may be gained by transseptal puncture from the right atrium to the left atrium, or even by direct percutaneous entry via the left ventricular apex. Although these other access sites may utilize the similar needle puncture, guidewire advancement, and sheath insertion skills outlined earlier for the femoral approach, the operator wishing to use one of the alternative percutaneous routes must master the local anatomy, details of maximal allowable catheter size, limitations on catheter selection, techniques for achieving postprocedure hemostasis, and range of complications that may ensue from bleeding or thrombosis at that anatomic location. Individuals interested in mastering one or more of these approaches are referred to the growing body of literature.

Percutaneous Entry of the Axillary, Brachial, Radial Arteries, and Lumbar Aorta

Axillary puncture has long been used as an alternative to femoral entry by the vascular radiologist (32). The patient's hand is brought behind his or her head to expose the axillary fossa, in which the artery can be felt to course. Using local anesthesia and needle puncture and guidewire techniques like those described earlier, the axillary artery is entered over the head of the humerus. The left axillary artery is generally preferred to allow use of preformed Judkins catheters and avoid the brachiocephalic trunk. Effective control of the puncture site after catheter removal is critical, since accumulation of even modest amounts of hematoma around the artery can cause nerve compression (33).

The *brachial* artery is, of course, readily approached by surgical cutdown (see Chapter 5) but may also be approached using percutaneous (needle and guidewire) techniques (34). The antecubital fossa is prepared and anesthetized as for the cutdown approach. A 21-gauge arterial needle, a special 0.021 heavy-duty guidewire, and a 5F or 6F sheath (MicroPuncture set, Cook) can be used to gain access, after which traditional percutaneous catheter techniques are used. Working from the right brachial artery, Amplatz coronary curves are preferred (see Chapter 11). At the end of the procedure, the sheath is removed, and the area is compressed manually. Alternatively, proximal occlusion can be obtained by inflation of a blood pressure cuff, while a gauze pad and a clear intravenous infusion pressure bag is inflated to above systolic pressure over the puncture site (35). Pressure is then released gradually over 20 to 25 minutes. Comparisons of this percutaneous brachial technique to brachial cut-down show a shorter procedure time (without the need for dissection or repair) and no increase in complications, although surgical repair may be needed occasionally (36). This represents a viable approach for outpatient catheterization or an excellent alternative for access in a patient with difficult femoral or iliac anatomy when a Sones-trained angiographer is not available.

The *radial* artery was previously viewed as a site for placement of monitoring lines in the coronary care unit, rather than an access route for cardiac catheterization. Largely through the efforts of champions like Kiemeneij, however, this has been adapted to the performance of diagnostic angiography and many types of percutaneous coronary intervention (including stent placement!) (37,38). The small caliber of this vessel makes the use of small (5F or 6F) catheters mandatory in most patients. In some patients (mostly males) who have larger-caliber radial arteries, 7F or 8F sheaths can be used (39). Liberal use of lidocaine, nitroglycerin, or a calcium

channel blocker through the sheath sidearm may be required to control local spasm that might render the procedure painful and catheter manipulation difficult. Controlling bleeding from the catheterization site at the end of the procedure usually is not difficult, and several "wristband" compression devices are available. Obviously, patients can get up and walk immediately after a radial arterial procedure. Although bleeding is not a problem, radial artery thrombosis occurs in roughly 5%, usually without clinical sequelae in patients for whom a preprocedure Allen test has confirmed adequate perfusion of the hand from the ulnar artery even while the radial artery is compressed firmly. The rapid (immediate) ambulation, availability of stents and other devices that can be used through current large-lumen guiding catheters, and paucity of entry-site complications (40) have made the percutaneous radial the preferred (or the preferred alternative to the femoral artery) approach in many laboratories.

Percutaneous puncture of the lumbar aorta is a technique that has been used by radiologists to study patients with extensive peripheral vascular disease since the early 1980s and was then adapted to the performance of coronary angiography (41). More recently, this approach has even been used for coronary stent placement (42), although the fact that the procedure must be done with the patient prone complicates angiographic views and limits resuscitative efforts. The inability to apply direct pressure over the arterial entry site (the posterior wall of the aorta) also limits aggressive anticoagulation. Because of these negative factors, direct aortic puncture should be considered a last resort for vascular entry.

Transseptal Puncture

With refinements and improvements in techniques for retrograde left heart catheterization, the use of transseptal puncture for access to the left atrium and left ventricle (43,44) had become an infrequent procedure in most adult cardiac catheterization laboratories (45). In these laboratories, transseptal puncture was reserved for situations in which direct left atrial pressure recording was desired (pulmonary venous disease), in which it was important to distinguish true idiopathic hypertrophic subaortic stenosis (IHSS) from catheter entrapment, and in which retrograde left-sided heart catheterization had failed (e.g., due to severe peripheral arterial disease or aortic stenosis) or was dangerous because of the presence of a certain type of mechanical prosthetic valve (e.g., Bjork-Shiley or St. Jude valves). The infrequency with which the procedure was performed made it difficult for most laboratories to maintain operator expertise and to train cardiovascular fellows in transseptal puncture and gave the procedure an aura of danger and intrigue. With the advent of percutaneous mitral valvuloplasty (Chapter 26) and the availability of improved equipment, however, transseptal catheterization has again become a relatively common procedure (46).

The goal of transseptal catheterization is to cross from the right atrium to the left atrium through the fossa ovalis. In approximately 10% of patients, this maneuver is performed inadvertently during right heart catheterization with a woven Dacron catheter because of the presence of a probe-patent foramen ovale, but in the remainder, mechanical puncture of this area with a needle and catheter combination is required to enter the left atrium. Although puncture of the fossa ovalis itself is quite safe, the danger of the transseptal approach lies in the possibility that the needle and catheter will puncture an adjacent structure (i.e., the posterior wall of the right atrium, the coronary sinus, or the aortic root). To minimize this risk, the operator must have a detailed familiarity with the regional anatomy of the atrial septum (Fig. 4.13). As viewed from the feet with the patient lying supine, the plane of the atrial septum runs from 1 o'clock to 7 o'clock. The fossa ovalis is posterior and caudal to the aortic root and anterior to the free wall of the right atrium. The fossa ovalis is located superiorly and posteriorly to the ostium of the coronary sinus and well posterior of the tricuspid annulus and right atrial appendage. The fossa ovalis itself is approximately 2 cm in diameter and is bounded superiorly by a ridge—the limbus.

This anatomy can be distorted somewhat by

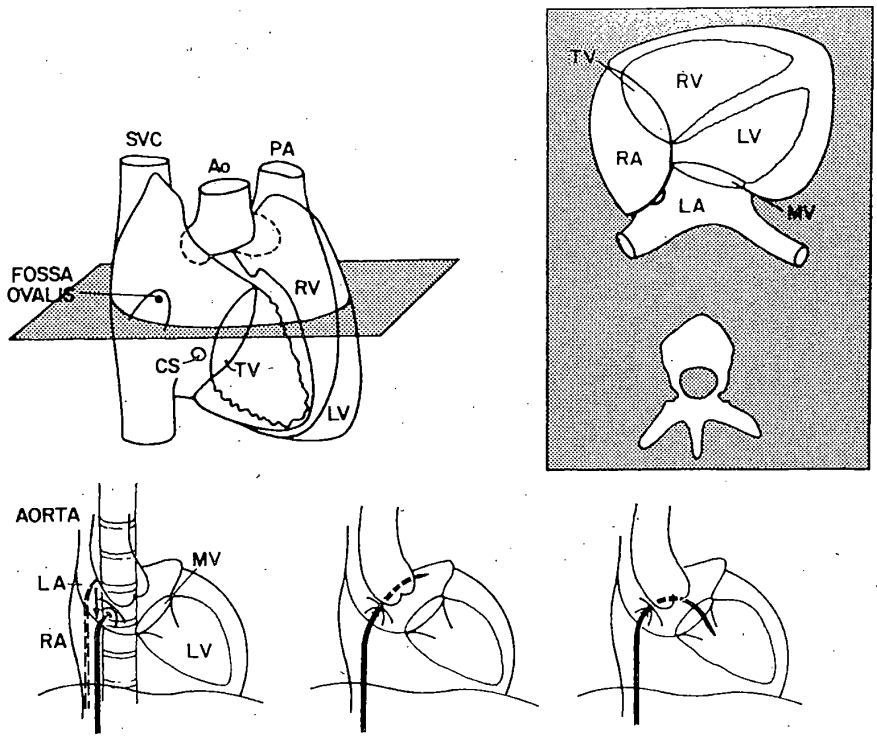


FIG. 4.13. Regional anatomy for transseptal puncture. **Upper left:** The position of the fossa ovalis is shown relative to the superior vena cava (SVC), aortic root (Ao), coronary sinus (CS), and tricuspid valve (TV). **Upper right:** A cross-section through the fossa (looking up from the feet) demonstrating the posteromedial direction of the interatrial septum (bold line) and the proximity of the lateral free wall of the right atrium. **Bottom row:** The appearance of the transseptal catheter as it is withdrawn from the SVC in a posteromedial orientation. As the catheter tip slides over the aortic root (bottom left, dotted position) it appears to move rightward on to the spine. Slight further withdrawal leads to more rightward movement into the fossa (solid position). **Bottom row, center:** Puncture of the fossa with advancement of the catheter into the left atrium. **Right:** Advancement into the left ventricle with the aid of a curved tip occluder. (Redrawn from Ross J Jr. Considerations regarding the technique for transseptal left heart catheterization. *Circulation* 1966;34:391.)

the presence of aortic or mitral valve disease (47). In aortic stenosis, the plane of the septum becomes more vertical, and the fossa may be located slightly more anteriorly. In mitral stenosis, the intraatrial septum becomes flatter with a more horizontal orientation, and the fossa tends to lie lower. Combined with the fact that the septum (and fossa) may then bulge into the right atrium, this makes detailed familiarity with the anatomy even more important when transseptal catheterization is attempted in patients with advanced valvular heart disease. In such patients, intraprocedural transthoracic (48), transesophageal (49), or intracardiac (50) ultrasound may aid in identifying the optimal location for puncture of the intraatrial septum. Alternatively, several algorithms using fluoroscopic landmarks determined by right and left atrial angiography, or the position of a pigtail catheter in posterior (noncoronary) aortic sinus of Valsalva, have been developed to aid localization of the best site for transseptal puncture (51,52) (Fig. 4.14).

Transseptal catheterization is performed only from the right femoral vein. We use a 70-cm curved Brockenbrough needle (USCI, Billerica,

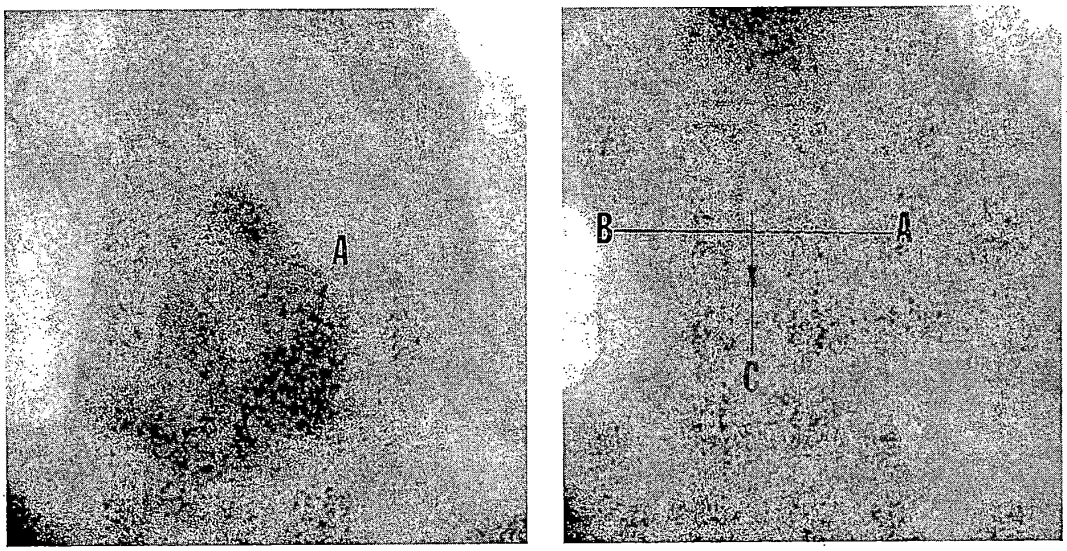


FIG. 4.14. Fluoroscopic landmarks for localizing the fossa ovalis. **Left panel:** As described by Inoue, right atrial injection can be used to locate the upper corner of the tricuspid valve (point *A*), which is marked on the TV monitor. **Right panel:** Continued filming during the levophase fills the left atrium. A horizontal line is drawn from point *A* to the back wall of the left atrium, defining point *B*. That line is divided in half, and a vertical line is dropped to the floor of the left atrium, defining point *C*. The location of the fossa (*x*) is along this vertical line, approximately one vertebral body height above point *C*. When the borders of the left atrium are visible fluorscopically, the position of a pigtail catheter in the noncoronary sinus of Valsalva can be substituted for point *A*, allowing localization of the ideal puncture site without contrast injection (see reference 40). A similar localization scheme (not shown) has been proposed in the 40° RAO projection by Croft and associates (reference 39), using the aortic pigtail and the posterior border of the left atrium. Puncture is made 1 to 3 cm below the midpoint of a line connecting the posterior wall of the aorta to the back wall of the left atrium.

MA) that tapers from 18 gauge to 21 gauge at the tip (Fig. 4.15). The needle is introduced via a matching Brockenbrough catheter or 8F Mullins sheath and dilator combination (53) (USCI) that has been inserted to the superior vena cava over a flexible 0.032-inch, 145-cm J guidewire. Once the wire has been removed and the catheter has been flushed, the Brockenbrough needle is advanced through the catheter, with an obturator (Bing stylet) protruding slightly beyond the tip of the needle to avoid abrasion or puncture of the catheter wall during needle advancement. As the needle and its stylet are advanced through the catheter, the patient may experience a slight pressure sensation due to distortion of the venous structures by the rigid needle. During needle advancement, it is thus essential to allow the needle and its direction indicator to rotate freely so that it may follow the curves of the catheter and venous structures; the hub of the needle should never be grasped and rotated at this point. The progress of the needle tip should be monitored fluoroscopically, looking for any sign of perforation of the catheter by the needle. The stylet is then removed at the diaphragm and the needle hub is connected to a pressure manifold using a stop-cock with a short length of tubing and is carefully flushed. The needle is then advanced to lie just inside the tip of the catheter or sheath, as indicated by measurements made by comparing the distance between the needle flange and the catheter hub, with similar measurements made with a sterile ruler before insertion (Fig. 4.16). Alternatively, current high-

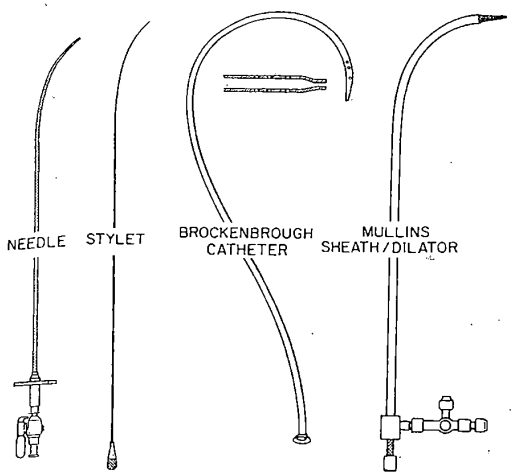


FIG. 4.15. Equipment for transseptal puncture. **Left:** The Brockenbrough needle. **Left center:** Bing stylet. These can be used in conjunction with the following. **Right center:** Traditional Brockenbrough catheter. **Right:** Mullins sheath/dilator system.

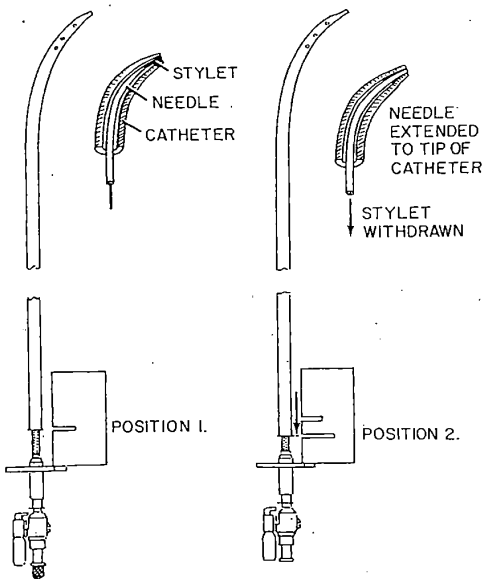


FIG. 4.16. The Brockenbrough system with the needle and stylet inserted into the catheter. Ruler measurement of the distance from the catheter hub to the needle flange is shown with the tip of the stylet at the tip of the catheter (position 1) and with the stylet withdrawn and the needle tip extended to the tip of the catheter (position 2). (Redrawn from Ross J Jr. Considerations regarding the technique for transseptal left heart catheterization. *Circulation* 1966;34:391.)

quality fluoroscopy can be used to visually monitor advancement of the needle to the catheter tip.

The superior vena caval pressure should then be recorded through the needle, with the needle rotated so that the direction indicator points anteriorly. Under continuous fluoroscopic and pressure monitoring, the needle and catheter are then held in constant relationship as they are withdrawn slowly, using both hands. The direction indicator is firmly controlled with the right hand and used to rotate the needle clockwise during this withdrawal from the superior vena cava, until the arrow is oriented posteromedially (4 o'clock when looking from below). As the tip of the catheter enters the right atrium, it moves slightly rightward (toward the patient's left). The needle and catheter are maintained in their posteromedial orientation, and they continue to be withdrawn slowly. As the catheter tip slips over the bulge of the ascending aorta, it again moves rightward to overlie the vertebrae in the anterior projection. Further slow withdrawal maintaining the 4 o'clock orientation will be associated with a third rightward movement as the catheter tip "snaps" into the fossa ovalis. This is confirmed by the fact that advancement will cause the catheter tip to flex slightly (rather than move back up the atrial septum) if its tip is lodged in the fossa. Clear fluoroscopic evidence of fossa engagement is thus essential to successful transseptal puncture.

If the foramen is patent, the catheter may actually cross into the left atrium spontaneously at this point, as indicated by a change in atrial pressure waveform and the ability to withdraw oxygenated blood from the needle. Otherwise, the catheter is advanced slightly to flex its tip against the limbus at the superior portion of the foramen ovale. Once the operator is satisfied with this position, she or he advances the Brockenbrough needle smartly so that its point

emerges from the tip of the catheter and perforates the atrial septum. Successful entry into the left atrium should be confirmed by both the recording of a left atrial pressure waveform and the withdrawal of oxygenated blood or the demonstration of the typical fluoroscopic appearance of the left atrium during a contrast puff through the needle. Once the operator is confident that the needle tip is across the interatrial septum, the needle and catheter are advanced as a unit, a short distance into the left atrium, taking care to control their motion so that the protruding needle does not injure left atrial structures. When the catheter is across the atrial septum, the needle is withdrawn, and the catheter is double-flushed vigorously and connected to a manifold for pressure recording.

The main risk during transseptal catheterization is inadvertent puncture of adjacent structures (the aortic root, coronary sinus, or posterior free wall of the right atrium) rather than the fossa ovalis. As long as the patient is not anticoagulated and perforation is limited to the 21-gauge tip of the Brockenbrough needle (i.e., perforation is recognized and the catheter itself is not advanced), this is usually benign. However, if the 8F catheter itself is advanced into the pericardium or aortic root, potentially fatal complications may occur, underscoring the need for the operator to monitor closely the location of the transseptal apparatus by fluoroscopic, pressure, and oxygen saturation at each stage of the procedure. Damped pressure waveform during attempted septal puncture may indicate puncture into the pericardium or simply incomplete penetration of a thickened interatrial septum. Injection of a small amount of contrast through the needle can be useful in this case by staining the atrial septum, and allowing confirmation of an appropriate position in the left anterior oblique (LAO) and RAO projection before more forceful needle advancement is attempted. If the initial attempt at transseptal puncture is unsuccessful, the operator may wish to repeat the catheter positioning procedure by removing the transseptal needle from the catheter, withdrawing the catheter slightly, and reinserting the 0.032-inch guidewire into the superior vena cava. In general, one should never attempt to reposition the catheter-needle combination in the superior vena cava in any other way, since perforation of the right atrium or atrial appendage is a distinct possibility during such maneuvers.

Once the catheter is safely in the left atrium, additional manipulation may be required to enter the left ventricle. If the tip of the catheter has entered an inferior pulmonary vein (as evident by its projection outside the posterior heart border in the right anterior oblique projection), the left ventricle can be approached by torquing the catheter 180° in a counterclockwise direction so that its tip moves anteriorly as it is withdrawn slightly. As the catheter tip moves anteriorly and downward, further advancement will usually allow it to cross the mitral valve and enter the left ventricle. If not, it may be necessary to insert a curved-tip occluder into the catheter through an O-ring sidearm adapter to tighten the tip curve and facilitate advancement into the ventricle. By converting the Brockenbrough catheter from an end-and-side-hole to a side-hole-only device, the tip occluder also minimizes the chance for left ventricular staining and perforation during contrast ventriculography. However, contrast angiography at 8 to 10 mL/sec for 40 to 50 mL total injection (as with the Sones catheter) can usually be accomplished safely without a tip occluder, if desired. Following the completion of hemodynamic and angiographic evaluation, the Brockenbrough catheter is withdrawn in the usual manner during continuous pressure recording.

The technique for transseptal catheterization using the Mullins sheath (53) is similar, except that care must be taken to advance both the dilator and the 8F sheath into the left atrium without injuring the opposite left atrial wall. Slight counterclockwise rotation and repeated puffs of contrast to define location of the catheter tip may be helpful in this regard. Once the sheath is secure in the left atrium, the needle and dilator are withdrawn and the sheath is flushed carefully. Either a specially curved pigtail catheter (in patients with a normal mitral valve) or a CO_2-inflated balloon flotation catheter (in patients with mitral stenosis) may then be inserted through the sheath and passed into the left ventricle. The current Mullins sheath designs have a sidearm

connection and backbleed valve, allowing ongoing measurement of left atrial pressure around the left ventricular catheter.

Complications of transseptal catheterization are generally infrequent (''needle tip'' perforation, less than 3%; tamponade, less than 1%; and death, less than 0.5%) in experienced hands. This is supported by experience in 1,279 cases from the Massachusetts General Hospital (54), 597 cases from Los Angeles (47), and 500 cases from Taiwan (52). The excellent results in these large series indicate that the technique for transseptal puncture has not been lost (or may even have improved) since its first wave of popularity in the 1960s and 1970s! Because serious complications can occur and are significantly more common early in an operator's experience or in high-risk patients, however, performance of this procedure should be limited to a few operators at each site who can do enough annual procedures to perfect their technique. This is particularly true in patients with distorted anatomy due to congenital heart disease, marked left or right atrial enlargement, significant chest or spine deformity, inability to lie flat, ongoing anticoagulation, or left atrial thrombus/tumor, in whom the technique should generally be avoided.

Apical Left Ventricular Puncture

Historically, a variety of direct puncture techniques were used to enter the cardiac chambers before the introduction of percutaneous left and right heart catheterization. These techniques included transbronchial (55) and transthoracic (56) approaches to the left atrium, the suprasternal puncture technique of Radner (57), and apical left ventricular puncture (58,59). Of these, only the last has survived, albeit as an infrequent (roughly one per year in our laboratory) way to measure left ventricular pressure in a patient where retrograde and transseptal catheterization of the LV are precluded by the presence of mechanical aortic and mitral prostheses.

The site of the apical impulse is located by palpation and confirmed by fluoroscopy of a hemostatic clamp placed at the intended puncture site. Alternatively, the true left ventricular apex can be located using echocardiography (60) and

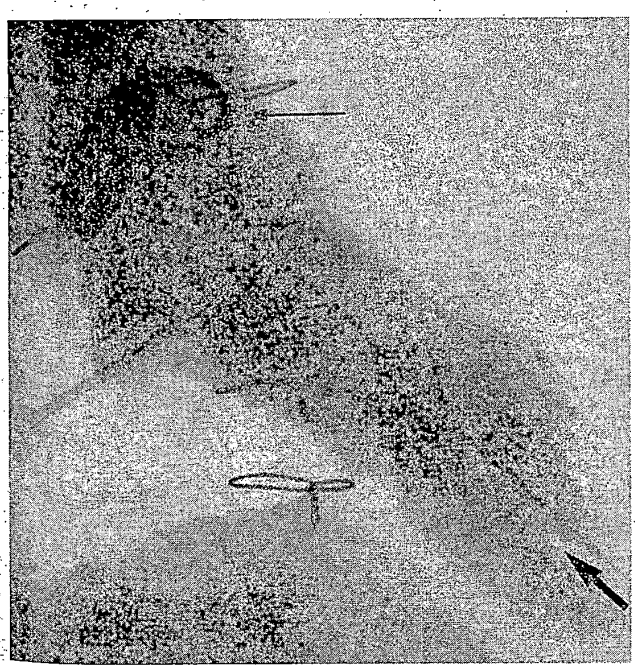


FIG. 4.17. Apical left ventricular puncture. In this patient with Björk-Shiley aortic and mitral valve prostheses *(arrow, upper left)*, percutaneous puncture of the left ventricular apex was performed to allow left ventricular pressure measurement and contrast ventriculography using a 4F angiographic pigtail catheter shown entering the LV apex *(arrow, lower right)*. This catheter was advanced into the left ventricle over a 0.035-inch guidewire, following apical puncture with an 18-gauge thin-wall needle. (See the text for details.)

may be found to lie significantly more lateral than the palpated "apical" impulse in patients with right ventricular enlargement. After liberal local anesthesia, an 18-gauge needle (like that used for internal jugular puncture) is introduced at the apex and directed along the long axis of the left ventricle. This is accomplished by aiming the needle tip roughly toward the back of the right shoulder. Contact with the left ventricular wall can usually be felt as a distinct impulse (and the onset of ventricular premature beats). Sharp advancement of the needle at this point will cause its tip to enter the left ventricular cavity, with pulsatile ejection of blood.

In the technique of Semple (59), an outer Teflon catheter was then advanced over the puncture needle and into the left ventricle (sometimes out through the aortic valve as well). We, however, have preferred the technique in which a 0.035-inch 65-cm-long J guidewire is advanced through the needle and into the left ventricle under fluoroscopic guidance. This allows the advancement of a 4F dilator followed by a 4F pigtail catheter to allow pressure measurement and/or left ventricular angiography (Fig. 4.17).

One series describes excellent results of apical puncture in 102 patients (61). Major complications (tamponade or pneumothorax) occurred in 3%, although tamponade was not seen at all in postoperative patients (who have adhesive pericardium). Other complications of apical puncture can include hemothorax, intramyocardial injection, ventricular fibrillation, as well as pleuritic chest discomfort (approximately 10%) and reflex hypotension due to vagal stimulation (approximately 5%). We thus reserve this technique for patients in whom it is essential to enter the left ventricle and in whom neither retrograde nor anterograde (transseptal) entry of the left ventricle is feasible.

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11
Coronary Angiography

Donald S. Baim and William Grossman

DSB: Harvard Medical School; Center for Innovative Minimally Invasive Therapy, Brigham and Women's Hospital, Boston, Massachusetts 02115.
WG: University of California, San Francisco, School of Medicine, Division of Cardiology, University of California, San Francisco Medical Center, San Francisco, California 94143

Because of progressive evolution in catheterization technique (including better radiographic imaging equipment and better-tolerated contrast media) coupled with the development of effective treatment options for coronary artery disease (i.e., bypass surgery and angioplasty), diagnostic coronary angiography (also called coronary *arterio*graphy) has become one of the primary components of cardiac catheterization. It is estimated that more than 1,200,000 coronary angiographic procedures (roughly 400 per 100,000 population) are performed each year in the United States (1,2) with a procedure-related mortality rate of 0.1%. In each procedure, the objective is to examine the entire coronary tree (both native vessels and any surgically constructed bypass grafts), recording details of coronary anatomy that include the individual pattern of arterial distribution, anatomic or functional pathology (atherosclerosis, thrombosis, congenital anomalies, or focal coronary spasm), and the presence of intercoronary and intracoronary collateral connections. Despite gains in noninvasive techniques such as magnetic resonance angiography (3) and fast computed tomography (fast-CT) (4) as *screening* tests for coronary artery disease (and limited imaging of the proximal coronary arteries in some patients); the use of intravascular ultrasound (see Chapter 19) and angioscopy to define the status of the local vessel wall and luminal surface; and the use of intracoronary pressure and flow measurement technology to assess physiologic significance (see Chapter 18), selective coronary angiography remains the clinical ''gold standard'' for evaluating coronary anatomy. By performing a series of intracoronary injections of contrast agents in carefully chosen angulated views using current high-resolution x-ray imaging (see Chapter 2), it is possible to define all portions of the coronary arterial circulation down to vessels as small as 0.3 mm, free of any artifacts caused by vessel overlap or foreshortening. The performance of high-quality coronary angiography, safely defining each and every coronary stenosis in an optimal view, is an important measure of an operator's skill in cardiac catheterization and is the foundation on which the operator's ability to perform successful coronary interventional procedures rests.

CURRENT INDICATIONS

There are a variety of indications for coronary angiography, which are summarized comprehensively in the most recent set of American College of Cardiology/American Heart Association (AHA/ACC) guidelines on coronary angiography (2). These indications continue to evolve as new applications of catheter-based

therapy are developed, but they are still best summarized by the principle stated by F. Mason Sones: coronary arteriography is indicated when a problem is encountered whose resolution may be aided by the objective demonstration of the coronary anatomy, provided competent personnel and adequate facilities are available and the potential risks are acceptable to the patient and physician.

The most frequent indication is the further evaluation of *patients in whom the diagnosis of coronary atherosclerosis is almost certain*, and in whom anatomic correction by means of coronary artery bypass surgery or transluminal coronary angioplasty is contemplated. Angiographic evaluation of coronary anatomy in such patients provides the crucial information needed to select the most appropriate treatment strategy—catheter intervention (see Chapters 23 through 25), bypass surgery, or medical therapy. Included in this category are patients with *stable angina pectoris* refractory to medical therapy. Recent data suggest that *asymptomatic* patients with noninvasive evidence of myocardial ischemia also benefit from revascularization and therefore are candidates for coronary angiography (5). Another target population is comprised of patients with *unstable angina* (new onset, progressive, or rest pain). Whereas intensive drug therapy (β-blocker, calcium channel blocker, nitrate, heparin, aspirin, one of the newer blockers of the platelet IIb/IIIa receptor) alone may be tried in patients with unstable angina, almost two thirds of such patients come to angiography within 6 weeks after presentation because of ongoing clinical symptoms or a positive exercise test (6). Such patients are candidates for early coronary angiography, particularly if they display indicators (rest pain, electrocardiographic [ECG] abnormalities, heart failure) that place them in the category of Braunwald class II or III unstable angina (7) with high risk of progression to myocardial infarction (MI). In such patients, diagnostic catheterization (with the ability to proceed to coronary intervention during the same procedure, if indicated) should be performed after or concurrent with the initiation of multidrug antianginal therapy. Patients with *acute myocardial infarction* may undergo immediate coronary angiography if primary angioplasty is planned (8) or early angiography (hours to days) if they have recurrent spontaneous or exercise-induced angina after thrombolytic therapy (9). The most recent AHA/ACC guidelines for the management of myocardial infarction (8) extend these indications to potentially include patients with post-MI arrhythmias, congestive heart failure, or an ejection fraction less than 40%; all patients after non–Q wave infarction; and patients in whom ongoing occlusion of the infarct vessel or three-vessel disease is suspected. Coronary angiography is also indicated if clinical evidence of a mechanical defect (ventricular septal defect or papillary muscle rupture) develops in the days after an MI. Routine post-MI coronary angiography in the stable patient without these indications is still advocated by some cardiologists (9), but its value (compared with the conservative strategy outlined here) has not been established.

A second group of indications for coronary angiography concerns patients in whom the *presence or absence of coronary artery disease is unclear* (2). This includes patients with troublesome chest pain syndromes but ambiguous noninvasive test results, patients with unexplained heart failure or ventricular arrhythmias, survivors of out-of-hospital cardiac arrest (10), patients with suspected or proven variant angina (11), and patients with risk factors for coronary artery disease who are being evaluated for major abdominal, thoracic, or vascular surgery (12). This category also includes patients scheduled for correction of congenital or valvular pathology. Patients with congenital defects such as tetralogy of Fallot frequently have anomalies of coronary distribution that may lead to surgical complications if unrecognized (13), whereas patients older than 45 years of age with valvular disease may have advanced coronary atherosclerosis without clinical symptoms. Although younger patients with valvular disease are commonly operated on without prior coronary angiograms, given the extraordinary low risk of diagnostic catheterization and the potential benefit of knowing the coronary anatomy, most surgical centers believe it is best to perform a preoperative diagnostic catheterization to identify (and

then correct) significant coronary lesions, so as to provide the best and safest outcome during concurrent valve replacement (14).

Finally, coronary angiography is frequently performed when a patient develops *recurrent angina after coronary intervention* (to detect and treat restenosis; see Chapter 27) *or after bypass surgery* (to detect vein graft failure, which might require catheter intervention or reoperation). Routine follow-up angiography 6 months after catheter intervention is not indicated clinically but may play an important role in the research evaluation of new technologies or drug therapies targeted at reducing restenosis (15).

GENERAL ISSUES

The initial attempts to perform coronary angiography used nonselective injections of contrast medium into the aortic root to simultaneously opacify both left and right coronary arteries, recording the angiographic images on conventional sheet film (16). To improve contrast delivery into the coronary ostia, some early investigators employed transient circulatory arrest induced by the administration of acetylcholine or by elevation of intrabronchial pressure, followed by occlusion of the ascending aorta by a gas-filled balloon and injection of the contrast bolus. Although nonselective aortic root injection is still used occasionally today to evaluate ostial lesions, anomalous coronary ostia, or coronary bypass grafts, intentional circulatory arrest is no longer practiced, and the nonselective technique has largely been replaced by selective coronary injection using specially designed catheters advanced from either the brachial or the femoral approach.

In most patients, successful coronary angiography can be performed by either the brachial cutdown or the percutaneous approach (from the femoral, brachial, or radial artery), leaving the choice up to physician and patient. Data from the Society for Cardiac Angiography and Intervention in 1990 (17) show that the percutaneous femoral approach was used in 83% of cases. The brachial approach, either by percutaneous (see Chapter 4) or cutdown (see Chapter 5) entry, may offer a selective advantage in patients with severe peripheral vascular disease or known abdominal aortic aneurysm. In either case, it is important for the catheterization team to meet the patient before the actual procedure to evaluate the best approach to catheterization, to gain an appreciation of the clinical questions that need to be answered by coronary angiography, to uncover any history of adverse reaction to medications or organic iodine compounds, and to explain the procedure in detail.

Although coronary angiography was traditionally performed as an inpatient procedure, in the 1990s outpatient protocols were developed for diagnostic catheterization in low-risk patients (2,18,19). This includes younger patients with relatively stable symptoms and few other comorbidities (e.g., heart failure, valve disease, renal insufficiency, peripheral vascular disease) who live within a 1-hour drive from the cardiac catheterization facility. More than half of the patients referred for coronary angiography may be suitable for such a procedure, which in each case may save up to $900 in hospital charges (but only roughly $200 in true costs), compared with inpatient catheterization (20,21). Outpatient procedures are performed identically to the technique described herein, using either the brachial or the femoral approach. After a percutaneous femoral procedure, at least 2 hours of bedrest (4 to 6 hours in many institutions) is required, unless a puncture-sealing device is employed.

In either inpatient or outpatient coronary angiography, preparation for catheterization should include proscription of oral intake except for medications and limited quantities of clear liquids over the 6 to 8 hours before catheterization, a baseline 12-lead ECG, and a suitable sedative premedication (usually diazepam, 5 to 10 mg PO, and diphenhydramine, 50 mg PO) administered on call to the catheterization laboratory. We do not routinely premedicate patients with either atropine or nitroglycerin, although both are immediately on hand if needed.

THE FEMORAL APPROACH

As described in Chapter 4, the femoral approach to left-sided heart catheterization involves insertion of the catheter either directly

over a guidewire or through an introducing sheath. Systemic anticoagulation (heparin, 3,000 to 5,000 units at the time of sheath introduction) is used in many laboratories (2), although others now omit heparin in brief diagnostic procedures. A series of preformed catheters are employed, starting with a pigtail catheter for left ventriculography, followed by separate catheters (either Judkins or Amplatz shapes) for cannulation of the left and right coronary arteries. Coronary catheters are available in 5F, 6F, 7F, or 8F endhole designs that taper further near the tip. They may be constructed of either polyethylene (Cook Inc., Bloomington, IN) or polyurethane (Cordis Corporation, Miami, FL, and USCI, Billerica, MA) and contain either steel braid, nylon, or other reinforcing materials within the catheter wall to provide the excellent torque control needed for coronary cannulation. In the 1970s, 8F catheters predominated, because they provided excellent torque control and permitted rapid contrast delivery. In the 1980s, improvements in the design of 7F catheters allowed for a comparable lumen diameter in standard 8F catheters, making them the standard in most laboratories. Smaller (6F and even 5F) coronary angiographic catheters are now available with technology similar to that used in guiding catheters to provide thinner catheter walls and larger lumens (6F lumens up to 0.064 inches), exceeding the lumen size once available in 8F diagnostic catheters (22). We now use such 6F catheters for all of our routine diagnostic procedures. Coronary catheters used for native coronary injection via the femoral or brachial approach are shown in Fig. 11.1.

Insertion and Flushing of the Coronary Catheter

The desired catheter is inserted into the femoral sheath and advanced to the level of the left mainstem bronchus over the guidewire. Alternatively, the tip of the coronary angiography catheter may be advanced "around the arch" and into the ascending aorta before the guidewire is re-

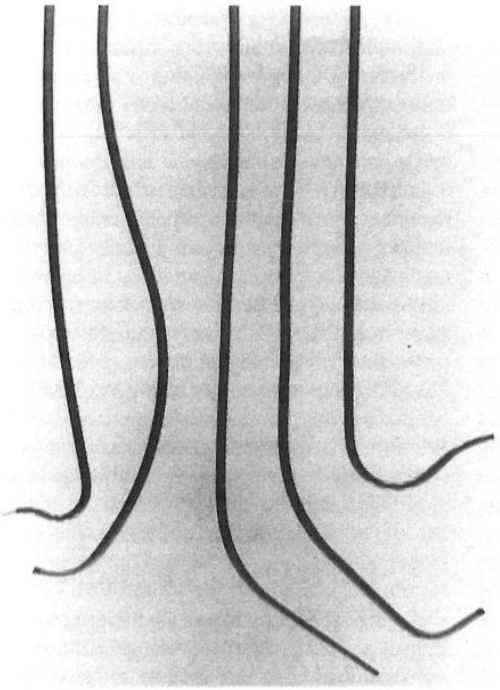


FIG. 11.1. Types of catheters currently in wide use for selective native coronary angiography *(left to right)*: Amplatz right, Judkins right, Sones, Judkins left, and Amplatz left.

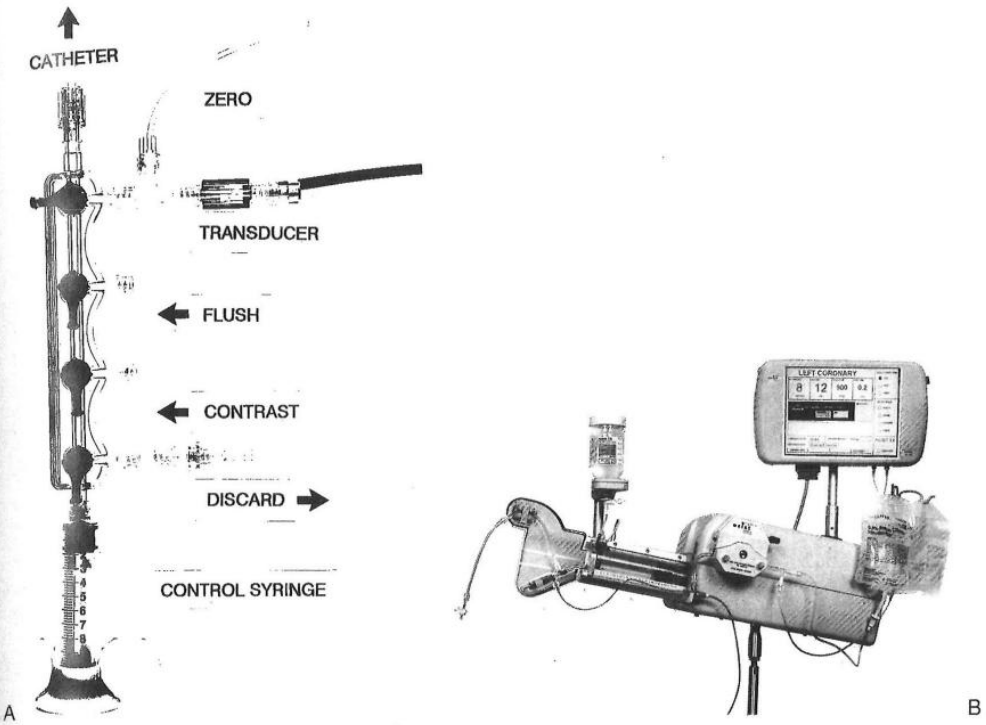


FIG. 11.2. A: Four-port coronary manifold. This manifold provides a closed system with which blood can be withdrawn from the catheter and discarded. The catheter can be filled with either flush solution or contrast medium, and the catheter pressure can be observed, all under the control of a series of stopcocks. The fourth port is connected to an empty plastic bag and is used as a discard port (for blood from the double flush, air bubbles) so that the syringe need not be disconnected from the manifold at any time during the procedure. Attachment of the transducer directly to the manifold allows optimal pressure waveform fidelity (see Chapter 7), and the fluid-filled reference line allows zeroing of the transducer to midchest level. **B:** The Bracco-Squibb Acist device consists of a contrast-filled power injector, controlled by a sterile pneumatic actuator to deliver contrast in amounts and rates up to the limits preprogrammed on the digital panel. A power flushing system and a pressure transducer are also included, duplicating many of the functions of the traditional four-port manifold.

moved. This may reduce snagging of the catheter tip on aortic wall irregularities, but it places greater emphasis on the initial catheter flushing. After removal of the guidewire, the catheter is attached to a specially designed manifold system which permits the maintenance of a ''closed system'' during pressure monitoring, catheter flushing, and contrast agent administration (Fig. 11.2). The catheter is immediately double-flushed: blood is withdrawn and discarded, and heparinized saline flush is injected through the catheter lumen. Difficulty in blood withdrawal suggests apposition of the catheter tip to the aortic wall, which can be rectified by slight advancement or rotation of the catheter until free blood withdrawal is possible. The lumen of the introducing sheath should also be flushed immediately before and after each catheter insertion and every 5 minutes thereafter, to prevent the encroachment of blood into the sheath. Alternatively, the sidearm of the sheath may be connected to a 30 mL/hr continuous-flow regulator (Intraflow II, Abbott, King of Prussia, PA). Once the catheter has been flushed with saline solution, tip pressure should

be displayed on the physiologic monitor at all times (except during actual contrast injections). Next, the catheter lumen should be gently filled with contrast agent under fluoroscopic visualization, avoiding selective contrast administration into small aortic branches. Filling with contrast results in slight attenuation of high-frequency components in the aortic pressure waveform, whose new shape should be carefully noted. Any alteration in that waveform during coronary angiography (see next section) may signify an ostial coronary stenosis or an unfavorable catheter position within the coronary artery. Once these measures are completed, the coronary angiographic catheter is advanced into the aortic root in preparation for selective engagement of the desired coronary ostium.

Damping and Ventricularization of the Pressure Waveform

A fall in overall catheter tip pressure (damping) or a fall in diastolic pressure only (ventricularization) during catheter engagement in a coronary ostium indicates restriction of coronary inflow (Fig. 11.3). The catheter tip may have been inserted into a proximal coronary stenosis or may have an adverse catheter lie that places it against the coronary wall. If either of these phenomena is observed, the catheter should be withdrawn into the aortic root immediately until the operator can analyze the situation further. The catheter may be reengaged and a cautious small-volume contrast injection made to further clarify the situation. It may disclose a proximal occlusion of the vessel, against which the tip of the coronary catheter is resting, in which case a cine run should be performed to document this finding. The test injection may also indicate ostial stenosis through absence of reflux into the aortic root or retention of the injected contrast material in the proximal and middle vessel. Along with damping or ventricularization of the pressure waveform, this indicates that the catheter tip is severely restricting or occluding ostial

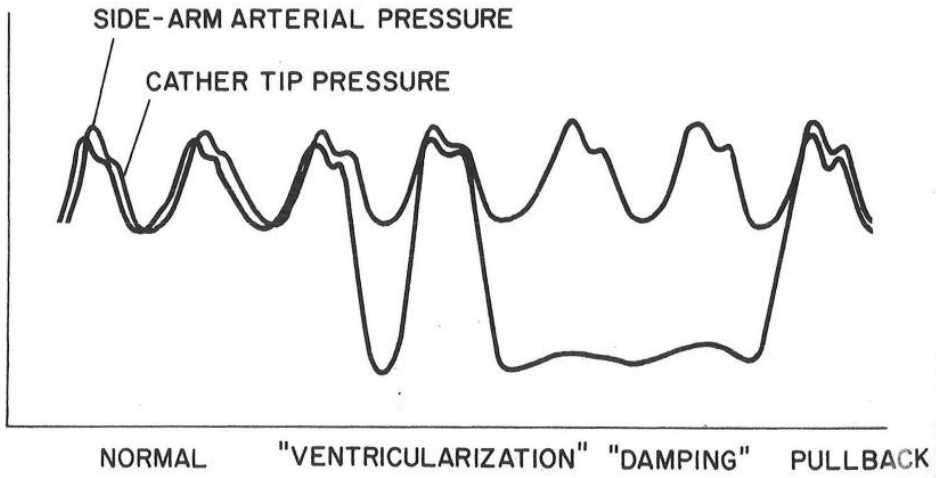


FIG. 11.3. Pressure tracings as recorded during coronary angiography. Except for its earlier phase and slightly lower systolic pressure, catheter tip pressure should resemble the pressure waveform simultaneously monitored by way of the femoral sidearm sheath or other arterial monitor (e.g., radial artery). In the presence of an ostial stenosis or an unfavorable catheter position against the vessel wall, the waveform shows either ventricularization (in which systolic pressure is preserved but diastolic pressure is reduced) or frank damping (in which both systolic and diastolic pressures are reduced). In either case, the best approach is to withdraw the catheter immediately until the waveform returns to normal and to attempt to define the cause of the problem by nonselective injections in the sinus of Valsalva. Alternatively, a catheter equipped with side-holes near the tip may be used to provide ongoing coronary perfusion.

inflow. In this case, a cautious injection may be documented on cine, with immediate removal of the catheter at the end of the cine run to restore antegrade flow. Continuing to inject and film as the catheter is removed from the ostium may capture a single frame or frames that show the ostial lesion clearly. Another approach is to perform a nonselective injection into the sinus of Valsalva in an appropriate view (one that displays the ostium of the vessel in question with no overlap by the sinus of Valsalva) to confirm the presence of an ostial stenosis. On occasion, an end-hole diagnostic catheter may be exchanged for an end- and side-hole angioplasty guiding catheter to overcome damping by preserving antegrade flow into the side-holes, through the lumen of the catheter, and into the coronary artery, even though the catheter tip may be obstructing entry of blood into the ostium itself (see Cannulation of the Right Coronary Ostium). *Vigorous injection despite a damped or ventricularized pressure waveform should be avoided, because it predisposes to ventricular fibrillation or dissection of the proximal coronary artery with major ischemic sequelae*. Such a dissection is manifested by tracking of contrast medium down the vessel over the course of the injection and by failure of contrast to clear on fluoroscopy after the injection is terminated. Prompt consideration of repair by catheter-based intervention or bypass surgery should be considered if creation of such a ''dye stain'' is associated with impeded antegrade coronary flow and signs of myocardial ischemia.

Cannulation of the Left Coronary Ostium

With the Judkins technique it is usually easy to engage the left coronary ostium. As Judkins himself stated, ''No points are earned for coronary catheterization—the catheters know where to go if not thwarted by the operator'' (23). If a left Judkins catheter with a 4-cm curve (commonly referred to as a JL4) is simply allowed to remain *en face* as it is advanced down into the aortic root, it will engage the left coronary ostium without further manipulation in 80% to 90% of patients (Fig. 11.4). Engagement should take place with the arm of the catheter traversing the ascending aorta at an angle of approximately 45°, the tip of the catheter in a more or less horizontal orientation, and no change in the pressure waveform recorded from the catheter tip.

In patients with a widened aortic root due to aortic valve disease or long-standing hypertension, the 4-cm left Judkins curve may be too short to allow successful engagement. In such a case, the catheter arm may lie almost horizontally across the aortic root with the tip pointing vertically against the roof of the left main artery, or it may even refold into its packaged shape during advancement into the aortic root (Fig. 11.4*D*). In this case, a left Judkins catheter with a larger curve (JL4.5, JL5, or even JL6) curve should be selected. In the long run, changing to a larger catheter under these circumstances may save time compared with persevering in trying to make an unsuitable catheter work. In the occasional patient with a short or narrow aortic root (usually a younger female, particularly if of short stature), even the 4-cm Judkins curve may be too long. When brought down into the aortic root, the catheter arm may lie almost vertically with the tip pointing downward, below the left coronary ostium. The left ostium may still be engaged, by pushing the catheter down into the left sinus of Valsalva for approximately 10 seconds to tighten the angle on the catheter tip and then withdrawing the catheter slowly. Having the patient take a deep breath during this maneuver also helps by pulling the heart into a more vertical position to assist in engagement of the left ostium. The most satisfactory approach, however, is to exchange for a JL3.5 catheter with a shorter curve.

On the rare occasions when the left coronary ostium lies ''out of plane'' (typically high and posterior), limited counterclockwise rotation of the left Judkins catheter in the right anterior oblique (RAO) projection may help orient the catheter's tip posteriorly and facilitate engagement. Too much rotation of this catheter, however, may result in a refolded catheter that requires guidewire reinsertion to straighten. In this case, it may be helpful to step up to the next larger Judkins curve. Alternatively, some opera-

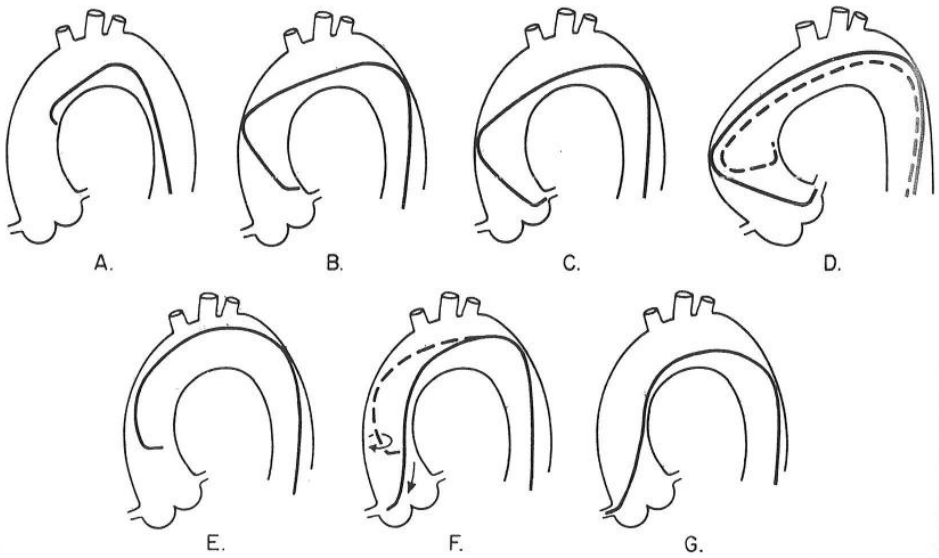


FIG. 11.4. Judkins technique for catheterization of the left and right coronary arteries as viewed in the left anterior oblique (LAO) projection. In a patient with a normal-size aortic arch, simple advancement of the JL4 catheter leads to intubation of the left coronary ostium (**A**, **B**, and **C**). In a patient with an enlarged aortic root **(D)** the arm of the JL4 may be too short, causing the catheter tip to point upward or even flip back into its packaged shape *(dotted line)*. A catheter with an appropriately longer arm (a JL5 or JL6) is required. To catheterize the right coronary ostium, the right Judkins catheter is advanced around the aortic arch with its tip directed leftward, as viewed in the LAO projection, until it reaches a position 2 to 3 cm above the level of the left coronary ostium **(E)**. Clockwise rotation causes the catheter tip to drop into the aortic root and point anteriorly **(F)**. Slight further rotation causes the catheter tip to enter the right coronary ostium **(G)**.

tors prefer to switch to a left Amplatz catheter (Fig. 11.1); these are available in progressively larger curves—AL1, AL2, AL3, and AL4. Amplatz catheters (24) are more tolerant of rotational maneuvering and allow easy engagement of left coronary ostia that lie out of the conventional Judkins plane, as well as subselective engagement of the left anterior descending (LAD) and circumflex coronary arteries in patients with short left main coronary segments or separate left coronary ostia. The left Amplatz is advanced around the arch, oriented toward the left coronary ostium (Fig. 11.5). The tip of the catheter usually comes to rest in the sinus of Valsalva below the coronary ostium. As the catheter is advanced further, the Amplatz shape causes the tip of the catheter to ride up the wall of the sinus until it engages the ostium. At that point, slight withdrawal of the catheter causes deeper engagement of the coronary ostium, whereas further slight advancement causes paradoxic retraction of the catheter tip.

Cannulation of the Right Coronary Ostium

The Judkins technique for engaging the right coronary ostium requires slightly more catheter manipulation than does cannulation of the left coronary ostium (16,23). After being flushed and filled with contrast medium in the descending aorta (with the catheter tip directed anteriorly to avoid injection into the intercostal arteries), the right Judkins catheter with a 4-cm curve (JR4) is brought around the aortic arch with the tip facing inward until it comes to lie against the right side of the aortic root with its tip aimed

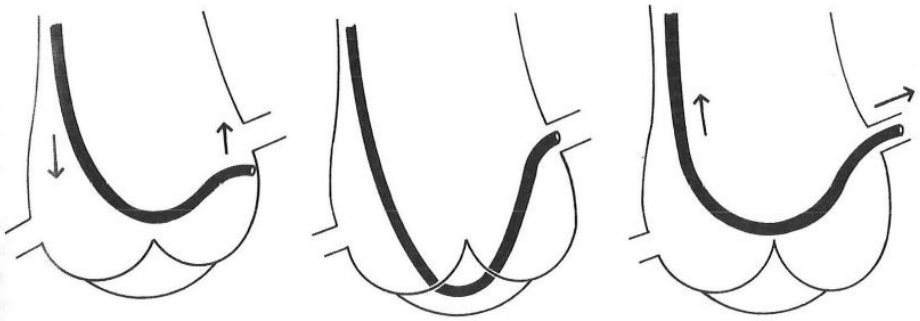


FIG. 11.5. Catheterization of the left coronary artery with an Amplatz catheter. The catheter should be advanced into the ascending aorta with its tip pointing downward, so that the terminal catheter configuration resembles a diving duck. As the Amplatz catheter is advanced into the left sinus of Valsalva, its tip initially lies below the left coronary ostium *(left)*. Further advancement causes the tip to ride up the aortic wall and enter the ostium *(center)*. Subsequently, slight withdrawal of the catheter causes the tip to seat more deeply in the ostium *(right)*.

toward the left coronary ostium (Fig. 11.4). In a left anterior oblique (LAO) projection, the operator slowly and carefully rotates the catheter clockwise by almost 180° to engage the right coronary artery. The tip of the right Judkins catheter tends to drop more deeply into the aortic root when the catheter is rotated toward the right ostium, as the tertiary curve rotates into alignment with the top of the aortic arch. To compensate for this effect, the operator must either begin the rotational maneuver with the tip 2 to 3 cm above the coronary ostium or withdraw the catheter slowly during rotation. Care must be taken to avoid "overrotation" of the catheter, which tends to cause undesirably deep engagement of the right coronary artery. To avoid this common technical error, the operator must not continue to apply clockwise torque when the tip of the catheter is "stuck" in the aortic root, and should be prepared to apply a small amount of counterclockwise torque immediately as the catheter enters the ostium.

Catheters with smaller (3.5-cm) or larger (5- or 6-cm) Judkins curves or right Amplatz catheters (AR1 or AR2) may be of value if aortic root configuration and proximal right coronary anatomy make engagement difficult. One such situation occurs when the right coronary ostium lies high and anterior, usually above the commissure of the left and right aortic valve leaflets but occasionally above the left sinus itself. In that case, a left Amplatz catheter (either AL0.75 or AL1) may be required to make contact with the aortic wall at the location of the ostium. Damping and ventricularization are far more common in the right coronary artery than in the left. It may be caused by (a) the generally smaller caliber of the vessel, (b) ostial spasm around the catheter tip, (c) selective engagement of the conus branch, or (d) true ostial stenosis. These problems in right coronary artery engagement can usually be elucidated by nonselective injections into the right sinus of Valsalva or cautious injections in the damped position with immediate postinjection withdrawal of the catheter. As mentioned earlier, a 6F or 7F angioplasty guiding catheter with side-holes near the tip may be used to allow uninterrupted coronary perfusion between contrast injections, if necessitated by true ostial or proximal right coronary disease.

Cannulation of Saphenous Vein Grafts

Despite the high initial rate of anginal relief after bypass surgery, 3% to 12% of saphenous vein grafts occlude due to thrombosis within the first month. Additional veins occlude between 1 month and 1 year after surgery due to exaggerated neointimal hyperplasia. By far the dominant failure mode of saphenous vein graft failure be-

yond 1 year—accounting for up to 50% graft closure by 7 years—is diffuse graft atherosclerosis (25). For these reasons, an increasing number of patients develop recurrent angina after prior bypass surgery, accounting for more than 20% of the diagnostic procedures in our laboratory.

The proximal anastomosis is placed on the right or left anterior aortic surface, several centimeters above the sinuses of Valsalva. Because many surgeons resist the practice of placing radiopaque markers on the proximal graft (26), the operator usually must rely on the surgeon's operative report or diagram and knowledge of surgical practice in the institution. The operative report should be obtained before elective angiography on any patient with prior bypass surgery, but is absolutely essential for patients who underwent their operation at another medical center (where local preference may include practices such as anastomosis to the right-*posterior* surface of the aorta; see later discussion). It can be quite frustrating to embark on coronary angiography in such a patient without a detailed graft map or operative note in hand.

Most commonly, *grafts to the left coronary artery* arise from the left anterior surface of the aorta, with grafts to the LAD coronary artery originating somewhat below grafts to the circumflex system. Some surgeons prefer to route grafts to the circumflex through the transverse sinus behind the heart, in which case the circumflex graft may originate from the posterior surface of the aorta. *Grafts to the right coronary artery* (or the distal portions of a dominant circumflex) usually originate from the right anterior surface of the aorta, above and somewhat behind the plane of the native right coronary ostium. We usually use the right Judkins (JR4) or Amplatz (AL1) catheter to engage anterior (i.e., left) coronary grafts. Special left coronary bypass, internal mammary, or hockey-stick catheters may be required for left grafts that originate with an upward trajectory (Fig. 11.6). For downward-pointing right coronary artery grafts, we prefer a soft catheter with no primary curve (a multipurpose, Wexler, or JR3.5 short-tip catheter), which provides better alignment with the proximal portion of the graft and therefore better opacification. The Wexler catheter can also be used for grafts originating from the left or posterior surface of the aorta, because its tip remains in contact with the aortic wall. Once the ostium has been selected, the shaft of this catheter may be rotated or the tip may be flexed to bring it into alignment with the proximal graft.

If no markers have been provided, the catheter tip should be oriented against the appropriate aortic wall and slowly advanced and then withdrawn until its tip "catches" in a graft ostium. The graft is injected in multiple projections that show its origin, shaft, distal anastomosis, and the native vessels beyond the anastomosis. This process must then be repeated until all graft sites have been identified. Grafts should not be written off as occluded unless a clear "stump" is demonstrated. If the myocardial territory supplied by a graft assumed to be occluded is still contracting and there is no evident native or collateral blood supply to that territory, there must be some "visible means of support," which may be a missed graft! It may be valuable to perform an aortogram in an appropriate view to try to demonstrate flow in and locate the origin of such a missed graft. The emergence of effective therapies for focal lesions in vein grafts has placed a premium on being able to find and fix such diseased grafts before they occlude (Fig. 11.7; see Chapters 23 through 25).

Internal Mammary Cannulation

Based on their superior demonstrated 10-year patency, the left and right internal mammary (also known as internal *thoracic*) arteries have become the conduits of choice. More than 90% of current elective bypass procedures involve placement of at least one internal mammary graft. Successful cannulation (27) requires knowledge of the left subclavian and brachiocephalic trunk as well as the right subclavian arteries, as shown in Fig. 11.8*A*. It is also important to understand some of the common anatomic variants in the internal mammary artery, including more proximal origin in the vertical portion of the subclavian, or origin as a common vessel with the thyrocervical trunk. Although uncommon, these grafts can develop significant

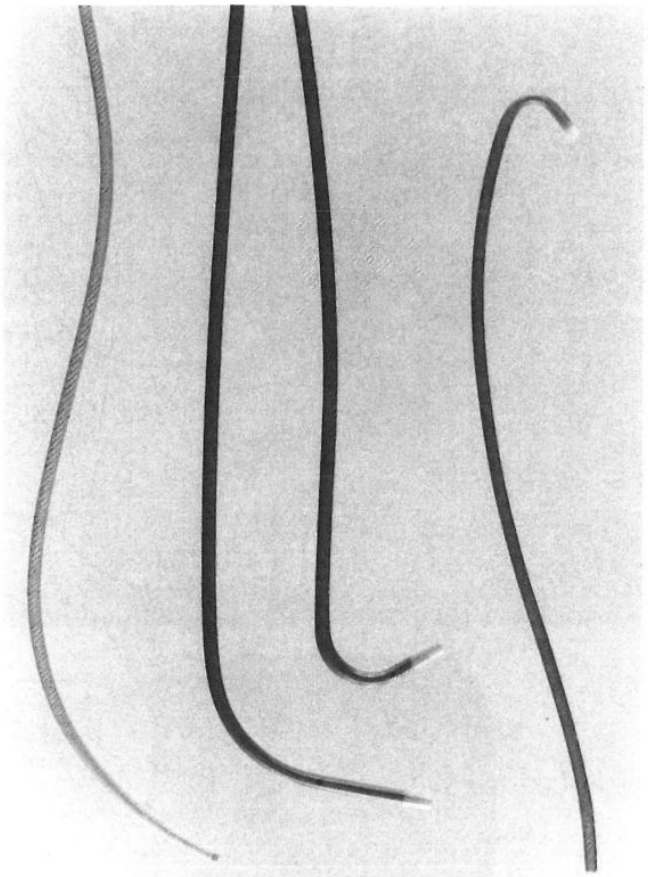
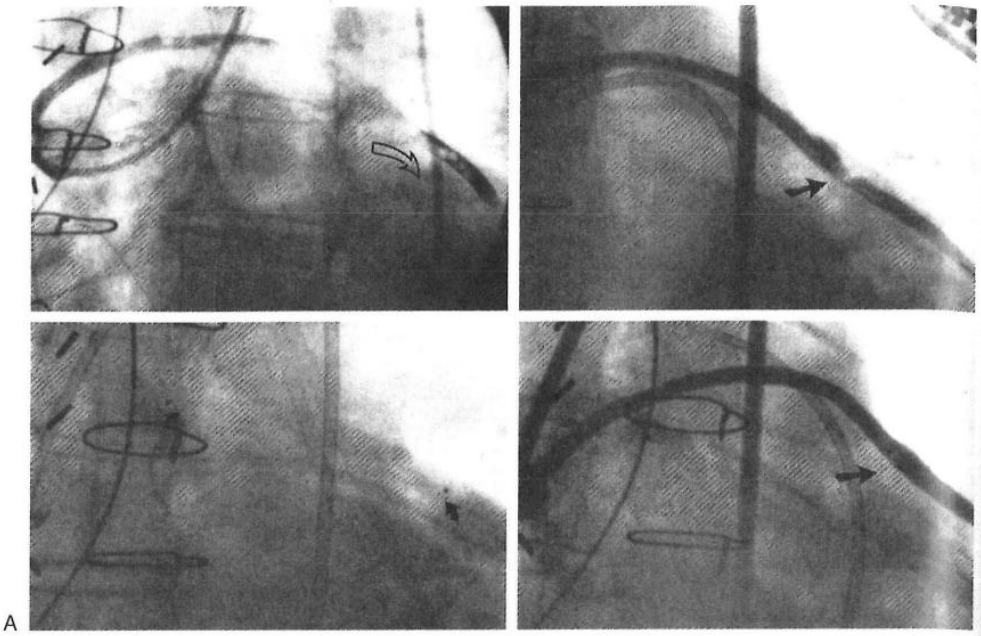


FIG. 11.6. Catheters used for bypass graft angiography. Although the right Judkins or Amplatz catheters can be used for many anterior takeoff vein grafts, catheters with the following shapes may be useful (left to right): Wexler, multipurpose, hockey-stick shape, and internal mammary.

lesions, making it important to evaluate such grafts during any postbypass catheterization. In patients with early recurrence of angina (within the first 6 months after surgery), the most common lesion is located at the distal mammary-coronary anastomosis. It is usually caused by local intimal hyperplasia rather than atherosclerosis and responds well to balloon angioplasty (see Chapter 23). Flow-limiting "kinks" may also be present in the midgraft, and ostial lesions at the origin of the internal mammary from the subclavian may also occur. Years after bypass surgery, significant lesions may develop in the native coronary artery beyond the internal mammary touchdown. In addition to establishing the patency of the internal mammary itself, it may also be important to look for large nonligated side branches that may divert flow from the coronary circulation, and whose occlusion (on occasion) may be required for angina relief (28). It is also important to look for stenoses in the subclavian artery before the takeoff of the internal mammary that may compromise the inflow to the graft and thereby cause myocardial ischemia



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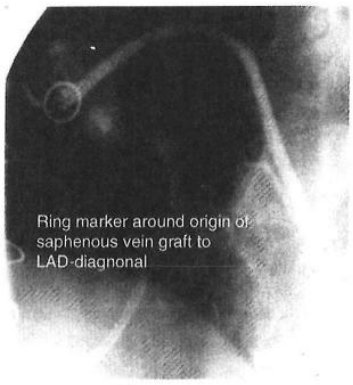


B

FIG. 11.7. A: Sample of saphenous vein graft angiography, showing an occluded graft to the circumflex, filled with thrombus *(upper left, open arrow)*. A drug-infusion catheter (Tracker, Target Therapeutics) was placed *(lower left, curved arrow)* and used to administer urokinase (50,000 IU/hr) overnight. The following morning *(upper right)*, the thrombus had been dissolved, revealing the underlying ulcerated culprit lesion. This was treated with a single Palmaz-Schatz coronary stent *(lower right)*, reestablishing full patency. **B:** Saphenous vein graft with origin localized by ring marker implanted at the time of surgery.

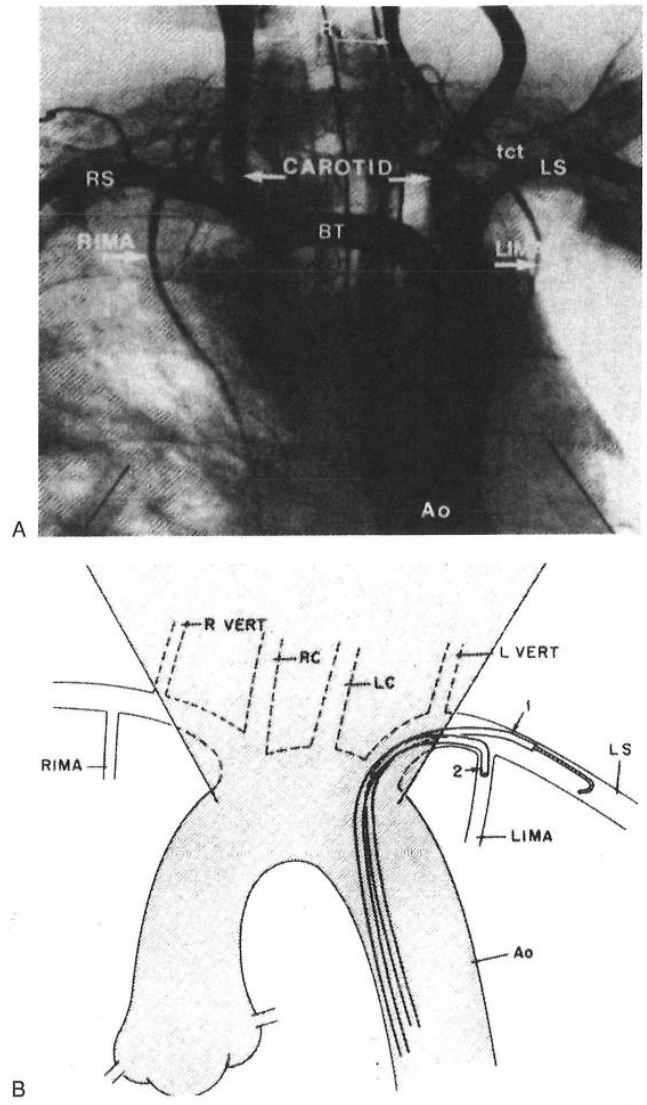
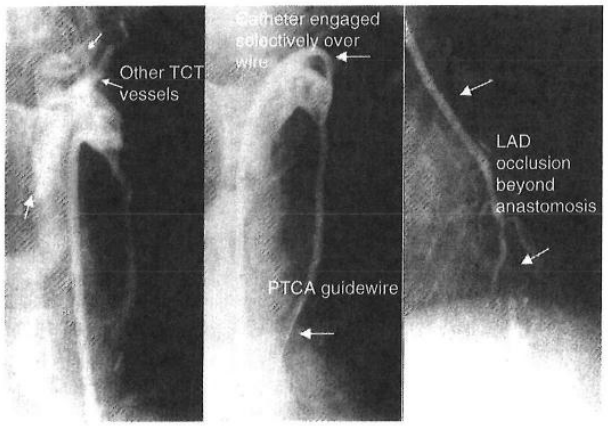


FIG. 11.8. Internal mammary angiography. **A:** Aortic arch injection shows the left internal mammary artery (LIMA) originating from the left subclavian (LS), just opposite the thyrocervical trunk (tct) and distal to the right vertebral artery (VERT). The right internal mammary artery (RIMA) originates from the right subclavian (RS) just distal to the bifurcation of the right carotid from the brachiocephalic trunk (BT). **B:** Schematic diagram shows the corresponding arch vessel origins. Note that the left subclavian artery originates just inside the leftmost edge of the wedge-shaped shadow cast by the upper-mediastinal structures in the left anterior oblique projection. Catheter manipulation in this projection facilitates advancement of a guidewire into the LS (step 1), facilitating selective cannulation of the LIMA during catheter withdrawal and slight counterclockwise rotation (step 2, see text). *Continues*



C

FIG. 11.8. *Continued.* **C:** Variant in which the internal mammary artery originates in common with the thyrocervical trunk, resulting in poor opacification. An angioplasty guidewire was placed down the internal mammary artery through the 6F diagnostic catheter and used to advance the tip of the diagnostic catheter selectively down the internal mammary artery. From that position, sufficient opacification was obtained to demonstrate occlusion of the distal left anterior descending artery beyond the anastomosis as the cause of the patient's recurrent angina.

(Fig. 11.9). Such lesions may require construction of a carotid-to-subclavian graft or catheter intervention (angioplasty, directional atherectomy, or stenting) (29) to restore normal graft flow through the internal mammary artery.

Although mammary grafts can be studied easily from the ipsilateral brachial approach, we prefer the femoral approach using a soft-tip preformed internal mammary catheter, which resembles a right Judkins catheter except for a tighter primary curve. This used to be a time-consuming process (up to 20 minutes for some

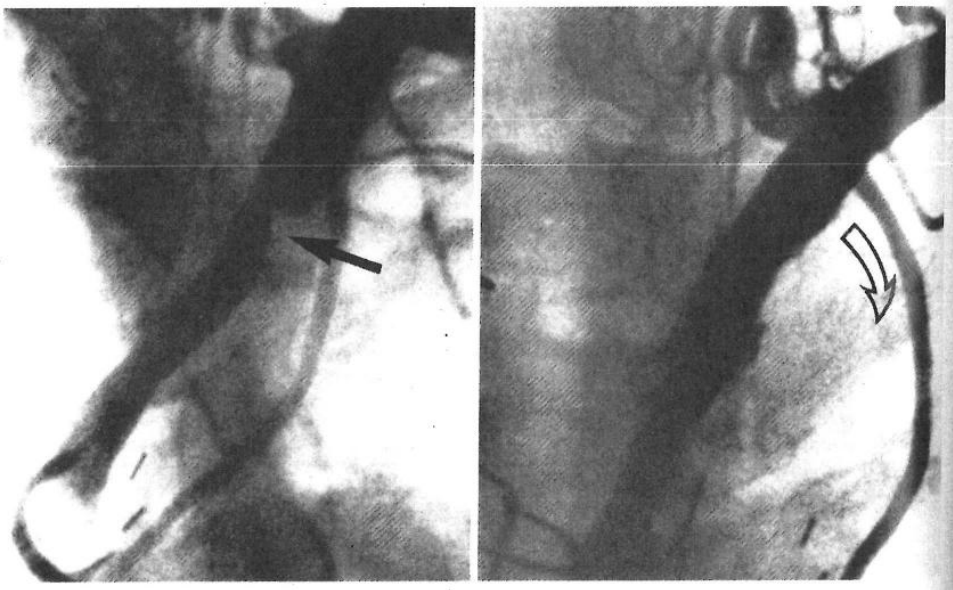


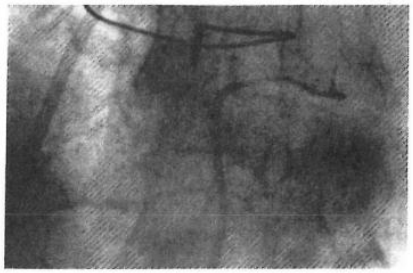
FIG. 11.9. Left subclavian artery stenosis in a patient with recurrent angina in the distribution of the otherwise patent left internal mammary artery *(left panel)*, treated by placement of Palmaz-Schatz biliary stents *(right panel)*.

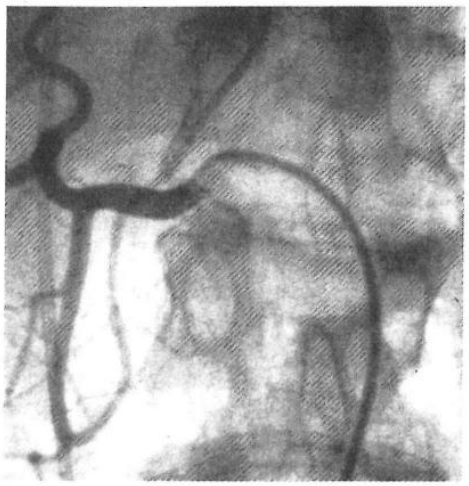
operators), but it has been reduced to less than 3 minutes in our laboratory by adoption of a systematic strategy (Fig. 11.8*B*) (27). In the LAO projection, cannulation of the left internal mammary artery begins by advancement of this catheter into the aortic arch, until it lies just inside the left edge of the wedge-like density formed by the shadow of the upper mediastinum against the lung fields. With 1 to 2 cm of J guidewire protruding from its tip, the mammary catheter is rotated counterclockwise until it falls into the subclavian artery origin. From there, the wire can be advanced well out into the axillary artery. The mammary catheter is then advanced over the wire, into the middle subclavian. The guidewire is then removed, and the catheter is flushed and filled with contrast medium. A low-osmolar contrast agent should be used to avoid causing central nervous system toxicity by reflux of hyperosmolar ionic contrast medium up the vertebral arteries. Switching to the straight anteroposterior projection, the catheter is rotated counterclockwise slightly (to make the tip point slightly anteriorly) and is withdrawn slowly until the internal mammary is engaged. Intermittent gentle puffs of contrast material help localize the mammary origin during this withdrawal. Great care should be taken to avoid catheter tip trauma and dissection of the relatively delicate mammary vessel. If selective cannulation is difficult because of tortuosity or anatomic variations, a variety of superselective or nonselective techniques can be used to permit angiographic evaluation. Nonselective injections into the subclavian artery usually allow opacification which is adequate to see that the internal mammary is open but usually not enough to provide detailed information about the distal native vessel. Inflation of a blood pressure cuff on the ipsilateral arm may help reduce runoff through the axillary artery and improve opacification of the internal mammary in cases in which selective cannulation is difficult.

Cannulation of the right internal mammary artery may be slightly more difficult because of the need to avoid the right carotid artery before entering the right subclavian itself. Again in the LAO projection, the upper mediastinal wedge is identified. The mammary catheter with protruding J wire is taken to the right edge of this shadow and rotated counterclockwise until it falls into the brachiocephalic trunk. The wire is then advanced toward the right subclavian artery. Predilection for the wire to advance into the right carotid artery may require removal of the guidewire and a nonselective contrast injection in the brachiocephalic trunk to identify the origin of the subclavian branch. The subclavian can then be cannulated with the use of a steerable (Wholey) guidewire. Once the wire is firmly out the subclavian artery, the mammary catheter is advanced as described previously. For cannulation of the right internal mammary artery, however, the catheter is rotated slightly clockwise during withdrawal to point its tip anteriorly.

Gastroepiploic Graft Cannulation

Taken together, the left and right internal mammary arteries can be used to revascularize most lesions in the LAD, proximal circumflex, and proximal right coronary arteries. Even with sequential distal anastomoses, however, the fact that there are only two internal mammary arteries means that most revascularization procedures still suffer the long-term limitations associated with the use of saphenous veins. Free segments of radial artery have also been used as bypass conduits, either from the ascending aorta (like a saphenous vein) or from the descending thoracic aorta (30) in some patients undergoing repeat bypass surgery. Although the radial artery may have slight benefit over the saphenous vein, it is prone to spasm in the early postoperative period and does not match the long-term patency record of the internal mammary artery (because it does not retain its blood supply and innervation when used as a free graft). The effort to perform ''all arterial'' bypass has brought back the use of the right gastroepiploic artery (as an arterial pedicle graft) for anastomosis to the posterior descending coronary artery or other vessels on the inferior surface of the heart (31,32). The right gastroepiploic normally supplies the majority of the greater curvature of the stomach but can be dissected free from that organ and tunneled through the diaphragm to reach the inferior wall of the heart. Angiography of this ves-





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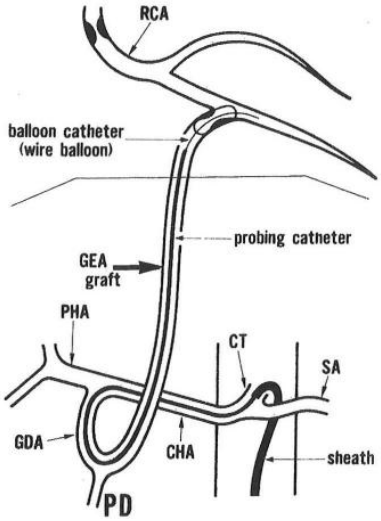

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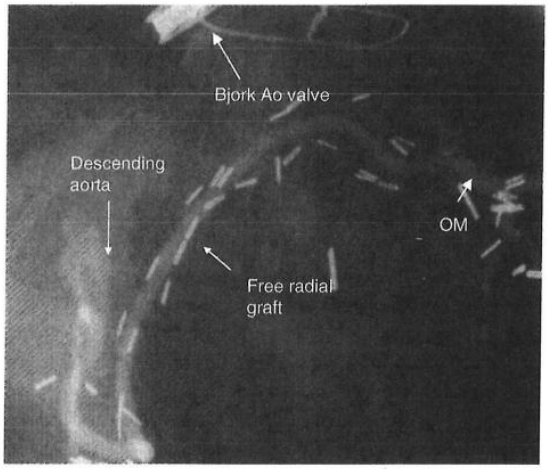

B

FIG. 11.10. A: Gastroepiploic graft anatomy. The common hepatic artery (CHA) originates with the splenic artery (SA) from the celiac trunk (CT). The gastroduodenal artery (GDA) originates from the CHA, which then becomes the proper hepatic artery (PHA). The terminal branches of the GDA are the pancreatoduodenal (PD) and the right gastroepiploic artery (GEA), shown here undergoing angioplasty of a lesion at its anastomosis to the right coronary artery (RCA). (Diagram from Ishiki T, et al. Percutaneous angioplasty of stenosed gastroepiploic artery grafts. *J Am Coll Cardiol* 1993;21:727, with permission.) **B:** Free radial graft from the descending aorta to an obtuse marginal graft, cannulated with the use of a cobra visceral angiographic catheter. Localization of the graft ostium was aided by the presence of multiple surgical clips, used to ligate small side branches of the radial artery at the time of bypass.

sel is possible with the use of standard visceral angiographic catheters (e.g., cobra catheter), which are designed to enter visceral arteries such as the celiac axis (33). From there, the catheter can be advanced into the common hepatic artery (as opposed to the splenic artery) and then turned downward into the gastroduodenal artery (Fig. 11.10). A 0.025-inch Glidewire (Terumo) can then be used to cannulate the right gastroepiploic artery (as opposed to the superior pancreatoduodenal artery) if more selective injection is desired.

THE BRACHIAL CUTDOWN APPROACH

The technique of performing brachial artery cutdown was the first used for selective coronary angiography, as described in Chapter 5. The original catheter designed by Dr. F. Mason Sones, Jr., was a thin-walled radiopaque woven Dacron catheter with a 2.67-mm (8F) external diameter to its shaft (16,34). The tip is open, and current models also include side-holes that are arranged in opposed pairs within 7 mm of the distal end. The shaft tapers abruptly to 5F external diameter at a point 5 cm from its tip. As Sones stated, this provides a ''flexible finger'' that may be curved upward into the coronary orifices by pressure of the more rigid shaft against the aortic valve cusps. This enables the Sones catheter to be used for cannulation of both the left and right coronary arteries, as well as entry into the left ventricle for ventriculography. The standard Sones catheter is available in lengths of 80, 100, and 125 cm and in 7F and 8F diameters.

Some operators use a Sones type of coronary catheter constructed of polyurethane and made by Cordis Corporation. This catheter has the same shape and taper as the woven Dacron catheter and has an end-hole with four side-holes within 7 mm of its tip. This catheter traverses a tortuous subclavian system with much greater facility and smoothness than does the woven Dacron catheter, and its enhanced torque control and reduced friction coefficient permit greater ease in engaging the coronary ostia. It can pass an 0.035-inch guidewire and is an excellent catheter for crossing a stenotic aortic valve. See Fig. 11.1 for a variety of coronary catheters that are also effective from the brachial approach.

When the Sones method is used, catheter-tip pressure should be monitored continuously once the catheter enters the brachial artery. Further passage of the catheter into the subclavian and innominate (brachiocephalic) arteries should be accomplished under both pressure monitoring and fluoroscopic visualization. Occasionally, it may be difficult to pass the catheter from the subclavian artery to the aortic arch, but a simple maneuver by the patient—such as a deep inspiration, shrugging the shoulders, or turning the head to the left—often facilitates passage of the catheter into the ascending aorta. If passage of the catheter from the subclavian artery to the ascending aorta is not accomplished immediately and with complete ease, the operator should stop catheter manipulation and use a soft J-tip 0.035-inch guidewire. Once the catheter is in the ascending aorta, the guidewire is removed and the catheter is aspirated, flushed, and reconnected to the rotating adapter of the manifold, either directly or by a short length of large-bore flexible connecting tubing.

With the Sones technique, selective engagement of the *left coronary artery* is accomplished as follows. In an LAO projection, the sinus of Valsalva containing the ostium of the left coronary artery lies to the left and the sinus containing the ostium of the right coronary artery lies to the right. The noncoronary sinus lies posteriorly. The operator advances the catheter to the aortic valve and then continues to advance the catheter until its tip bends cephalad and points toward the left coronary ostium. When the catheter is properly positioned with its tip bent cephalad, slight advancement or rotation of the catheter usually results in selective engagement of the left coronary ostium, which is verified by a small injection of radiographic contrast agent. Occasionally, a deep breath taken by the patient can facilitate this selective engagement. Once the catheter tip is engaged, it commonly (but not always) appears to be fixed by the coronary orifice. There is more than one way to successfully engage the left coronary artery with the Sones catheter. Our usual approach, illustrated in the

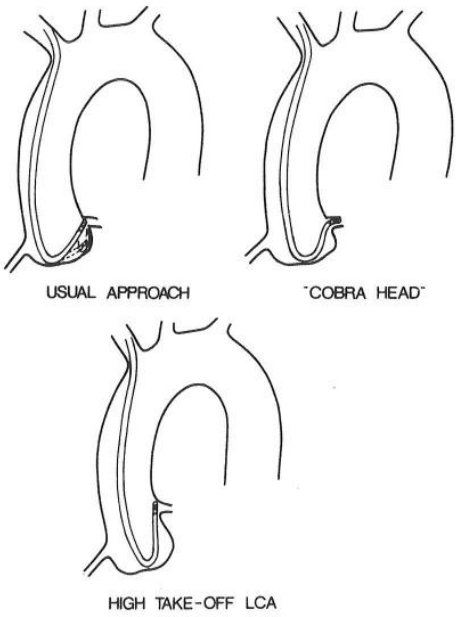

FIG. 11.11. Selective catheterization of the left coronary artery using the Sones catheter. The standard approach involves forming a smooth, shallow loop and gradually "inching up" to the ostium from below. If the distal 2 to 3 mm of the catheter tip bends downward during this inching-up process, the tip may enter the left coronary artery, giving a "cobra-head" appearance *(upper right)*. When the left coronary ostium originates high in the left sinus of Valsalva ("high takeoff" left coronary artery), the catheter may have the appearance seen in the bottom panel, where the tip is lying across the ostium, at right angles to the course of the left main coronary artery. During coronary injection in this instance, coronary blood flow usually carries the contrast agent down the vessel, giving good opacification of the entire left coronary artery.

upper left panel of Fig. 11.11, involves forming a smooth, shallow loop and gradually ''inching up'' to the ostium from below. If the distal 2 to 3 mm of the catheter tip bends downward during this inching-up process, the tip may enter the left coronary artery, giving a ''cobra head'' appearance (Fig. 11.11, upper right panel) similar to that achieved with the left Amplatz catheter (Fig. 11.5). This is a stable position that allows rotation of the patient in a cradle-type table top without disengagement of the catheter. For the high-takeoff left coronary ostium, the catheter may have an appearance in which the catheter tip is lying across the ostium, at right angles to the course of the left main coronary artery (as in Fig. 11.11, bottom). During contrast injection in this instance, coronary blood flow usually carries the contrast agent down the vessel, giving good opacification of the entire left coronary artery.

Once the catheter tip has engaged the coronary ostium and no damping of pressure from the catheter tip is observed, cineangiography may be performed with selective injection of radiopaque material in a variety of views, as described later.

Selective engagement of the *right coronary* orifice may be accomplished as illustrated in steps 1 through 3 of Fig. 11.12. In the shallow LAO projection, the catheter is curved up toward the left coronary artery (step 1) and clockwise torque is applied. While the operator is gradually applying clockwise torque, a gentle to-and-fro motion of the catheter (excursions of not more than 5 to 10 mm) helps to translate the applied torque to the catheter tip. When the tip starts moving in its clockwise sweep of the anterior wall of the aorta, the operator maintains (but does not increase) a clockwise torque tension on the catheter and simultaneously pulls the catheter back slightly (step 2, Fig. 11.12), because the right coronary ostium is lower than that of the left coronary artery. At this point, the catheter usually makes an abrupt turn into the right coronary ostium, at which time the operator must release all torque to prevent the catheter tip from continuing its sweep past the ostium. On occasion, the Sones catheter literally leaps into the right coronary artery and 4 to 5 cm down its lumen. If this occurs, the catheter should be gently withdrawn until its tip is stable just within the ostium. Another technique for catheterizing the right coronary artery involves a more direct approach by way of the right coronary cusp. With the catheter in the right sinus, the operator should make a small curve on the tip, directed rightward. A small dose of contrast material in the right sinus of Valsalva allows visualization of the right coronary orifice and facilitates selec-

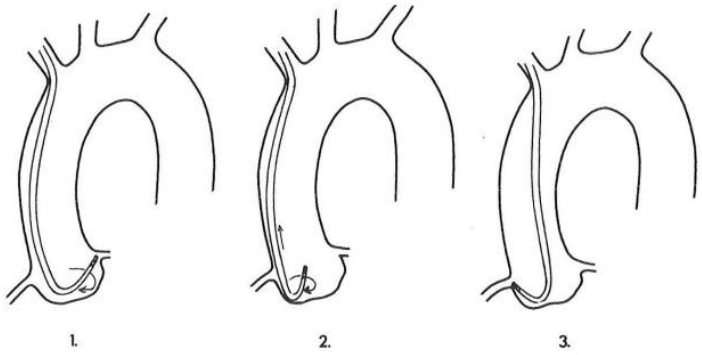

FIG. 11.12. Selective catheterization of the right coronary artery using the Sones catheter. In the shallow left anterior oblique projection, the catheter is curved upward and to the left (1) and clockwise torque is applied. While the operator is gradually applying clockwise torque, a gentle to-and-fro motion of the catheter helps to translate the applied torque to the catheter tip. When the tip starts moving in its clockwise sweep of the anterior wall of the aorta, the operator maintains (but does not increase) a clockwise torque tension on the catheter and simultaneously pulls the catheter back slightly (2), because the right coronary ostium is lower than that of the left coronary artery. At this point the catheter usually makes an abrupt leap into the right coronary ostium (3), at which time the operator must release all torque to prevent the catheter tip from continuing its sweep and passing by the ostium. (See text for details and alternative methods.)

tive engagement. Occasionally, a deep inspiration by the patient accompanied by gentle advancement of the catheter to the right of the aortic root, results in selective engagement of the right coronary artery.

In addition to the Sones catheter, many other catheters may be used for coronary arteriography from the brachial cutdown or percutaneous approach, including the Amplatz (24), Schoonmaker (35), Bourassa, Judkins, and other specially designed catheters. Some of these catheters are illustrated in Fig. 11.1. Although the Amplatz catheters were originally devised for use from the percutaneous femoral approach, we have found these catheters highly useful from the brachial approach in cases in which there was difficulty in seating the Sones catheter. Amplatz catheters come in different shapes for the right and left coronary artery and basically incorporate a preformed curvature that is like that of an already-engaged Sones catheter. We have found the AL2 to be adequate for most patients with normal aortic roots, whereas the AL3 may be necessary for a dilated ascending aorta or in large men. Occasionally an AL4 is needed for pronounced aortic dilatation or for a left coronary artery whose ostium originates very high in the left sinus of Valsalva (high-takeoff left coronary artery). The AR1 right coronary catheter is usually adequate for patients with a normal aortic root, whereas the AR2 may be required for patients with an enlarged aortic root or for engagement of saphenous vein bypass grafts. The Amplatz catheters should be introduced through the subclavian artery over a guidewire and (unlike the Sones catheter) cannot be used safely for ventriculography. When working from the left brachial approach, it is also possible to use standard Judkins catheters, since the course of the catheter around the arch emulates its path from the femoral approach in that area. We have not had experience with the Bourassa or Schoonmaker catheters from the brachial approach, but large published series using these catheters from the femoral artery approach suggest that they should also be effective from the brachial approach. Of course, all of these considerations about coronary angiography from the brachial cutdown approach also apply to the percutaneous brachial, axillary, or radial approaches (see Chapter 4).

ADVERSE EFFECTS OF CORONARY ANGIOGRAPHY

Once the coronary vessels have been engaged, selective angiography requires transient but nearly complete replacement of blood flow with a radiopaque contrast agent. A wide variety of iodine-containing agents are currently used for coronary angiography and have already been discussed in greater detail in Chapter 2.

Coronary injection of a high-osmolar contrast agent may have potentially deleterious effects (see Chapters 2 and 3) that include (a) transient (10- to 20-second) hemodynamic depression marked by arterial hypotension and elevation of the left ventricular end-diastolic pressure; (b) ECG effects with T-wave inversion or peaking in the inferior leads (during right and left coronary injection, respectively), sinus slowing or arrest, and prolongation of the PR, QRS, and QT intervals (36,37); (c) significant arrhythmia (asystole or ventricular tachycardia/fibrillation) (38); (d) myocardial ischemia due to interruption of oxygen delivery or inappropriate arteriolar vasodilation (coronary ''steal''); (e) allergic reaction (39); and (f) cumulative renal toxicity (40). Some (but not all) of these adverse effects are eliminated by use of a low-osmolar contrast agent, albeit at a modestly increased expense (41).

To recognize, treat, and hopefully prevent these adverse effects, patients undergoing coronary angiography should be monitored continuously in terms of clinical status, surface ECG, and arterial pressure from the catheter tip. In patients with baseline left ventricular dysfunction or marked ischemic instability, we also like to display pulmonary artery pressure continuously on the same scale as the arterial pressure, because this provides the earliest indication of procedural problems or decompensation. A significant rise in pulmonary artery mean or diastolic pressure should prompt temporary suspension of angiography and initiation of treatment (e.g., intravenous furosemide, nitroglycerin, nitroprusside) before frank pulmonary edema develops.

If right-sided heart catheterization is to be performed, the venous sheath provides a ready route for the rapid administration of fluid or medications through its sidearm and allows rapid insertion of a temporary pacing electrode if needed.

We do not, however, endorse the routine prophylactic placement of temporary pacing electrodes in patients undergoing coronary angiography (42). Most episodes of bradycardia or asystole are brief and are resolved promptly by having the patient give a forceful cough, which elevates central aortic pressure and probably helps wash residual contrast material out of the myocardial capillary bed. True life-threatening bradycardia is very uncommon and can be managed successfully by having the patient cough at 1- to 2-second intervals while a temporary pacing lead is inserted through the indwelling venous sheath and attached to a generator kept at standby at the foot of the catheterization table. Similarly, prophylactic drugs are not given routinely to prevent ventricular tachyarrhythmias, although drugs (e.g., lidocaine, procainamide, atropine, epinephrine), a defibrillator, and airway management equipment are always kept at the ready and can be brought into play within seconds.

One of the most common adverse effects seen during coronary angiography is the provocation of myocardial ischemia, particularly in patients with unstable angina. In such patients, we commonly do not interrupt any precatheterization heparin infusion (and usually give additional heparin during the catheterization itself) and do not reverse heparin at the completion of the procedure. In very unstable patients, we modify our usual practice of performing the left ventriculogram before coronary angiography (lest an adverse reaction to the ventriculogram compromise the more crucial coronary study). When myocardial ischemia does occur during coronary angiography, the best course of action is to remove the catheter from the coronary ostium and temporarily suspend injections until angina resolves. If this takes more than 30 seconds, we typically administer nitroglycerin (200-μg bolus, repeated at 30-second intervals up to a total of 1,000 μg) into either the involved coronary artery or the pulmonary artery catheter. If marked arterial hypertension is present and fails to respond to nitroglycerin, we may administer

other vasodilators as needed to bring the blood pressure down. In patients with inappropriate tachycardia in the setting of angina and reasonable systolic left ventricular function, intravenous propranolol (1 mg every minute to a total dose of 0.1 to 0.15 mg/kg) or an infusion of a short-acting β-blocking agent (esmolol) is frequently beneficial. Only rarely (in patients with severe three-vessel disease and/or left main coronary artery disease and those whose ischemia is associated with hypotension) is myocardial ischemia severe enough and refractory to this management program to prompt placement of an intraaortic counterpulsation balloon in the contralateral femoral artery before completion of coronary angiography (see Chapter 21). In any patient with prolonged or refractory ischemia during diagnostic coronary angiography, it may be worthwhile to perform limited reexamination of the coronary vessels to determine whether the angiographic procedure has caused a problem (spasm, dissection, thrombosis) that might require immediate treatment with additional vasodilators, balloon angioplasty, thrombolysis, or emergency bypass surgery.

Severe allergic reactions are uncommon during coronary angiography and are best prevented by 18 to 24 hours of premedication (prednisone, 20 to 40 mg, and cimetidine, 300 mg every 6 hours) (32) and/or use of a nonionic contrast agent in patients with a history of prior allergic reaction to radiographic contrast media (41). When a severe unexpected reaction does occur, it usually responds promptly to the intravenous administration of epinephrine (0.1 mg = 1 mL of the 1:10,000 solution available on most emergency carts, repeated every 2 minutes until the blood pressure and/or wheezing improves). Larger bolus doses of epinephrine are to be avoided, because they may provoke marked tachycardia, hypertension, and arrhythmia.

Renal insufficiency may develop after coronary angiography, particularly in patients who are hypovolemic, who receive large volumes of contrast material (more than 3 mL/kg), or who have had prior renal insufficiency, diabetes, or multiple myeloma (33). In these patients, every effort should be made to give adequate hydration before and after the procedure (see Chapters 2 and 3). Use of low-osmolar contrast agents may be helpful in this situation, but their real benefit remains controversial (41).

INJECTION TECHNIQUE

As mentioned previously, high-quality coronary angiography requires selective injection of radiographic contrast material at an adequate rate and volume to transiently replace the blood contained in the involved vessel with slight but continuous reflux into the aortic root. Too timid an injection allows intermittent entry of nonopaque blood into the coronary artery (producing streaming, which makes interpretation of lesions difficult) and prevents visualization of the coronary ostium and proximal coronary branches. However, too vigorous an injection can cause coronary dissection or excessive myocardial blushing, and too prolonged an injection may contribute to increased myocardial depression or bradycardia.

We train our fellows to adjust the rate and duration of manual contrast injection to match the observed filling pattern of the particular vessel being injected. Injection velocity is built up gradually during the initial 1 second until the injection rate is adequate to completely replace antegrade blood flow into the coronary ostium (Fig. 11.13). The associated rate and volume required to accomplish this goal have been measured (43) and found to average 7 mL at 2.1 mL/sec in the left and 4.8 mL at 1.7 mL/sec in the right coronary artery. In patients with occlusion, much smaller rates and volumes are required, and in patients with left ventricular hypertrophy (e.g., aortic stenosis, hypertrophic myopathy), much larger volumes and higher rates of injection may be required.

The injection is maintained until the entire vessel is opacified. If there is any question as to whether the body of the injection has provided adequate reflux to visualize the coronary ostium, an additional burst of contrast agent (extra reflux) should be given before the injection is terminated. The injection is then terminated abruptly by turning the manifold stopcock back to monitor pressure, although cine filming continues until opacification of distal vessels or late-

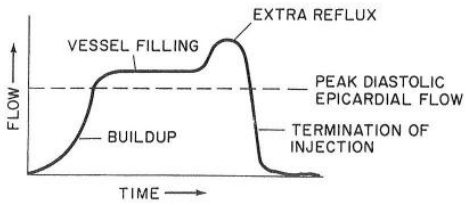

FIG. 11.13. Suggested injection pattern for coronary angiography. To appropriately replace antegrade coronary blood flow with contrast medium throughout the cardiac cycle, the operator should build up the velocity of injection over 1 to 2 seconds until no unopacified blood is seen to enter the ostium and there is reflux of contrast medium into the aorta during systole and diastole. This injection is maintained until the entire coronary artery is filled with contrast medium. If the ostium has not been well seen, a brief extra push should be given to cause adequate reflux into the aortic root, and then the injection is terminated. Prolonged held inspiration with some degree of Valsalva maneuver is sometimes used during Sones angiography to reduce coronary flow and make it much easier to replace blood flow during manual contrast injection.

filling branches is complete. The operator monitors for excessive bradycardia or hypotension, reviews the video playback, and sets the gantry angles for the next injection. To avoid problems, each injection should begin with a completely full (and bubble-free) injection syringe, held with the handle slightly elevated so that any microbubbles will drift up toward the plunger. Recent changes in labeling of contrast agents also suggest that the injection syringe be managed in such a way as to avoid mixtures of blood and contrast material, because such mixtures may promote formation of thrombi (particularly when nonionic contrast agents are used).

Although manual contrast injection is the standard technique in coronary angiography, some operators favor use of a power injector (as used in left ventriculography or aortography) to perform coronary injections (44). The injector is preset for a rate to match the involved vessel (2 to 3 mL/sec for the right and 3 to 4 mL/sec for the left coronary artery) and is activated by a foot switch for a period sufficient to fill the coronary artery with contrast medium (usually 2 to 3 seconds). This approach allows a single operator to perform injections and move the table and has proved safe in thousands of procedures. A new power injector has been introduced (Acist, Bracco Bristol Myers Squib) (Fig. 11.2) that can perform such power injections under rate control by finger pressure on a sterile control handle, reverting automatically to pressure monitoring when the injection is terminated. This may be of value when a single operator must both perform injections and pan the table during diagnostic coronary angiography.

ANATOMY, ANGIOGRAPHIC VIEWS, AND QUANTITATION OF STENOSIS

Coronary Anatomy

The coronary angiographer must develop a detailed familiarity with normal coronary arterial anatomy and its common variants. For those just learning coronary anatomy, the main coronary trunks can be considered to lie in one of two orthogonal planes (Fig. 11.14). The anterior descending and posterior descending coronary arteries lie in the plane of the interventricular septum, whereas the right and circumflex coronary trunks lie in the plane of the atrioventricular valves. In the 60° LAO projection, one is looking down the plane of the interventricular septum, with the plane of the atrioventricular (AV) valves seen *en face*; in the 30° RAO projection, one is looking down the plane of the AV valves, with the plane of the interventricular septum seen *en face*. The major segments and branches have each been assigned a numeric identification in the Bypass Angioplasty Revascularization Investigation (BARI) modification (45) of the Coronary Artery Surgery Study (CASS) nomenclature (Fig. 11.15).

Right-dominant Circulation

The right coronary artery gives rise to the conus branch (which supplies the right ventricular outflow tract) and one or more acute marginal branches (which supply the free wall of the right ventricle), whether or not the circulation is right-dominant. In the 85% of patients who have a

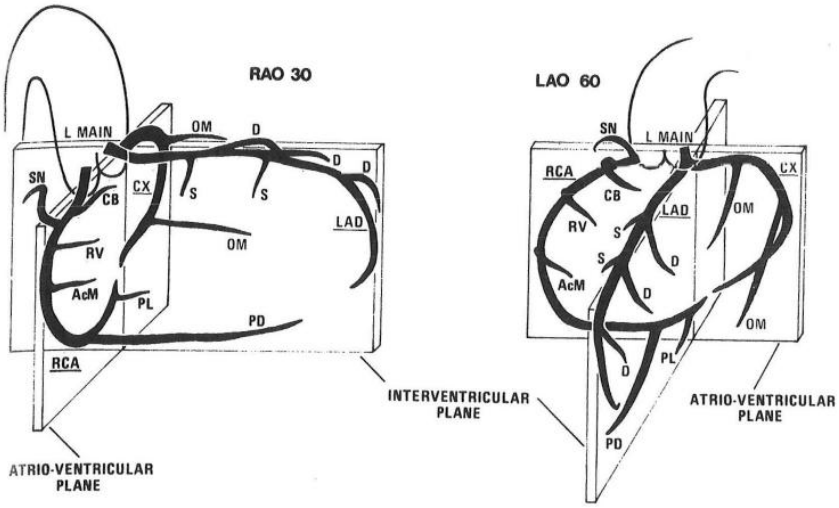

FIG. 11.14. Representation of coronary anatomy in relation to the interventricular and atrioventricular valve planes. Coronary branches are as indicated: L Main, left main; LAD, left anterior descending; D, diagonal; S, septal; CX, circumflex; OM, obtuse marginal; RCA, right coronary; CB, conus branch; SN, sinus node; AcM, acute marginal; PD, posterior descending; PL, posterolateral left ventricular.

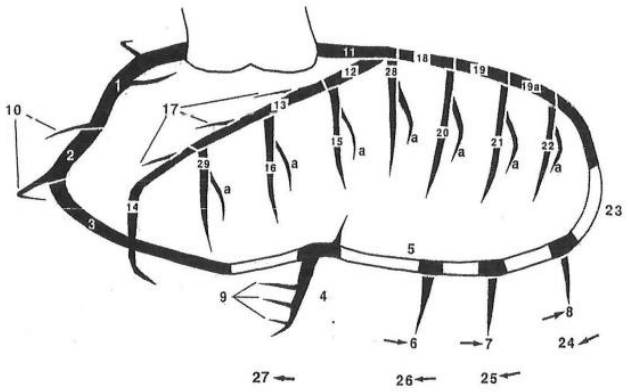

FIG. 11.15. The numeric coding system and official names of the coronary segments, as used in the Bypass Angioplasty Revascularization Investigation (BARI) study. *Right coronary:* 1, proximal; 2, middle; 3, distal; 4, posterior descending; 5, posteroatrioventricular; 6, first posterolateral; 7, second posterolateral; 8, third posterolateral; 9, inferior septals; 10, acute marginals. *Left coronary:* 11, left main; 12, proximal left anterior descending; 13, middle left anterior descending; 14, distal left anterior descending; 15, first diagonal (a, branch of first diagonal); 16, second diagonal; 17, septals (anterior septals); 18, proximal circumflex; 19, middle circumflex; 20, distal circumflex; 21, 22, and 23, first, second, and third obtuse marginals; 23, left atrioventricular; 24, 25, and 26, first, second, and third posterolaterals (in left- or balanced-dominant system); 27, left posterior descending (in left-dominant system); 28, ramus (ramus intermedius); 29, third diagonal. (From The BARI protocol. Protocol for the Bypass Angioplasty Revascularization Investigation. *Circulation* 1991;84:V1, with permission.)

right-dominant coronary artery, it goes on to form the AV nodal artery, the posterior descending, and the posterolateral left ventricular branches which supply the inferior aspect of the left ventricle and interventricular septum (Fig. 11.14). The left main trunk branches after a short (but variable) distance into the LAD and the circumflex coronary arteries. The LAD artery gives rise to septal branches, which curve down into the interventricular septum, as well as diagonal branches, which wrap over the anterolateral free wall of the left ventricle. Some patients have a "twin" LAD system, in which one trunk (frequently intramyocardial) supplies the entire septum and the other trunk runs on the surface of the heart, supplying all the diagonal branches. The circumflex artery courses clockwise in the AV groove (viewed from the apex) as it gives rise to one or more obtuse marginal branches which supply the lateral free wall of the left ventricle, but it does not reach the crux in patients with a right-dominant circulation. In some patients, a large intermedius or ramus medianus branch (neither a diagonal nor a marginal) may originate directly from the left main trunk, bisecting the angle between the LAD and circumflex arteries, to create a trifurcation pattern of the left main coronary artery. Regardless of whether the patient is right- or left-dominant, the sinus node originates as a proximal branch of the right coronary in 60% of patients and as a left atrial branch of the circumflex in the remaining 40% of patients.

Left-dominant Circulation

In 8% of patients, the coronary circulation is left-dominant; that is, the posterolateral left ventricular, posterior descending, and AV nodal arteries are all supplied by the terminal portion of the left circumflex coronary artery. In such patients, the right coronary artery is quite small and supplies only the right atrium and right ventricle. It may be important to visualize, as a potential source of right-to-left collaterals, but the small diameter of a nondominant right coronary artery predisposes it to damping and catheter-induced spasm (see later discussion), which make limited injections advisable.

Balanced-dominant Circulation

In about 7% of hearts, there is a codominant or balanced system, in which the right coronary artery gives rise to the posterior descending artery and then terminates, and the circumflex artery gives rise to all the posterior left ventricular branches and perhaps also to a parallel posterior descending branch that supplies part of the interventricular septum. In some patients, the supply to the inferior wall is further fractionated among a short posterior descending branch of the right coronary (which supplies the inferobase), branches of the distal circumflex (which supply the midinferior wall), and branches of the acute marginal (which extend to supply the inferoapex).

Anatomic Variants

Although these basic concepts describe the general pattern of the coronary circulation, it must be noted that there is considerable patient-to-patient variability in the size and position of the various coronary arterial branches (46). In 1% to 2% of patients, these coronary anatomic features are sufficiently divergent to qualify as *coronary anomalies*. Every operator must be thoroughly familiar with these anatomic anomalies and continually vigilant for their occurrence, lest failure to recognize an anomaly result in an incomplete and therefore inadequate examination. In a review of 126,595 cases from the Cleveland Clinic (47), the most common of these anomalies was separate ostia of the LAD and left circumflex arteries (0.41%). When this anomaly is present, the catheter usually sits with its tip in the LAD, although there is generally adequate spillover to opacify the circumflex. If not, separate cannulation of the circumflex may be necessary, using the next-larger size left Judkins catheter (e.g., JL5 instead of JL4) or a left Amplatz catheter. A similar situation may exist in the right coronary artery, where the conus branch may have a separate ostium whose separate cannulation may be necessary to demonstrate important collaterals when reflux during the right coronary injection is not adequate to opacify the conus (Fig. 11.16).

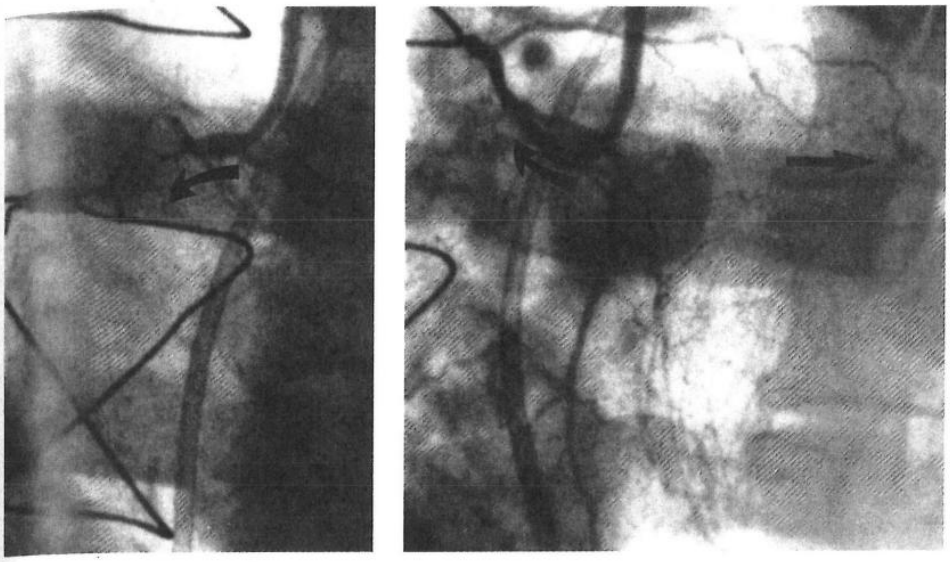

FIG. 11.16. Importance of adequate reflux or separate injection of the conus branch when it is a major source of collaterals. **Left,** Selective injection of the native right coronary artery showing proximal occlusion, with no filling of the conus branch. **Right,** Selective gentle injection into the conus branch shows extensive collaterals to the left anterior descending as well as the distal right coronary artery.

The next most common anomaly is origin of the circumflex from the right coronary artery or right sinus of Valsalva (0.37%). This should be suspected when the left main coronary artery is unusually long and a paucity of vessels to the lateral wall are identified. Careful review of the RAO left ventriculogram may show a ''dot'' of contrast material just behind the aortic valve when the anomalous circumflex runs posterior to the aorta (48). If an anomalous circumflex is not filled adequately during right coronary injection, it must be cannulated separately (usually with an AL1 catheter). We have seen patients in whom the only coronary lesion was located in such an anomalous circumflex, and failure to identify and opacify this vessel would have led to failure to diagnose and treat the problem. In another common variant, anomalous vessels (particularly the right coronary artery) may originate unusually high in the aortic root or out of the normal coronary plane (38), making them easier to cannulate with left Amplatz rather than right Judkins catheters. The left coronary may originate from the right sinus of Valsalva (Fig. 11.17), either as a separate ostium (49) or as part of a single coronary (50). Origin of a coronary artery from the ''noncoronary'' sinus of Valsalva is rare but has been reported (47,51). The main effect of these coronary anomalies is to test the patience, knowledge, and resourcefulness of the angiographer. Other anomalies, however, may themselves cause myocardial ischemia (even in the absence of atherosclerotic stenosis); they are described later in the section on nonatherosclerotic coronary artery disease.

Angiographic Views

Accurate coronary diagnosis requires coronary injections in multiple views, to be sure that all coronary segments are seen clearly without foreshortening or overlap. The angulation of each view is given in two terms. The first term denotes *rotation.* For example, the term RAO designates a view in which the image intensifier is located over the patient's right anterior chest

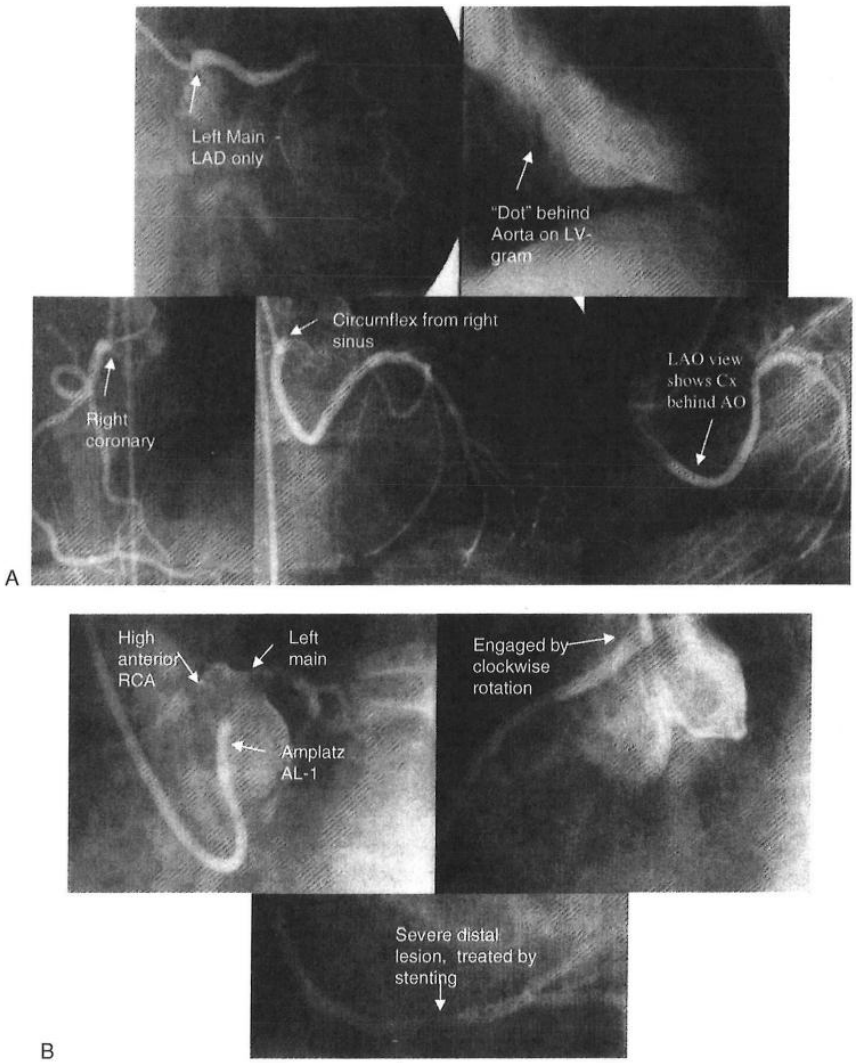

FIG. 11.17. A: Anomalous origin of the circumflex from the right coronary artery (RCA). Note the "long left main" *(upper left)* and absence of a circumflex during injection of the left coronary artery. Review of the right anterior oblique (RAO) left ventriculogram *(upper right)* shows the telltale "dot" behind the aortic root, created by an end-on look at the anomalous circumflex coursing behind the aorta. The right coronary originates normally *(lower left)*, with the anomalous circumflex *(lower center)* originating from the right sinus of Valsalva immediately posterior to the RCA origin, then coursing behind the aorta to reach the lateral wall of the left ventricle. The left anterior oblique (LAO) projection *(lower right)* shows the anomalous circumflex coursing behind the aorta. **B:** Anomalous origin of the right coronary from the left sinus of Valsalva. Nonselective injection through a Amplatz left (AL1) catheter in the left sinus *(upper left)* shows filling of both the left main and the high anterior right (RCA) coronary. The anomalous RCA is engaged by clockwise rotation of the catheter *(upper right)*, allowing successful stenting of the severe distal lesion *(bottom)*.

wall, and LAO refers to a view in which the image intensifier is located over the patient's left anterior chest wall. The second term denotes *skew*—the amount of angulation toward the patient's head (cranial) or foot (caudal). Although the full nomenclature of skew specifies first the source of the beam and then the location of the imaging device—such as "caudocranial," to denote that the x-ray tube is toward the patient's feet and the image intensifier is located toward the patient's head—in practice this is simplified to give just the location of the imaging device. The term RAO caudocranial is thus stated as RAO-cranial.

When cradle systems were used in the 1970s, these views were usually limited to different degrees of left or right anterior obliquity in the transverse plane, including the classic 60° LAO and 30° RAO projections (Fig. 11.14). To allow concurrent cranial angulation of the x-ray beam, cradle systems were modified by propping the patient's shoulders up on a foam wedge (hence the name "sit-up view") to provide compound LAO-cranial projection. In the 1980s, cradle systems were abandoned in favor of systems in which the x-ray tube and image intensifier are mounted on a parallelogram or on a rigid U-arm supported by a rotating pedestal (see Chapter 2) to allow compound beam angulation in any combination of conventional transverse (LAO, RAO) and skew (cranial, caudal) angulation (Fig. 11.18). Although these views place increased demands on the generator and increase the amount of scattered radiation, there is no doubt that they have improved our ability to define coronary anatomy (52–54).

Not all potential views are necessary in a given patient to constitute an adequate study. Rather, a series of screening views should be used as the foundation of the study, adjusted or supplemented by one or more additional views selected especially to more completely define suspicious areas. This requires the operator to interpret the coronary anatomy as each injection is made, or at least during in-room review from the digital storage system—rather than simply shooting a series of routine views and hoping that the study will prove adequate when reviewed later. Although some laboratories rely on a technician to set up shots and pan the table during coronary angiography, each operator should know how to do this, in order to develop a good understanding of how changes in gantry angulation influence the projected coronary

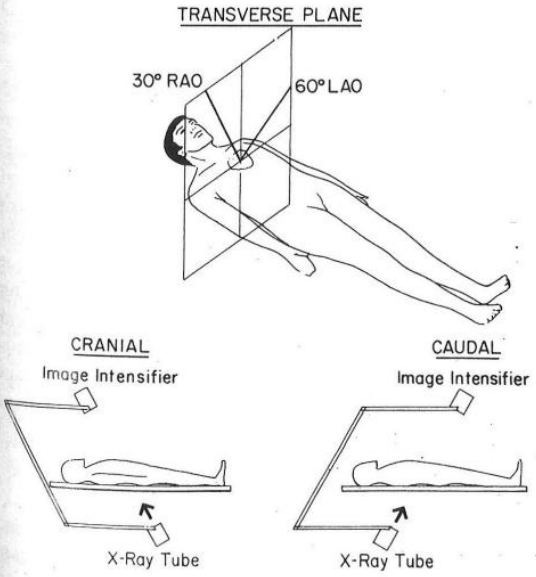

FIG. 11.18. Geometry of angulated views. Conventional coronary angiography was performed with angulation only in the transverse plane *(top)*, as demonstrated by the 60° left anterior oblique (LAO) and 30° right anterior oblique (RAO) views. Improved x-ray equipment now permits simultaneous cranial or caudal angulation in the saggital plane. Each view is named according to the location of the image intensifier, rather than by the older nomenclature, which specified the location of both the x-ray tube and intensifier. For example, "cranial" is equivalent to caudocranial.

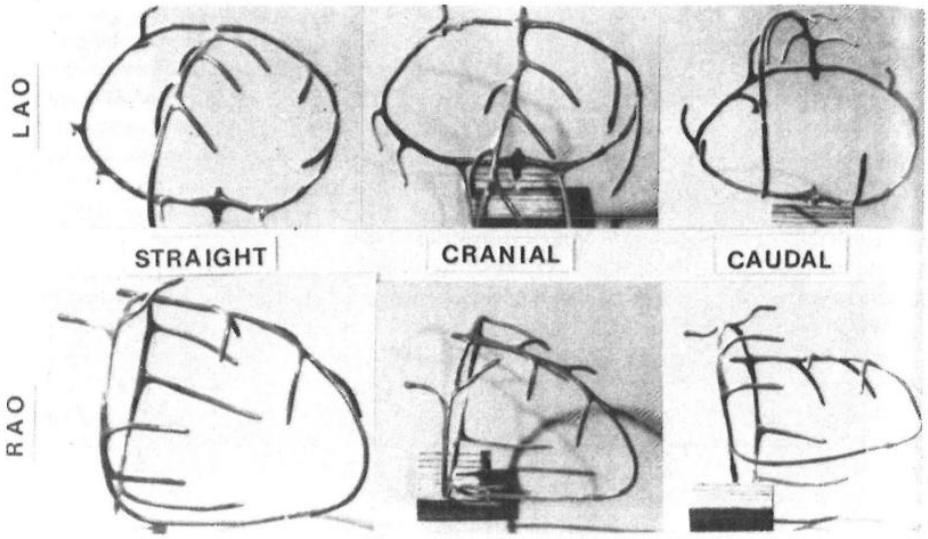

FIG. 11.19. Demonstration of angiographic projections using the author's coronary model. Left anterior oblique (LAO) and right anterior oblique (RAO) projections are photographed straight (i.e., with no cranial or caudal angulation) and with moderate cranial and moderate caudal angulation (see text for details).

anatomy. One valuable training tool in this respect is a simple wire model of the coronary anatomy which is viewed as it is moved into different angles (Fig. 11.19) (55). A computer program that simulates the effect of changing angles on the projected coronary anatomy is also available. Although there is no substitution for this type of ''hands-on'' learning, the discussion below is provided as a rough introduction.

Right Anterior Oblique Projections.

For historic reasons relating to cradle systems, the screening views used in many laboratories were the straight LAO-RAO angulations. With the availability of more modern gantry systems, it is clear that certain cranial and caudal angulated views offer far better anatomic definition. Therefore, we generally avoid the straight 30° RAO projection of the left coronary artery, because it suffers from overlap and foreshortening of both the LAD and circumflex vessels (Fig. 11.19). Instead, we have made the RAO-caudal projection (0° to 10° RAO and 15° to 20° caudal) our initial view of choice in studying unstable patients, because it provides an excellent view of the left main bifurcation, the proximal LAD artery, and the proximal to middle circumflex artery. The second view we perform is a shallow RAO-cranial projection (0° to 10° RAO and 25° to 40° cranial), which provides a superior view of the middle and distal LAD, with clear visualization of the origins of the septal and diagonal branches. This shallow RAO cranial view is also quite good for examination of the distal right coronary artery or distal circumflex, because it effectively ''unstacks'' the posterior descending and posterolateral branches and projects them without foreshortening. It seldom, however, provides any useful information about the left main or circumflex coronary artery, because it causes them to be overlapped and foreshortened.

Left Anterior Oblique Projections

The conventional 60° LAO projection is limited by overlap and foreshortening of the left

coronary artery, although it is very useful in the evaluation of the proximal and middle right coronary artery. The LAO-cranial view, created by the addition of 15° to 30° of cranial angulation, elongates the left main and proximal LAD arteries while projecting the intermedius or first diagonal branch downward off the proximal circumflex. If radiographic penetration in this view is difficult, reducing the LAO angulation to 30° to 40° usually allows the LAD artery to fall into the lucent wedge between the right hemidiaphragm and the spine. Alternatively, performing the cine run during a sustained maximal inspiration usually pulls the diaphragm down and improves x-ray penetration. The LAO-caudal view (40° to 60° LAO and 10° to 20° caudal) projects the left coronary artery upward from the left main in the appearance of a spider, and it usually offers improved visualization of the left main, proximal LAD, and proximal circumflex arteries. It is particularly valuable in patients whose heart has a horizontal lie (i.e., the origin of the left main artery projects at or below the proximal LAD artery in the standard LAO projection). This "spider view" (LAO-caudal) can often be enhanced by filming during maximal expiration, which accentuates a horizontal cardiac position and allows a better look from below, although it stresses the radiographic capacity of most older installations.

Posteroanterior and Left Lateral Projections

The straight posteroanterior (PA, or "0–0") and left lateral projections tend to be underutilized in the era of complex angulation. Because the left main coronary artery curves from a more leftward to an almost anterior direction along its length, the *PA projection (sometimes referred to incorrectly as the "AP" projection)* frequently provides the best view of the left main ostium. On the other hand, the shallow RAO-caudal view frequently provides a better look at the more distal left main artery. The *left lateral projection* is particularly useful in examining the proximal circumflex and the proximal and distal LAD arteries, particularly when combined with slight (10° to 15°) cranial angulation. This projection also provides the best look at the anastomosis of a left internal mammary graft to the mid-distal LAD, and it offers an excellent look at the midportion of the right coronary artery, free of the excessive motion seen when this portion of the vessel is viewed in the straight RAO projection. The left lateral projection also has the advantage of allowing easy radiographic penetration in most patients when it is performed with both of the patient's hands positioned behind the head, although it generates the highest degree of backscatter to the operator given the proximity of the beam entry point on the patient's right side.

Over the past several years, operators in our laboratory have adopted a uniform sequence of these views, adjusting the exact angles slightly in each patient as dictated by test puffs of contrast agent. Beginning with the left coronary artery, these views include

1. RAO-caudal to visualize the left main, proximal LAD, and proximal circumflex
2. RAO-cranial to visualize the middle and distal LAD without overlap of septal or diagonal branches
3. LAO-cranial to visualize the middle and distal LAD in an orthogonal projection
4. LAO-caudal to visualize the left main and proximal circumflex.

One or more supplemental views (PA, lateral-cranial, lateral-caudal) may then be taken to clarify any areas of uncertainty. The right coronary catheter is then placed, after which three screening views are obtained:

1. LAO to visualize the proximal right coronary artery
2. RAO-cranial to visualize the posterior descending and posterolateral branches
3. Lateral to visualize the middle right coronary artery.

Lesion Quantification

To quantify a coronary stenosis accurately, it must be seen in profile, free from artifact related to foreshortening or obfuscation by a crossing vessel. Multiple views are important, because many lesions leave a lumen that is markedly ec-

centric (elliptic rather than round) (56). When seen across its major axis, the width of the lumen may appear almost normal; the only clue to the severe degree of narrowing may be marked lucency, caused by thinning of the contrast column. Any such suspicious lesions must be examined in a variety of other projections to reveal their true severity and to distinguish the lucency caused by eccentric stenosis from a similar lucency that may be seen adjacent to an area of denser contrast (caused by tortuosity or overlapping vessels in the absence of any true abnormality at the site) through a perceptual artifact known as the Mach effect (57).

The ability of coronary angiography to quantify the degree of stenosis at different points in the coronary circulation is fundamentally limited by the fact that it consists of a ''lumen-ogram,'' in which each stenosis can be evaluated only by comparison to an adjacent ''reference'' segment that is presumed to be free of disease. In fact, both intravascular ultrasound (56) (see Chapter 19) and pathologic examination (58) show that even segments that appear smooth on angiography may harbor substantial plaque. It is therefore important to have a sense of the normal caliber of the major coronary arteries: 4.5 ± 0.5 mm for the left main coronary artery, 3.7 ± 0.4 mm for the LAD, 3.4 ± 0.5 mm for a nondominant versus 4.2 ± 0.6 mm for a dominant circumflex, and 3.9 ± 0.6 mm for a dominant versus 2.8 ± 0.5 mm for a nondominant right coronary artery (59). By comparing the diameter of a presumably disease-free segment of coronary artery to the size of the diagnostic catheter (6F = 2 mm), the operator can identify vessels that fall below these normal size ranges and therefore may be diffusely diseased.

Other than the difficulty in finding a disease-free reference segment, another major problem in the interpretation of a coronary angiogram is deciding on the severity of any given stenosis. Both animal data (60) and human data (61) show that a stenosis that reduces the lumen diameter by 50% (hence reducing the cross-sectional area by 75%) is ''hemodynamically significant'' in that it reduces the normal three- to four-fold flow reserve of a coronary bed (Fig. 11.20). A 70% diameter stenosis (90% cross-sectional area)

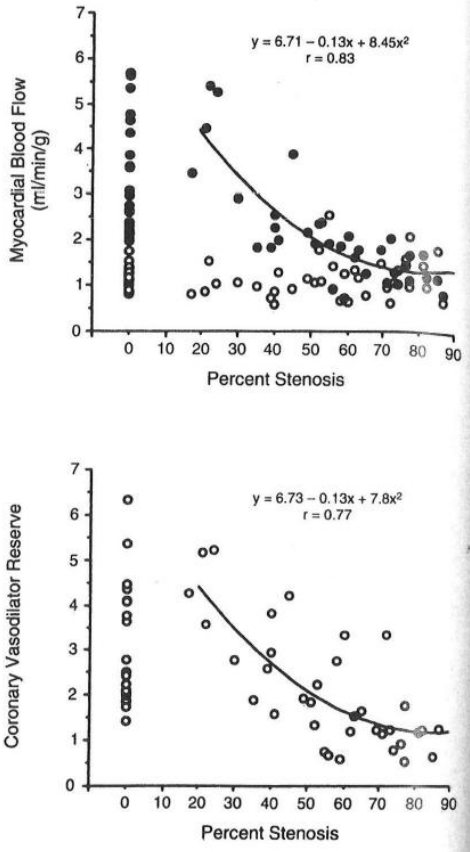

FIG. 11.20. Effect of coronary stenosis on myocardial blood flow and coronary vasodilator reserve. The top panel shows that resting flow *(open circles)* is well maintained at approximately 1 mL/min per gram of myocardium, throughout the range of evaluated diameter stenosis. The ability to increase flow during vasodilator stimulus *(closed circles)*, however, becomes impaired for stenosis greater than 50% and is virtually abolished at more than 70% stenosis. The bottom panel shows the vasodilator reserve (dilated flow/resting flow), which has a normal value of 3 to 4 but is reduced with stenosis greater than 50% and falls to 1 with stenosis greater than 70%. (From Uren NG, et al. Relation between myocardial blood flow and the severity of coronary artery stenosis. *N Engl J Med* 1994;330:1782, with permission).

eliminates virtually any ability to increase flow above the resting level (see Chapter 18). Stenoses that reduce the lumen diameter by 90%, however, rarely exist without reducing antegrade flow (i.e., grade 1 or 2, rather than grade 3 normal flow, on the TIMI [Thrombolysis in Myocardial Infarction] scale). Other than the subjective TIMI flow grading system, Gibson et al. (62) have created norms for the number of cine frames (at 30 frames per second) required for contrast material to leave the catheter tip and reach standardized distal landmarks in each coronary artery (e.g., the LAD "mustache," the first posterolateral branch of the right coronary). Contrast medium normally reaches these points in 20 frames for the RCA and 36 frames for the LAD. TIMI 2 (partial) flow corresponding to more than a doubling of those frame counts. Of course, even more precise data about hemodynamic lesion significance can be determined by performance of flow or pressure gradient measurements, at rest and during arteriolar vasodilation (e.g., after adenosine administration) to calculate the coronary flow or fractional flow reserve (63). Lesions that permit a flow increase of more than two-fold or that have a ratio of distal pressure to aortic pressure greater than 0.75 in the setting of peak flow after adenosine injection are generally considered not to be hemodynamically significant, and usually have a diameter stenosis less than 50% by quantitative angiography and no exercise perfusion defect on thallium scintigraphy (see Chapter 18).

In clinical practice, however, the degree of lesion stenosis usually is simply estimated visually from the coronary angiogram. The operator must develop a sense of what constitutes a 50%, 70%, or 90% diameter stenosis (Fig. 11.21). Although the process of visually estimating the degree of coronary stenosis may *seem* straightforward, it is subject to significant operator variability (the standard deviation for repeat estimates is up to 18%) (64), as well as a systematic form of "stenosis inflation" that causes operators to estimate a diameter stenosis that is roughly 20% higher than that measured by quantitative coronary angiography (65). A stenosis

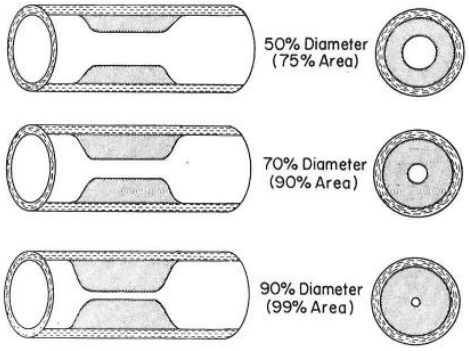

FIG. 11.21. Coronary stenoses of 50%, 70%, and 90% diameter reduction are shown in longitudinal and cross-section. The corresponding reductions in cross-sectional area are indicated in parentheses.

that measures 50% may typically be called 70%, and one that measures 70% may be called 90%.

Tools are available to resolve this problem. The simplest is to project the coronary image on a wall-mounted viewing screen and to use inexpensive digital calipers (available from machinist supply houses) to measure the relative diameters of the stenotic and reference segments (66). Percent stenosis then can be calculated as $100 \times [1 - (\text{stenosis diameter}/\text{reference diameter})]$ to provide a more accurate estimate. This technique also reduces the standard deviation for diameter stenosis 6% to 8% (64,66). Even greater precision can be obtained by using computer-assisted algorithms to perform automated edge detection on digitally acquired images to measure the coronary lumen with a standard deviation of less than 5% (67,68). The amount of variation in diameter stenosis readings for one study (69) using these different methods concurrently is shown in Fig. 11.22.

The good news is that angiographers who have trained their eye in actual stenosis quantification (by using digital calipers or computer-assisted quantitative coronary angiography) can then actually give visual estimates much closer to true measurements (70). This allows angiographers to be more uniform in their visual estimates and to move away from reporting physiologically impossible findings such as a 95%

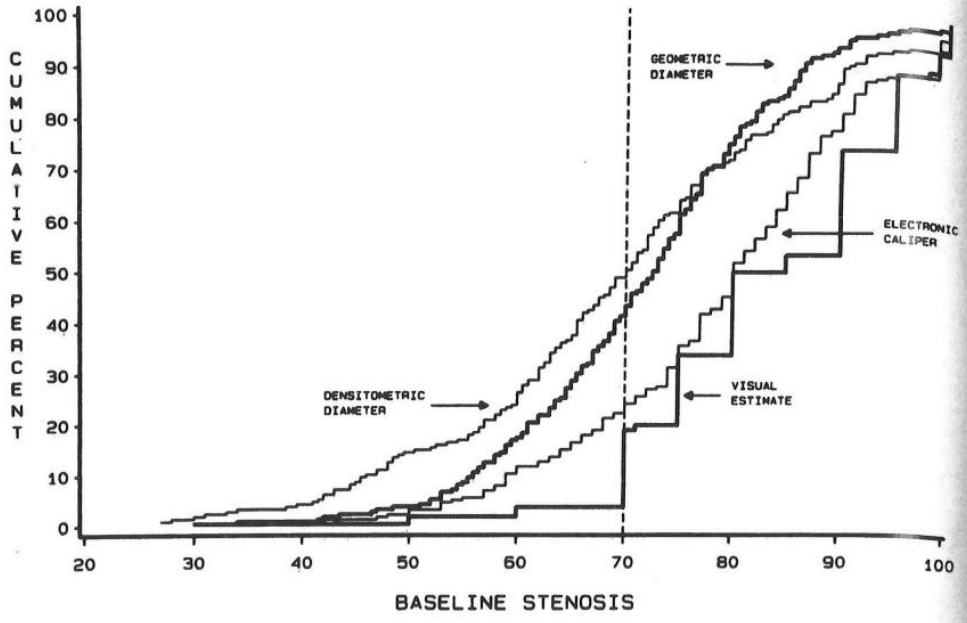

FIG. 11.22. In a series of 227 patients with single-vessel disease, visual estimates (*right curve*, average almost 90%) were consistently higher than either caliper measurements (average approximately 80%) or computer-assisted quantitative angiography by either geometric or densitometric techniques (*left curves*, average approximately 70% diameter stenosis). (From Folland ED, Vogel RA, Hartigan P, et al. Relation between coronary artery stenosis assessed by visual, caliper, and computer methods and exercise capacity in patients with single-vessel coronary artery disease. *Circulation* 1994;89:2005, with permission).

stenosis with normal distal flow. Until there is a ''stenosis reading reform,'' so that those of us who call such lesions accurately (e.g., 70%) will not be accused of intervening on mild lesions, there can be no substitute for ''seeing the films yourself'' before making any clinical decisions!

It has also become important to evaluate lesion morphology more accurately from the coronary angiogram. Features such as eccentricity, ulceration, and thrombus may be associated with unstable clinical patterns (71,72), and features such as calcification, eccentricity, or thrombus may influence the choice of catheter intervention. Many of these features can be recognized from careful study of high-quality cineangiograms, although angiography is clearly not as sensitive to these features as intravascular ultrasound (73) or angioscopy (for thrombus or dissection) (74). It may also be difficult to predict the physiologic significance of a coronary lesion from angiography alone, in which case it may need to be supplemented by other techniques such as direct flow or distal pressure measurements (63). Finally, the absence of lesions that narrow the coronary lumen by more than 50% does not necessarily confer immunity from subsequent coronary events, because it is frequently a less severe stenotic lesion that has a large lipid core and a thin fibrous cap that predisposes to subsequent plaque rupture and the resulting coronary thrombosis (75). Despite these recognized limitations in quantification and morphology assessment, contrast coronary angiography remains the clinical standard on which lesions are evaluated and decisions are made regarding the need for (and best mode of providing) revascu-

larization in the patient with ischemic heart disease.

Coronary Collaterals

In reviewing the coronary angiogram, one basic principle is that there should be evident blood supply to all portions of the left ventricle. Previously occluded vessel branches are usually manifested as truncated stumps, but no stump may be evident if there has been a flush occlusion at the origin of the involved vessel. These occluded or severely stenotic vessels are seen frequently to fill late in the injection by antegrade (so-called bridging) collaterals or collaterals that originate from the same (intracoronary) or an adjacent (intercoronary) vessel; this phenomenon is reviewed in an excellent paper by Levin (76) and illustrated in Figs. 11.23 through 11.25. Finally, coronary occlusion may manifest in some patients simply as an angiographically arid area to which there is no evidence of either antegrade or collateral flow and no evident vascular stump. If such an area fails to show re-

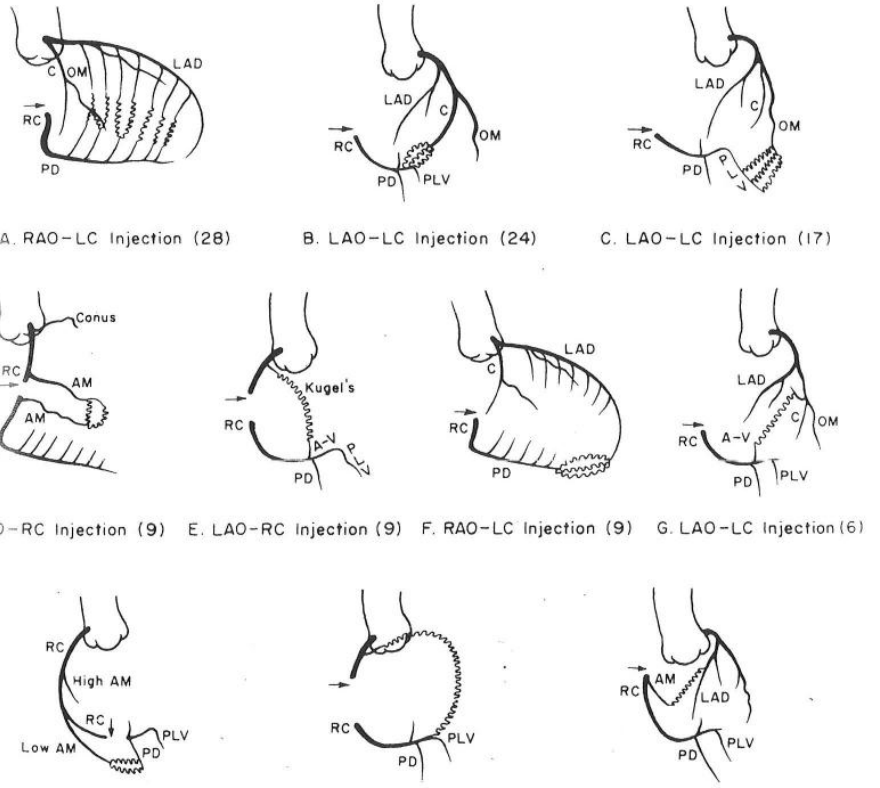

FIG. 11.23. Ten collateral pathways observed in patients with right coronary (RC) obstruction (total occlusion or greater than 90% stenosis). LAD, left anterior descending; C, circumflex; OM, obtuse marginal; PD, posterior descending; PLV, posterior left ventricular branch; AM, acute marginal branch of right coronary artery; A-V, atrioventricular nodal; LC, left coronary. Numbers in parentheses represent numbers of cases in this series. (From Levin DC. Pathways and functional significance of the coronary collateral circulation. *Circulation* 1974;50:831. By permission of the American Heart Association, Inc.)

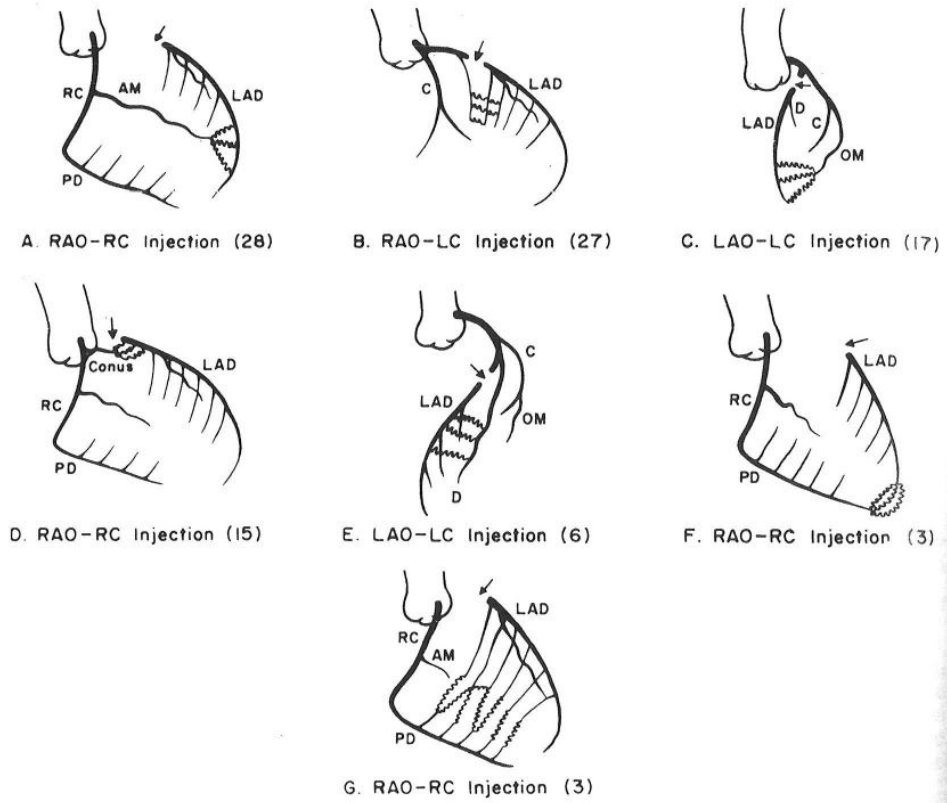

FIG. 11.24. Seven collateral pathways observed in patients with left coronary artery obstruction. Abbreviations and format are the same as in Fig. 11.23. (From Levin DC. Pathways and functional significance of the coronary collateral circulation. *Circulation* 1974;50:831. By permission of the American Heart Association, Inc.)

gional hypokinesis on the left ventriculogram, however, the operator should search carefully for blood supply by way of anomalous vessels or unopacified collaterals (i.e., a separate origin conus branch that was not opacified during the main right coronary artery injections), because the myocardium cannot continue to function normally with no visible means of support. Functioning collaterals can maintain a coronary wedge pressure that averages almost 40% of mean aortic pressure (77,78), thereby maintaining myocardial viability in the collateral-fed distribution. Along with other measures of retained or augmentable wall motion, redistributing defects on perfusion imaging, and positron emission tomographic (PET) evidence of ongoing glucose metabolism, the angiographic presence of collateral flow to an area in the distribution of an occluded coronary artery is one of the strongest evidences of ongoing myocardial viability and an important factor in determining the best revascularization strategy.

Although it is uncommon, what appears to be a network of collaterals may be the vascular supply to an organized thrombus (in the left ventricle or left atrium) or a cardiac tumor. Those entities should be suspected when filling of an apparent collateral network is seen in the absence of occlusion or severe stenosis of the normal supply to a myocardial territory.

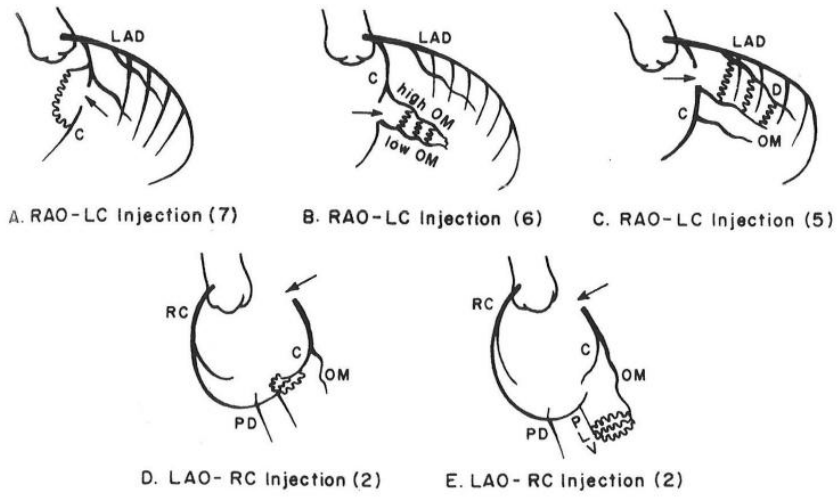

FIG. 11.25. Five collateral pathways observed in patients with circumflex coronary artery obstruction. Abbreviations and format are the same as in Fig. 11.23. (From Levin DC. Pathways and functional significance of the coronary collateral circulation. *Circulation* 1974;50:831. By permission of the American Heart Association, Inc.)

NONATHEROSCLEROTIC CORONARY ARTERY DISEASE

Although atherosclerotic stenosis is by far the most common pathologic process responsible for myocardial ischemia, the angiographer must be aware of a variety of other potential causes (79). These include certain congenital anomalies of coronary origin (46,80–82), such as an anomalous coronary artery that courses between the aorta and the pulmonary artery, in which flow may be compromised by deformation of the ostium or compression of the proximal vessel, potentially even causing sudden death. Other abnormalities include coronary fistulas (Fig. 11.26), coronary aneurysms (83,84), and muscle bridges (Fig. 11.27) (85,86). *Coronary fistulas*, connections mostly from a coronary artery to the right ventricle, right atrium, pulmonary artery, or coronary sinus, are found in roughly 0.1% of patients coming to cardiac catheterization. When they are large (or in the setting of proximal coronary disease), these fistulas may cause chronic volume overload or myocardial ischemia and must be closed, using surgery or newer catheter techniques (e.g., embolization coils, covered stents) (87). Smaller, asymptomatic fistulas may close spontaneously and can be managed conservatively (88). *Muscle bridges* are sections of a coronary artery (almost always the LAD) that run under a strip of left ventricular muscle, which compresses the lumen during ventricular systole despite a normal appearance during diastole (85,86). Similar systolic compression of the first septal branch (saw-toothing) is also seen in many patients with hypertrophic cardiomyopathy (89). *When one of these congenital anomalies is present in a patient with ischemic symptoms in whom catheterization has failed to demonstrate the expected finding of coronary atherosclerosis, the angiographer should be able to recognize it as a potential cause of ischemia and recommend additional functional testing with an eye toward surgical or catheter-assisted repair* (e.g., *fistula coil embolization, muscle bridge stent placement).*

The coronary arteries may also be affected by medium-size-vessel *vasculitis* (90), including polyarteritis nodosa and the mucocutaneous lymph node syndrome (Kawasaki disease). The latter is largely a childhood illness, in which cor-

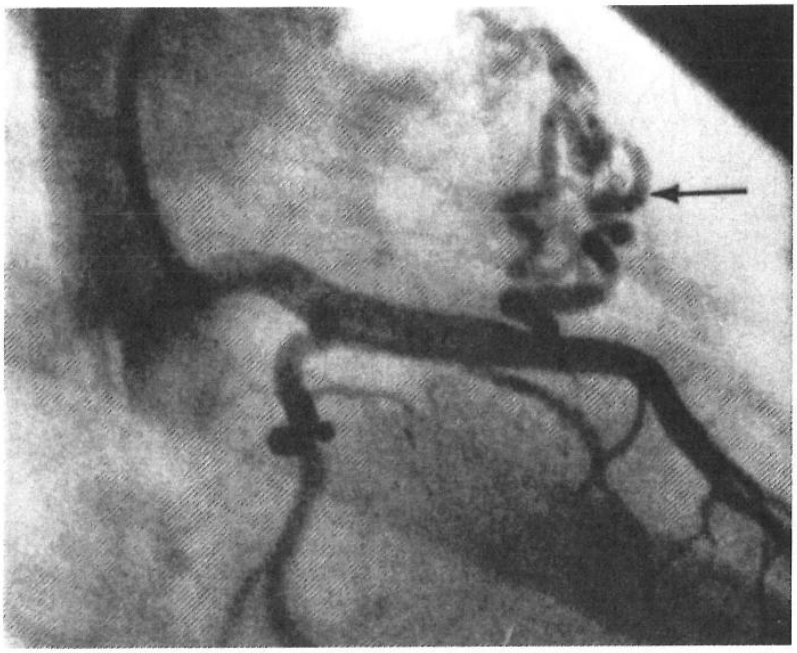

FIG. 11.26. Coronary artery fistula *(arrow)* between the middle left anterior descending coronary artery and the pulmonary artery, shown in the right anterior oblique view.

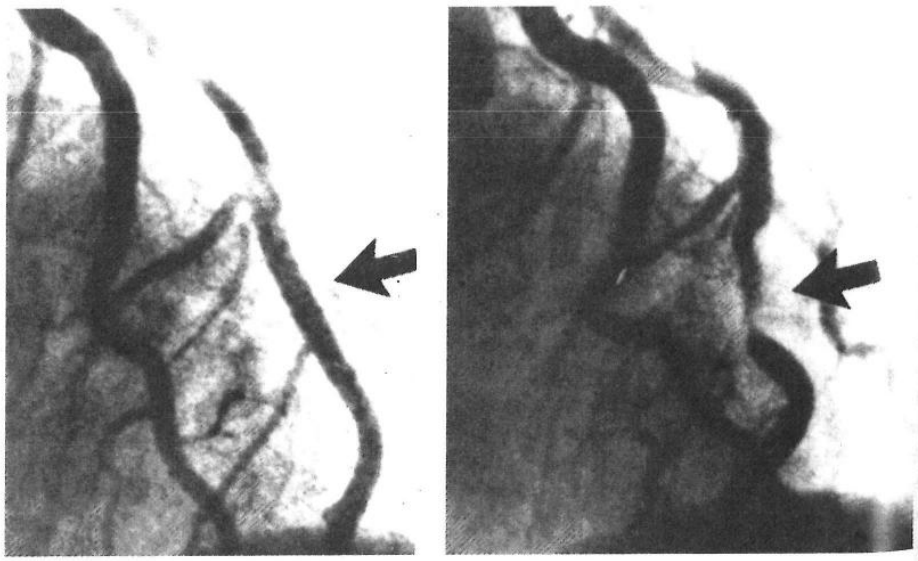

FIG. 11.27. Muscle bridge. Moderately severe muscle bridge of the left anterior descending coronary artery *(arrows)* as seen in diastole *(left)* and systole *(right)*.

onary arteritis may lead to aneurysm, stenosis, or thrombosis that, before the use of high-dose gamma globulin to treat the acute illness, was often fatal (usually in the first month of the illness). When coronary aneurysms are found in adults, it may be difficult to determine whether they represent atherosclerotic damage to the vessel wall or the remainders of childhood Kawasaki disease (83). However, the treatment for the stenotic lesions (bypass or catheter-based intervention) is the same regardless of the cause. Although not an arteritis, *cardiac allograft vasculopathy* (91) is one of the most troublesome long-term complications of heart transplantation. The mechanism seems to be an immune-mediated diffuse vascular proliferative response involving distal as well as proximal coronary arteries, with superimposed focal lesions of the proximal vessels. The latter may be amenable to catheter-based revascularization. Patients who have received prior mantle radiation therapy for Hodgkin disease may be at risk for *radiation-induced coronary stenosis* (92), particularly of the left and right coronary ostia and the proximal left coronary artery, up to 20 years after completing their course of therapy. The pathology is most commonly fibrotic contraction of the vessel wall, rather than intimal proliferation or plaque formation.

Finally, some patients who come to catheterization have no demonstrable coronary abnormality to account for their clinically suspected ischemic heart disease. Although angina-like pain can be seen in patients with noncoronary cardiac abnormality (e.g., mitral valve prolapse, hypertrophic cardiomyopathy, aortic stenosis, myocarditis) or extracardiac conditions (esophageal dysmotility (93), cholecystitis), one must also consider the possibility of coronary vasospastic disease (94).

Coronary Vasospasm

Vasospasm of an epicardial coronary artery typically presents as variant (Prinzmetal) angina in which episodes of rest pain occur despite well-preserved effort tolerance at other times (94). An ECG recorded during an episode of spontaneous pain usually shows ST elevation in the territory supplied by the vasospastic artery. Absence of a significant coronary lesion in such a patient confirms the diagnosis of variant angina due to focal coronary spasm (Fig. 11.28). In these patients, coronary angiography is performed mainly to look at the extent of underlying atherosclerosis (95); we generally do not attempt to provoke spasm in such patients as was once done to evaluate drug therapy (96). When the diagnosis of variant angina is uncertain and a patient with troublesome chest pain fails to manifest sufficient disease to explain its origin, however, provocative testing for coronary spasm may be helpful.

If provocational testing for coronary spasm is contemplated, the patient should be withdrawn from calcium channel blockers for at least 24 hours and long-acting nitrates for at least 12 hours before the study and should not be premedicated with either atropine or sublingual nitroglycerin. Ongoing therapy with any of these agents may render provocative tests falsely negative (96). Although a variety of provocative tests have been used (methacholine, epinephrine and propranolol, hyperventilation and tris-buffer, cold pressor), the most commonly used provocative agents are ergonovine and methylergonovine maleate (Methergine, Sandoz, East Hanover, NJ) (97–99), stimulants of the α-adrenergic and serotonin receptors in coronary vascular smooth muscle. Ergonovine is no longer generally available in the United States, and the availability of methylergonovine is limited. If it is not available, one easy alternative provocative test is hyperventilation (100), done vigorously for 6 minutes in the early morning (between 6 and 8 a.m., when spasm is more active). This produces spasm in most patients with variant angina and can obviate the need for further drug testing. Although this finding is 100% specific for coronary spasm, it is only 62% sensitive. If spasm is suspected but has not been elicited by hyperventilation, and if methylergonovine is not available, further testing can be performed using intracoronary acetylcholine (50 to 100 μg injected into the left coronary or 20 to 50 μg injected into the right coronary artery).

Testing for coronary spasm should be performed only after baseline angiographic evalua-

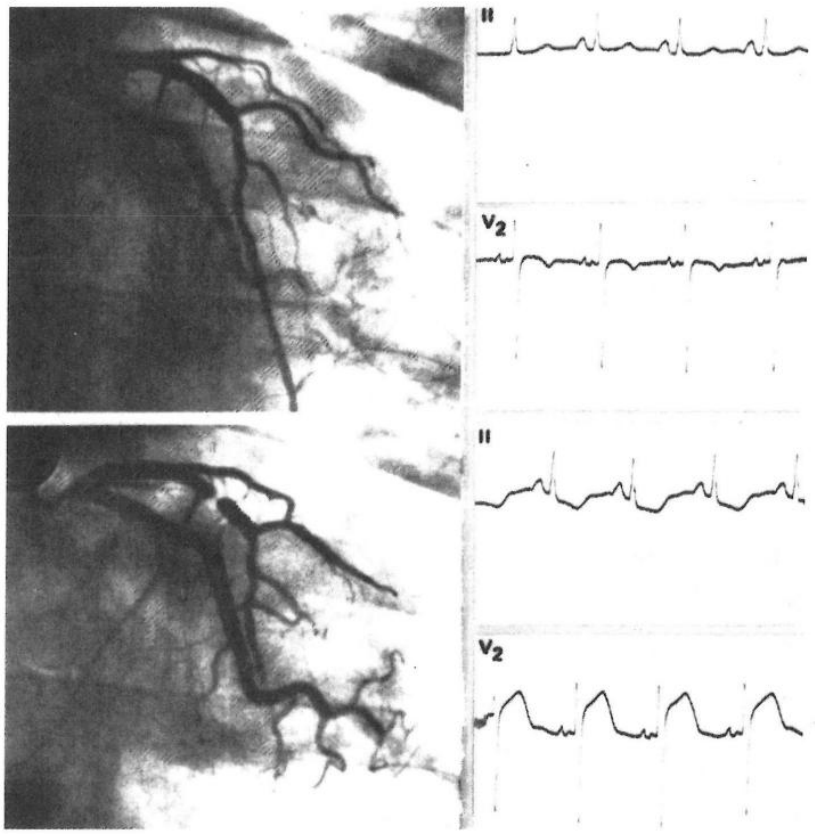

FIG. 11.28. True coronary spasm. Intense focal vasospasm of the left anterior descending coronary artery is shown in right anterior oblique projection in a patient with variant angina. Note the absence of a significant underlying atherosclerotic stenosis in the top panel, the absence of vasoconstriction of other vessel segments, and the marked ST elevation in the anterior leads during the spontaneous vasospastic episode. (From Baim DS, Harrison DC. Nonatherosclerotic coronary heart disease. In: Hurst JW, ed. The Heart, 5th ed. New York: McGraw-Hill, 1985, with permission.)

tion of both the left and right coronary arteries. It should not be performed in patients with severe hypertension or severe anatomic cardiac pathology (left ventricular dysfunction, left main or multivessel disease, aortic stenosis). As an example, our protocol for using ergonovine calls for a total of 0.4 mg (400 μg = 2 ampules) of ergonovine maleate to be diluted to a total volume of 8 mL in a 10-mL syringe that is appropriately labeled. The provocative test consists of graded intravenous administration of 1 mL (0.05 mg), 2 mL (0.10 mg), and 5 mL (0.25 mg) of this mixture at 3- to 5-minute intervals. Parenteral nitroglycerin (100 to 200 μg/mL) must be premixed and loaded in a labeled syringe before the testing is begun. At 1 minute before each ergonovine dose, the patient is interrogated about symptoms similar to those of her or his clinical complaint, and a 12-lead ECG is recorded. After each ECG, coronary angiography is performed, looking either at both arteries or only at the artery of highest clinical suspicion for vasospasm. In the absence of clinical symptoms, ECG changes, or focal coronary vaso-

spasm exceeding 70% diameter reduction, the next ergonovine dose is administered, and the cycle is repeated until the total dose of 0.4 mg has been given. Some operators have employed an *intracoronary* methylergonovine administration protocol, in which doses of 5 to 10 μg are given into a coronary artery, and after 3 minutes imaging studies are performed before a second dose is given (maximal total dose 50 μg). This may be advantageous in that it produces less systemic effect (e.g., hypertension, esophageal spasm).

If provocative testing produces *clinical* symptoms, but no ECG changes or angiographic evidence of vasospasm in either coronary artery, an alternative diagnosis such as esophageal dysmotility is suggested (93). Even if there are no symptoms or ECG changes, both coronary arteries should be opacified at the end of the provocative test, and any generalized vasoconstrictor effect should be terminated by administration of nitroglycerin. Coronary artery spasm may occur in two vessels simultaneously (Fig. 11.29), and visualization of only one vessel may fail to adequately assess the magnitude of the vasospastic response. The provocative test should be considered positive only if focal spasm (greater than 70% diameter stenosis) occurs and is associated with clinical symptoms and/or ECG changes. The patient is then treated immediately with parenteral nitroglycerin, 200 μg administered either by vein or, preferably, directly into the spastic coronary artery. The involved artery should then be reopacified 1 minute after nitroglycerin administration, to document the resolution of spasm and the extent of underlying atherosclerotic stenosis. The operator should be prepared to use additional doses of parenteral nitroglycerin, sublingual nifedipine, or sodium nitroprusside to treat refractory spasm or the occasional severe hypertensive reaction that can occur after ergonovine administration. Low doses of intracoronary verapamil (100 to 200 μg) or diltiazem (500 to 1,000 μg) have also proved useful in refractory spasm, although care must be taken to avoid excessive bradycardia or myocardial depression. Temporary pacing and defibrillatory equipment should also be available to treat the bradyarrhythmias or tachyarrhythmias that sometimes accompany coronary spasm. Because finding spasm is so uncommon now that the syndrome is detected clinically in most patients, and because it is treated so effectively by calcium channel blockers, the risk of ergonovine testing to evaluate patients with atypical symptoms and minimal fixed coronary disease is remarkably low. In the Duke study of ergonovine testing in 3,447 patients without significant coronary disease or variant angina, significant complications occurred in only 11 patients (0.03%), including myocardial infarction in 4 patients and ventricular tachycardia/ventricular fibrillation in 7 (101).

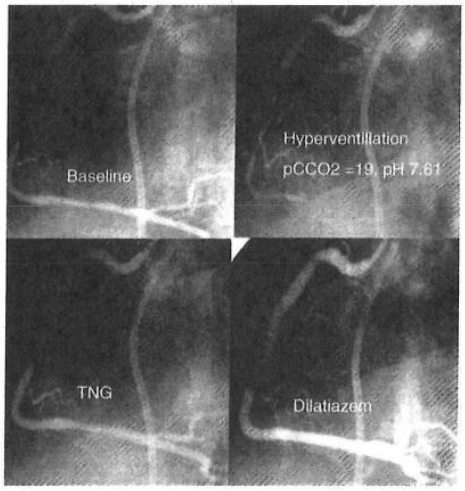

FIG. 11.29. This 37-year-old man was admitted with rest pain and ST-segment elevation in the inferior leads. Emergency catheterization was performed for presumed acute myocardial infarction within 30 minutes after presentation *(upper left)*. It disclosed a dominant right coronary artery with only mild disease at a time when pain had resolved after nitrate and heparin therapy. Hyperventilation (30 breaths per minute for 5 minutes) was performed with reduction of the partial pressure of carbon dioxide (P_{CO_2}) to 19 mm Hg and elevation of the pH to 7.61, resulting in provocation of occlusive focal spasm of the distal right coronary artery with return of chest pain and ST segment elevation *(upper right)*. Relief of vasospasm and marked general dilation of the right coronary artery were produced by intracoronary administration of trinitroglycerin 200 μg *(lower right)* and diltiazem 500 μg *(lower right)*.

Several additional comments about ergonovine are in order. Our group avoids ergonovine testing in patients with severe atherosclerotic stenosis (80% or greater), in whom spasm is not required to explain the clinical symptoms. In these patients, however, we frequently do repeat coronary angiography of the stenotic vessel after the intracoronary administration of 200 mg of nitroglycerin, to exclude the possibility that spontaneous focal vasospasm is contributing to the appearance of severe atherosclerotic stenosis. Second, the operator should be aware that the positivity rate of ergonovine testing depends strongly on which patients are studied; the test is almost always positive in patients with known variant angina (if their disorder is active and medications have been withheld) and is positive in approximately one third of patients with clinically suspected variant angina, but it is positive in fewer than 5% of patients whose symptoms do not suggest variant angina (99). The Duke group (101) reported an overall positivity rate of 4% in such patients, with two independent predictors of a positive test: mild to moderate disease on the angiogram (spasm often takes place at the point of such disease) and a history of smoking, whose presence increased the positivity rate to 10%. It is also important to distinguish the intense focal spasm seen in patients with variant angina from the normal mild (15% to 20%) diffuse coronary narrowing seen as a pharmacologic response to ergonovine in normal patients (102). True coronary spasm must also be distinguished from spasm induced by mechanical interventions such as rotational atherectomy (see Chapter 28) or catheter tip spasm (Fig. 11.30). Catheter tip spasm is most common in the right coronary artery, is not associated with clinical symptoms or ECG changes, and does not indicate variant angina (103). It should

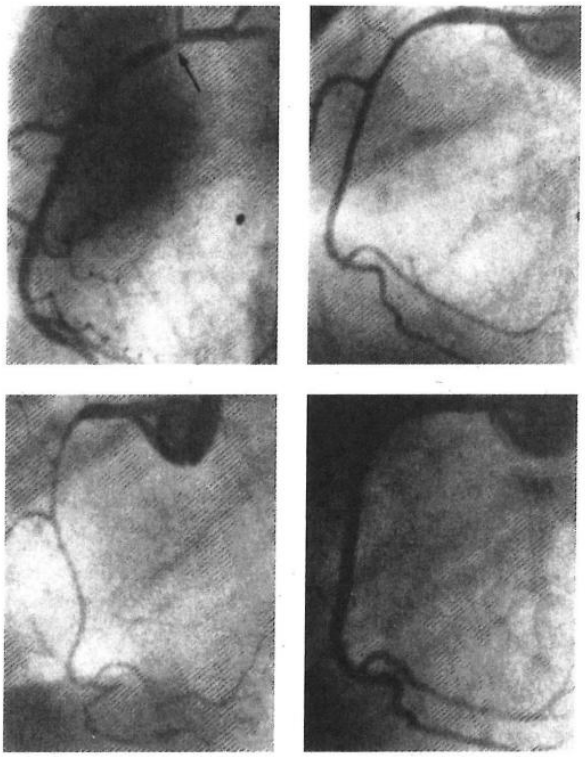

FIG. 11.30. Vasomotor changes not representing true coronary spasm. During right coronary catheterization with a Judkins catheter *(upper left)*, this patient developed severe catheter-tip spasm. Recatheterization 24 hours later with an Amplatz catheter *(upper right)* showed neither catheter-tip spasm nor an atherosclerotic stenosis. After administration of ergonovine 0.4 mg, marked diffuse coronary narrowing was observed *(lower left)*, without angina or electrocardiographic changes. After the intracoronary administration of nitroglycerin 200 μg *(lower right)*, there is marked diffuse vasodilation.

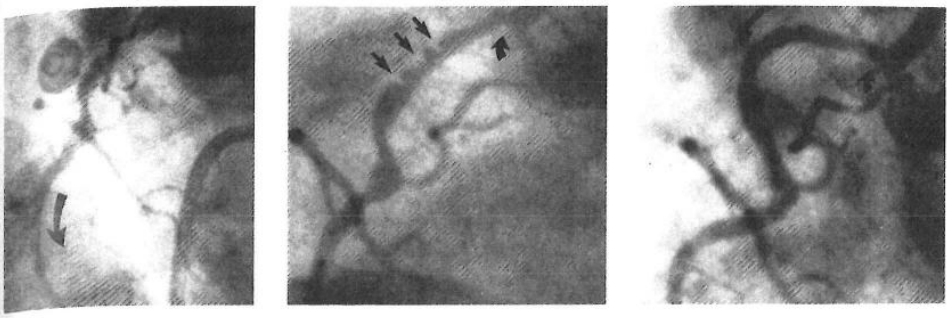

FIG. 11.31. Right coronary artery "pleating" artifact. Baseline injection *(left)* shows diffuse disease in this tortuous right coronary artery selected for rotational atherectomy. Middle panel shows straightening of the proximal vessel by the stiff "type C" wire, creating three areas of infolding of the vessel wall *(arrows)*, as well as the appearance of ostial stenosis *(curved arrow)*. Immediately on withdrawal of the guidewire, the artery returned to its baseline curvature, and these defects resolved *(arrows)*.

be recognized as such, however, and treated by withdrawal of the catheter, administration of nitroglycerin, and nonselective or cautious repeat selective opacification of the involved vessel, to avoid mistaking catheter-tip spasm for an atherosclerotic lesion. Spasm should also be distinguished from a "pleating" artifact that may occur when a curved artery is straightened out by a stiff guidewire (Fig. 11.31), causing folds of the vessel wall to impinge on the lumen. Pleating is refractory to nitroglycerin but resolves immediately when the stiff guidewire is withdrawn (104).

Abnormal Coronary Vasodilator Reserve

Evidence has been accumulating that the population of patients with angina and angiographically normal coronary arteries may contain a subgroup of patients who have myocardial ischemia on the basis of abnormal vasodilator reserve. Despite angiographic normality, intravascular ultrasound examination may show normal vessel wall architecture, intimal thickening, or atheromatous plaque (84). In these patients, coronary blood flow (as described in Chapter 18) may fail to rise normally with pacing tachycardia or exercise, and the coronary vascular resistance is increased abnormally (105). Also, many of these patients show an abnormal rise in left ventricular end-diastolic pressure after pacing tachycardia and show less lactate consumption than normal subjects in response to pacing tachycardia (106). A failure of small vessel coronary vasodilation, inappropriate vasoconstriction at the arteriolar level, or functional abnormalities of capillary endothelial cells in releasing endothelium-derived relaxing factor (EDRF) have been postulated to account for these findings. Many patients with so-called syndrome X respond at least partially to treatment with a calcium channel blocker, but other patients show no clear flow or contractile abnormality, so that the primary problem may be simply increased sensitivity to pain (11).

MISTAKES IN INTERPRETATION

An inexperienced operator often produces an incomplete or uninterpretable study, especially if poor equipment is used. Such an operator is also likely to misinterpret the angiographic findings, with potentially serious clinical consequences. The following discussion summarizes some of the most common pitfalls that may lead the inexperienced coronary angiographer to mistaken conclusions.

Inadequate Number of Projections

There is no standard number of projections that will always provide complete information.

Each major vessel must be viewed in an isolated fashion as it stands apart from other vessels. Usually, the angulated views discussed earlier in this chapter are necessary to visualize clearly the anatomy of the proximal LAD and circumflex arteries.

Inadequate Injection of Contrast Material

The inexperienced operator or assistant has a tendency to hold back on the volume and force of injection into the coronary circulation. This results in inadequate or intermittent, pulsatile opacification of the coronary arterial tree, because contrast flow fails short of peak coronary flow during diastole. There is inadequate mixing of contrast agent and blood, and pockets of nonradiopaque blood in such inadequate injections may even give the appearance of arterial narrowing.

Superselective Injection

It is not uncommon to catheterize the LAD or circumflex coronary artery superselectively, especially when the left main coronary artery is short and its bifurcation is early. To the inexperienced operator, this may give the impression of total occlusion of the nonvisualized vessel (e.g., if only the circumflex artery is opacified, the operator may conclude that the LAD artery is occluded). If adequate filling of the noncannulated vessel cannot be achieved by reflux, selective cannulation of the LAD may be obtained by counterclockwise rotation or use of a Judkins catheter of the next smaller size (e.g., JL3.5), whereas selective cannulation of the circumflex may be obtained by clockwise rotation or use of the next larger size (e.g., JL5). With the right coronary artery, superselective injection may occur if the catheter tip is too far down the vessel, leading to failure to visualize the conus and sinus node arteries. Because these are important sources of collateralization of the left coronary system, important information may be missed (Fig. 11.13). Injection that is adequate to provide a continuous (nonpulsatile) reflux of contrast agent back into the sinus of Valsalva helps the operator to recognize vessels that originate proximally to the catheter tip and thereby avoid the interpretation error of superselective injection.

Selective cannulation of a coronary artery may also fail to detect significant ostial stenosis, particularly if the catheter tip lies beyond the lesion and adequate contrast reflux is not produced. If ostial stenosis is suspected (e.g., if there is partial ventricularization or damping), we have found it helpful to perform a final injection during withdrawal of the catheter from the ostium (Fig. 11.32).

Catheter-induced Coronary Spasm

Coronary artery spasm may be related to the catheter itself, possibly caused by mechanical irritation and a myogenic reflex (Fig. 11.30). It is seen most commonly when the right coronary artery is engaged selectively, although it may occur rarely in the LAD artery as well. Although catheter-tip spasm can occur with either the brachial or femoral approach, it is probably more common with the right Judkins catheter, especially if the catheter tip enters the right coronary ostium at an angle and produces tenting of the proximal vessel. If coronary narrowing suggests the occurrence of spasm to the operator, sublingual, intravenous, or intracoronary nitroglycerin should be given and the injection repeated.

Congenital Variants of Coronary Origin and Distribution

This topic has been discussed earlier in this chapter, but it bears reemphasis. Variation in origin and distribution of the coronary artery branches may confuse the operator and lead to a mistaken diagnosis of coronary occlusion. For example, a small right coronary artery that terminates in the AV groove well before the crux may be interpreted as an abnormal or occluded artery, whereas it is a normal finding in 7% to 10% of human hearts. Double ostia of the right coronary artery or origin of the circumflex artery from the right coronary artery may be similarly confusing and lead to misdiagnosis.

Myocardial Bridges

As discussed earlier, coronary arteries occasionally dip below the epicardial surface under

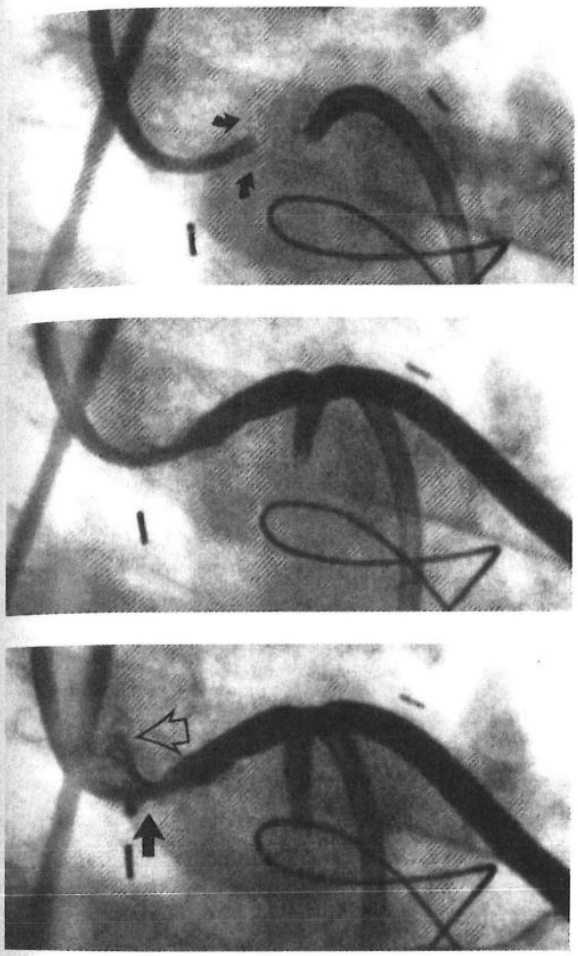

FIG. 11.32. Masking of ostial stenosis during superselective cannulation. Ostial stenosis of previously stented vein graft is not apparent with the tip of the catheter well beyond the stenosis *(top and middle panels)*. Continued injection during catheter withdrawal *(bottom panel)* causes reflux into the aorta *(solid arrow)* and clearly shows significant ostial stenosis.

small strips of myocardium. During systole, the segment of the artery surrounded by myocardium is narrowed and appears as a localized stenosis. These myocardial bridges occur most commonly in the distribution of the LAD artery and its diagonal branches. The key to recognition of these bridges is that the apparent localized stenosis returns to normal during diastole. Studies using the flow wire (see Chapter 18) show clear derangement in phasic flow dynamics in muscle bridge segments, and their normalization by stent placement. Although some severe muscle bridges can therefore cause true myocardial ischemia under certain circumstances, they are seen in at least 5% of normal angiograms obtained in patients with no evidence of ischemia in the LAD territory.

Total Occlusion

If a coronary artery or branch is totally occluded at its origin, it may not be visualized, and the occlusion may be missed. If the occlusion is flush with the parent vessel, no stump will be seen. Such occlusions are recognized primarily by visualization of the distal segment of the oc-

cluded vessel by means of collateral channels or by noting the absence of the usual vascularity seen in a particular portion of the heart.

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23

Coronary Angioplasty

Donald S. Baim

Harvard Medical School; Center for Innovative Minimally Invasive Therapy, Brigham and Women's Hospital, Boston, Massachusetts 02115

The concept of transluminal angioplasty—enlargement of the lumen of a stenotic vessel by a catheter technique—was first proposed by Dotter and Judkins in 1964 (1). Their idea was to advance a spring-coil guidewire across an atherosclerotic arterial stenosis. As this guidewire remained in place, it would serve as a rail over which a series of progressively larger rigid dilators could be advanced to enlarge the vessel lumen. Although this technique proved to be effective in peripheral arteries, the need to insert large-caliber rigid dilators through the arterial puncture (and the high shear forces applied by the dilators as they crossed the atherosclerotic lesion) ultimately limited the clinical application of this "Dotter" technique. In 1974, Gruentzig (2) modified the technique, by replacing the series of rigid dilators with an inflatable nonelastomeric balloon mounted on a comparatively smaller catheter shaft. The balloon catheter could be introduced percutaneously with minimal trauma, advanced easily across a vascular stenosis in its smaller (collapsed) state, and then inflated with sufficient force to enlarge the stenotic lumen. Although others had speculated about the possibility of balloon dilatation, Gruentzig was the first to develop it into a practical device, and perfect it as a usable clinical tool. Within 4 years he and his colleagues (3) had performed a series of experiments in animals, cadavers, peripheral arteries, and the coronary arteries of patients undergoing bypass surgery, culminating in their performance of the first percutaneous balloon angioplasty of a stenotic coronary artery in a conscious human (September 16, 1977).

Although the new technique of balloon angioplasty was viewed with skepticism by most, a small number of cardiologists around the world recognized the great potential it might hold (4). In 1979 they met to form a registry of all coronary angioplasty cases worldwide under the sponsorship of the National Heart, Lung, and Blood Institute (NHLBI). That registry grew to 3,000 cases by 1981, although no more than 1,000 angioplasties were performed in any given year during that period. From these humble beginnings, progressive improvements in equipment and technique have produced dramatic growth in percutaneous transluminal coronary angioplasty (PTCA), transforming it into a major therapeutic modality that is used to benefit large numbers of patients with ischemic syndromes caused by anatomically suitable coronary artery lesions (5) (Fig. 23.1). Roughly 35% of the 1.4 million people who undergo diagnostic cardiac catheterization each year are referred for revascularization by catheter-based techniques exceeding the number referred for revascularization by bypass surgery. Despite a progressive broadening in its clinical and anatomic indications, the success rate for coronary angioplasty has risen to 98%, with a procedural mortality rate and an emergency bypass rate each less than 1%.

Much of the improvement over the past 6 to 8 years, however, has come from the introduction of new adjunctive technologies such as ath-

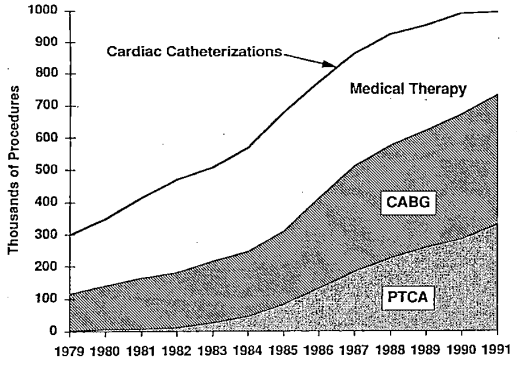

FIG. 23.1. Growth in the number of coronary angioplasty procedures between 1979 and 1991 is shown by the bottom *(stippled)* band, increasing from less than 1,000 per year in 1979–1981 to more than 300,000 per year in 1991. This was similar to the annual number of bypass operations *(cross-hatched band).* The number of annual catheterizations has now grown to approximately 1.4 million, with one of three patients who undergo diagnostic cardiac catheterization being referred for coronary angioplasty, compared with one of four being referred for bypass surgery. CABG, coronary artery bypass grafting; PTCA, percutaneous transluminal coronary angioplasty. (From American College of Cardiology; see also Bittl JA. Advances in coronary angioplasty. *N Engl J Med* 1996;335:1290.)

erectomy and stent implantation (see Chapters 24 and 25), as well as refinements in anticoagulant and antiplatelet pharmacology. Newer devices are now used in more than 80% of coronary interventions, so that stand-alone plain old balloon angioplasty (POBA) has become a minority interventional procedure. But balloon predilation or postdilation still plays an important *adjunctive* role in almost all such procedures (5), and the roots for our understanding of the strengths and weaknesses of any percutaneous coronary intervention lie in the history of balloon angioplasty. The intent of this chapter is to examine the basic equipment, strategy, results, and current indications of balloon angioplasty, indicating the situations in which newer stent and atherectomy devices have become dominant through their ability to address the previous limitations of stand-alone balloon angioplasty (6). Even more importantly, this chapter makes it clear that the evolution of catheter-based intervention is continuing, at an ever-increasing pace. The coronary interventionalist must have strong grounding in these fundamental principles, as well as the flexibility to master rapidly changing techniques and indications as they develop, if his or her patients are to receive the greatest benefits of safety, predictability, and durability from percutaneous coronary intervention.

EQUIPMENT

A coronary angioplasty system consists of three basic components (Fig. 23.2): (a) a guiding catheter, which provides stable access to the coronary ostium, a route for contrast administration, and a conduit for the advancement of the dilatation equipment; (b) a leading guidewire that can be passed through the guiding catheter, across the target lesion, and well into the distal coronary vasculature to provide a rail over which a series of therapeutic devices can be advanced; and (c) a nonelastomeric balloon dilatation catheter filled with liquid contrast medium. Technologic advances lead to refinements in specific equipment each year, so any detailed description of current products would be outdated too soon to be of value here, but some general principles hold true.

Guiding Catheters

Guiding catheters remain a crucial component in PTCA. Compared with the small lumens, minimal torque control, and sharp edges of the crude initial Teflon guiding catheters, current designs more closely emulate the performance of catheters used for diagnostic coronary angiography. To allow passage of therapeutic instruments, however, guiding catheters must have a lumen diameter at least twice that of a typical diagnostic catheter (e.g., 0.080-inch [2 mm] vs. 0.040-inch [1 mm]). To achieve this lumen in a catheter whose outer diameter is 8F (2.7 mm, or 0.107-inch), the wall thickness must be less than 0.010-inch (0.5 mm). Yet the catheter must still incorporate a Teflon liner to reduce friction, metal or plastic braid to transmit torque and provide sufficient stiffness to offer "backup" support during device advancement, and a smooth outer

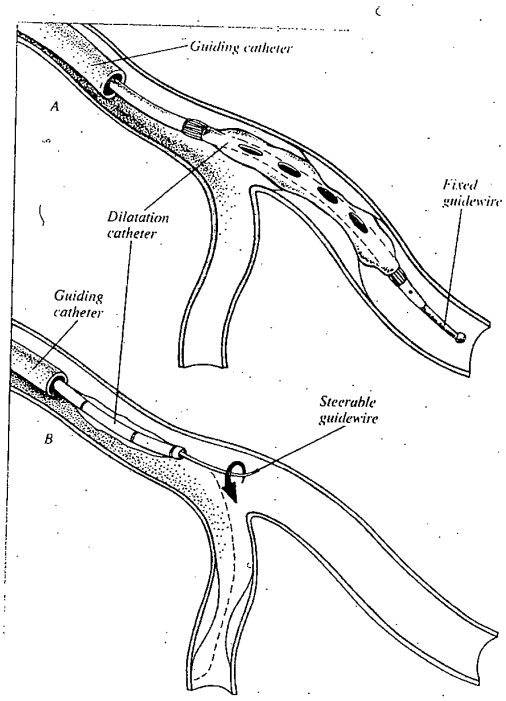

FIG. 23.2. Components of the coronary angioplasty system. The original Gruentzig fixed guidewire balloon **(A)** is compared with the steerable guidewire system **(B)**. Although both are advanced through a guiding catheter positioned in the coronary ostium, neither the wire shape nor its orientation could be changed once the original Gruentzig catheter was introduced, whereas the steerable design allows the guidewire to be advanced, withdrawn and reshaped, and steered independently of the balloon catheter to select the desired vessel. Once in place in the distal vessel beyond the target lesion, the guidewire serves as a rail over which the angioplasty balloon or other device can be advanced. (From Willerson JT, ed. *Treatment of heart diseases.* New York: Gower Medical, 1992.)

coating to resist thrombus formation. The complexity of this design goal requires use of special materials whose properties are typically varied along the length of the catheter to optimize the balance between support and flexibility at each point. Most guiding catheters now also include a very soft material in the most distal 2 mm of the catheter to reduce the chance of vessel trauma during engagement of the nontapered tip. Current guiding catheters are available in shapes similar to conventional Judkins and Amplatz curves, as well as a wide range of custom shapes, such as hockey-stick, multipurpose, and Voda (7), that are designed to ease engagement or provide better support for balloon advancement.

Although 9F guiding catheters predominated in the early 1980s, 8F (2.7 mm) catheters are in common use today. Improvements in catheter design have enabled routine lumen diameters of 0.088 inches in 8F guiding catheters to facilitate passage of bulkier devices such as stents and Rotablator burrs. Smaller (7F and even 6F) guiding catheters are also available, with a more restricted inner diameter (0.076 inches or 1.9 mm), well suited to the current generations of balloon catheters and bare-mounted stents and ideal for use from alternative access sites such as the radial artery (see Chapter 4). Larger (9F and 10F) guiding catheters with lumen diameters up to 3 mm (0.120 inch) are still used occasionally for certain procedures such as directional or extraction atherectomy. The standard guiding catheter length is 100 cm, but shorter (90 cm) guides are available to allow more distal passage of devices with limited working lengths during procedures on distal lesions in saphenous vein or internal mammary grafts (see later discussion). Much of the technology developed for interventional guiding catheters (e.g., thin walls, soft tips) has now been fed back into the design of diagnostic catheters to allow safer coronary engagement and brisk contrast injections through 6F (and even 5 or 4F) catheters.

To function adequately, the guiding catheter must be able to selectively engage the ostium. This requires the selection of an appropriate catheter shape and the ability to manipulate the catheter under fluoroscopic guidance (see Chapter 11). Engagement of the desired vessel, however, must not interfere with arterial inflow. Although this is routinely possible in the left coronary artery, damping of the guiding catheter in the right coronary artery ostium was once a common and vexing problem before the introduction of guiding catheters equipped with sideholes that allow ongoing perfusion despite wedged engagement. However, because the guiding catheter must deliver small boluses of contrast medium into the involved vessel (as needed to visualize vascular side branches and

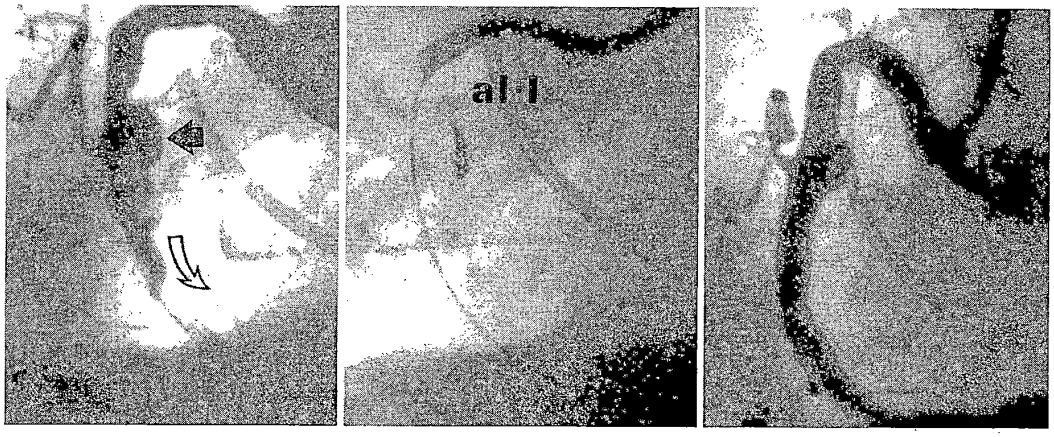

FIG. 23.3. Use of deep-guiding catheter engagement to facilitate coronary intervention. **Left:** Complex lesion in the right coronary artery including aneurysm *(dark arrow)* and diffuse distal disease *(open curved arrow)*. **Center:** Left Amplatz guiding catheter (AL-1) is deeply engaged to provide optimal support for stent placement. **Right:** After stent placement, the vessel is widely patent, but replacement of the Amplatz catheter with a conventional right Judkins catheter (JR4) shows how effective the Amplatz has been in straightening out a severe upward bend (shepherd's hook) in the proximal right coronary artery. Although progressive improvements in device profile and trackability have made such deep engagement less necessary, the technique is still of great value in selected cases. Deep seating of the guiding catheter needs to be done with great care and with coaxial advancement of the guiding catheter over a balloon catheter, to avoid injuring the proximal coronary artery.

the target lesion for angioplasty), contrast flow out of such side-holes may increase the total contrast load used during a procedure. A second important function of the guiding catheter is to provide adequate support for advancement of the dilatation catheter across the target stenosis. This support derives from the intrinsic stiffness of the guiding catheter material, a catheter shape that buttresses it against the opposite aortic wall, and/or deep engagement of the guiding catheter into the coronary ostium (Fig. 23.3). Deep engagement was routinely required in the mid-1980s, when poor balloon catheter performance demanded a large measure of support if the balloon was to be forced across a severe stenosis. Unfortunately, deep engagement of the guiding catheter was also a well recognized cause of complication (i.e., ostial dissection). Although deep-guiding catheter engagement is still required on occasion (particularly with smaller, 6F guiding catheters), guiding catheter–induced dissection has become far less frequent with the incorporation of an atraumatic bumper on the tip of most guiding catheters and deep engagement of the guiding catheter only by its coaxial advancement over the balloon catheter. When a deeply engaged guiding catheter is used to push a dilatation balloon or other device across the lesion, the operator cannot forget to then withdraw the guiding catheter back to a more neutral position (just within the vessel ostium) to avoid its migration into an even deeper position as the device is withdrawn. In this sense, the ability to actively use the guiding catheter constitutes one of the important skills required for effective management of the overall angioplasty equipment system.

Guidewires

The original dilatation catheter designed by Gruentzig had a short segment of guidewire (spring coil) attached to its tip to help it follow the vessel lumen and avoid subintimal passage as the catheter was passed to and across the stenosis (Fig. 23.2). Because the shape and orienta-

tion of this leading wire could not be modified once the catheter had been introduced, it provided the operator relatively little control over whether the catheter followed the desired path or was diverted into a side branch proximal to the lesion. In contrast, the movable guidewire system designed by Simpson in the early 1980s contained a standard 0.018-inch Teflon-coated wire that extended and moved freely through the central lumen of the dilatation catheter (8). If this guidewire selected the desired vessel, it could continue to be advanced until it crossed the target lesion. If the guidewire instead selected a more proximal side branch, the balloon catheter could be advanced into the main vessel to a point just before the side branch to hold that place as the wire was withdrawn and reshaped in an effort to choose the desired path beyond. By a series of such iterative advancements of wire and dilatation catheter, many lesions could be crossed with the guidewire and then with the dilatation catheter. The first ''steerable'' guidewires were introduced in 1983, and guidewire technology has continued to improve with the evolution of a wide range of wire sizes (down to 0.009-inch), tip stiffness, shaft support, and lubricious coating.

In contrast to crude early guidewires, modern guidewires are designed to combine tip softness, trackability around curves, radiographic visibility, and precise torque control, which allow the guidewire to be steered past vascular side branches and through tortuous or stenotic segments. With these refinements, crossing a subtotal lesion with the guidewire has become a task that takes seconds rather than minutes to hours, helping to open up the more distal portions of the coronary circulation to a variety of interventional devices. The basic guidewire consists of a solid core (stainless steel or the superelastic alloy known as nitinol) that is ground to a progressive taper in its distal portion. This taper helps retain torque control when the wire is steered around the series of bends located in the guiding catheter and proximal coronary anatomy and allows the stiffer proximal portions of the wire to follow the soft tip into side branches. This core is covered by a spring coil, which is usually Teflon-coated stainless steel on the body of the wire and platinum on the distal 3 to 25 cm (for greater radiographic visibility). A family of plastic-covered guidewires with a hydrophilic coating is available to aid in crossing vessels with extreme tortuosity or total occlusion, but the spring-coil design is still dominant. At the tip of the guidewire, the coil is welded to the tapered core, either directly or through an intermediary shaping ribbon that allows the operator to kink or bend the tip of the wire to a shape that is appropriate for navigating the vessel features it must pass—such as larger-diameter bends for selecting left anterior descending (LAD) versus circumflex artery, smaller kinks or bends for selecting diagonal versus LAD. If greater probing force is required (e.g., for crossing a chronic total occlusion), stiffer tip designs (intermediate or ''standard'' rather than floppy) are available. When more shaft support is needed to help advance a stiff device (e.g., a stent) around a bend, extra support wires are available with a thicker and stiffer inner core. To allow exchange of one device for another, double-length (300-cm) exchange wires are widely available. I use these exchange-length wires as my initial wire in most cases, because they help retain access to the distal vessel as a series of devices (balloons, rotational atherectomy burrs, stents) is employed, without the risk of subintimal passage of the second guidewire as it crosses the partially dilated segment (9). A similar strategy can be followed with shorter (175 cm) guidewires if ''rapid-exchange'' balloon catheters and stent delivery systems are used (see later discussion). These advanced features are now obtainable in guidewires whose diameters range from 0.010 to 0.018 inch (0.25 to 0.5 mm) in 0.002-inch increments. The largest-diameter guidewire that is compatible with the lumen of the particular device (usually 0.014 inch in current practice) is employed, to minimize any potential mismatch between the wire and the tapered tip of the balloon catheter that might impede smooth passage. Although the movable guidewire concept (implemented in the current spectrum of highly sophisticated steerable guidewires) has simplified, shortened, and improved the success rate of coronary angioplasty, it is still important to heed the advice of

Dotter and Judkins (1) that "the guidewire is passed across the atheromatous block more by the application of judgment than of force."

Dilatation Catheters

The dilatation catheters for coronary angioplasty have undergone radical evolution since 1977. As described previously, the original Gruentzig catheters were designed with a short segment of guidewire permanently affixed to the catheter tip to decrease the risk of subintimal passage during advancement down the coronary tree. The shaft of this catheter had two lumens—one for inflation and deflation of the balloon and one for distal pressure measurement and/or contrast injection. This reflected the reliance on monitoring of transstenotic (i.e., aortic root to distal coronary) pressure gradients as a way of assessing lesion severity, given the difficulty in performing adequate contrast injections through small-lumen guiding catheters around the large (4.3F, or 1.3 mm) shafts of early balloon catheters. In contrast, virtually all dilatation catheters since 1982 have used an independently movable and/or steerable guidewire extending the entire length of the dilatation catheter, as described by Simpson and coworkers (Fig. 23.2). The central lumen of such dilatation catheters must have a sufficient caliber to allow free movement of the guidewire, but it is generally no longer used for either pressure measurement or contrast injection. The concept of using transstenotic pressure gradients to evaluate the significance and completeness of correction of coronary stenoses, however, has undergone renewed interest with the advent of solid-state pressure measurement guidewires (see Fractional Flow Reserve in Chapter 18).

An important feature of the dilatation catheter is the diameter of the smallest opening through which the deflated balloon can be passed (its "profile"). Compared with the 0.060-inch (1.5-mm) profile of the original Gruentzig design, current over-the-wire dilatation catheters have profiles of 0.032 in (0.8 mm) or less. Specially designed "fixed-wire" devices, which consist of a balloon mounted directly on a steerable wire core, were developed and used widely in the late 1980s to provide deflated profiles as small as 0.020 in (0.5 mm). Ongoing refinements in balloon technology, however, have allowed competitive performance from over-the-wire systems, thereby restricting the use of such fixed-wire devices to special situations (e.g., dilating side branches through the struts of a stent placed in the parent vessel). To preserve the best balloon profile, a "negative" or "aspiration" preparation (rather than a "positive" preparation, in which the balloon is first aspirated and then inflated with contrast material) is generally preferred, maximizing the probability that the balloon will cross a severe lesion. Although the primary and secondary (i.e., after an initial inflation) balloon profiles are important aspects of performance, the ability of the balloon to bend so as to advance easily through tortuous vascular segments (trackability) and the presence of sufficient shaft stiffness (pushability) to force it through the stenosis are also important. Delivery of the balloon is also aided by the incorporation of a friction-resistant coating (silicone or a hydrophilic coating such as polyethylene oxide) to improve surface lubricity. Other specialized properties of balloon catheters include whether the catheter travels over the wire along its full length or just in its tip (rapid-exchange or monorail style) to allow quick removal and reinsertion of a catheter over a short (i.e., 175-cm) guidewire. So-called "perfusion balloon" catheters also have been designed with either a series of side-holes in the shaft proximal and distal to the balloon segment or a spiral channel within the balloon to allow ongoing antegrade blood flow and thereby mitigate myocardial ischemia during prolonged balloon inflations (Fig. 23.4). Although prolonged inflations do help control elastic recoil and stabilize some dissections (10), they do not improve long-term results (freedom from restenosis). In the era when stents are used for recoil and dissection, the use of perfusion balloons has become rare.

Other than these factors that influence the ability to deliver the balloon catheter across the target lesion, the most important characteristic of the dilatation catheter is its ability to inflate to a precisely defined diameter despite application of pressures that average 10 atm (150 psi).

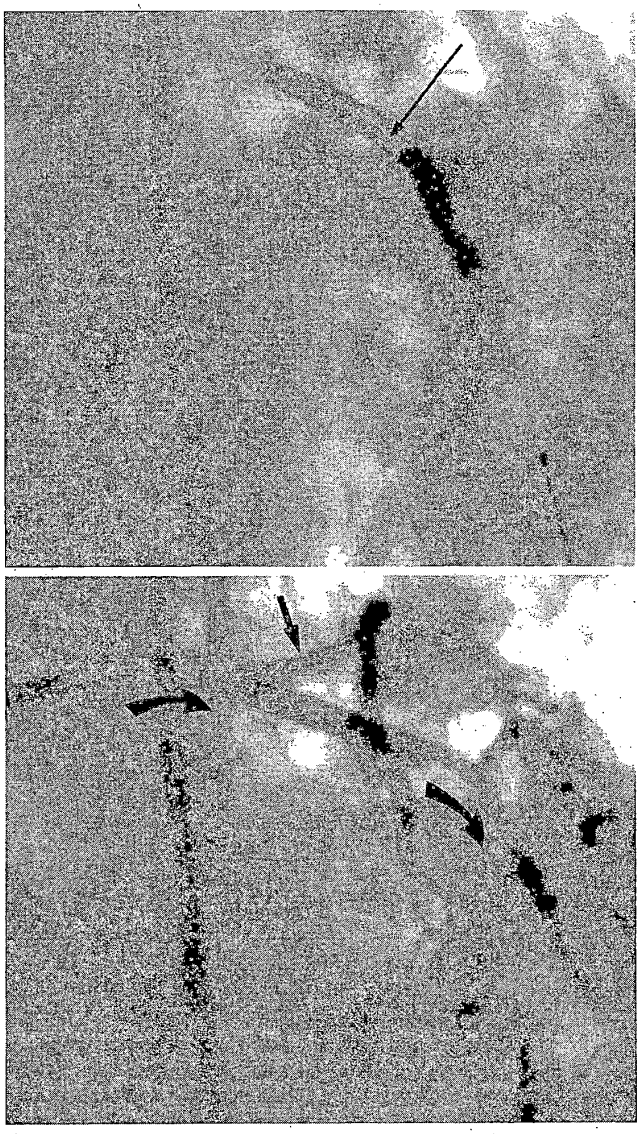

FIG. 23.4. Use of a perfusion balloon catheter. **Top:** The inflated perfusion balloon *(arrow)* is shown in the left anterior descending artery (LAD) and can be recognized by the presence of the non-contrast-filled *(white)* perfusion lumen running through the center of the balloon. **Bottom:** Injection through the guiding catheter *(left curved arrow)* shows direct opacification of the circumflex *(straight arrow)* as well as contrast flow into the distal LAD: this flow enters through proximal side-holes, passes through the perfusion lumen within the balloon, and flows out into the distal vessel *(right curved arrow)*. The 40- to 60-mL/min flow to the distal vessel through the perfusion lumen helps mitigate myocardial ischemia during prolonged balloon inflations, but use of such high-profile devices has become less common since the advent of more effective ways (i.e., stents) to stabilize coronary dissections.

This was not possible with early balloons manufactured from polyvinyl chloride, whose compliance led to balloon oversizing and rupture at pressures as low as 6 atm. More suitable performance can be readily achieved today with balloons manufactured from polyethylene, polyethylene teraphalate (PET), polyolyfin (POC, SciMed), or nylon, with a wall thickness as low as 0.0003 to 0.0005 inch. More compliant balloon materials such as polyolofin tend to reach their rated (nominal) diameter at 6 atm (90 psi) and then grow by up to 20% above their nominal size (e.g., a 3.0-mm balloon growing to 3.5 mm) at 10 atm. Semicompliant balloon materials such as polyethylene or nylon grow by less than 10% over this pressure range, whereas truly noncompliant balloon materials such as PET can retain their defined diameter up to 20 atm (300 psi) to allow dilatation of calcific stenoses or full expansion of coronary stents (Fig. 23.5).

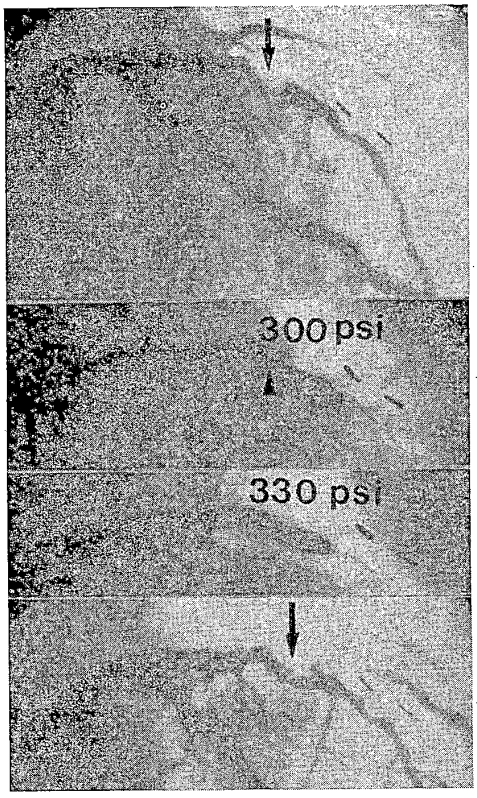

FIG. 23.5. Successful dilatation of a rigid calcific lesion. This rigid lesion (**top,** *arrow*) in the middle left anterior descending coronary artery of a patient who had undergone coronary artery bypass surgery (note surgical clips) resisted dilatation at 300 psi (20 atm) but yielded to an inflation pressure of 330 psi (22 atm) **(middle two photographs)**, with an excellent angiographic result (**bottom**). Such pressures are obtainable only with special high-pressure balloon construction, because most standard angioplasty balloons have rated rupture pressures of only 180 psi (12 atm). In current practice, such lesions would more likely be treated by rotational atherectomy (see Chapter 24).

Balloon compliance characteristics must be kept in mind especially whenever a compliant or semicompliant balloon is inflated to pressures above 6 to 8 atm (90 to 120 psi), to avoid overdistending the adjacent normal vessel. Because the noncompliant balloon materials preclude growth in normal segments upstream and downstream of a rigid lesion, they may be desirable whenever high pressures are needed, and they may also help to treat resistant lesions by concentrating dilating force on the stenosis itself (rather than in balloon growth and dilatation of the adjacent vessel). Regardless of which balloon type is used, staying within the prescribed range of inflation pressures is also important to prevent balloon rupture. This pressure range is specified in terms of the *rated burst pressure* (i.e., an inflation pressure at which the probability of balloon rupture is less than 0.1%). Taking any balloon catheter above its rated burst pressure increases the risk of balloon rupture, with the potential for air embolization (if the balloon was incompletely purged), local dissection, or difficulty in removing the balloon from an incompletely dilated lesion (11). This risk grows the further above rated burst pressure that the balloon is inflated, until it reaches 50% risk of rupture when the average burst pressure is reached. With the availability of effective therapies for calcified or fibrotic lesions (e.g., rotational atherectomy [see Chapter 24]), it is usually unnecessary to take any balloon catheter to pressures more than 1 to 2 atm above the rated burst pressure except in rare circumstances such as stent postdilatation in a calcified or fibrotic lesion that has not been adequately predilated or pretreated with rotational atherectomy.

Dilatation catheters that meet these design specifications are currently available from a variety of manufacturers with inflated diameters of 1.5 to 4.0 mm, in 0.5-mm increments, to match the size of the coronary artery in which the stenosis is located. Larger balloons (i.e., 4.5, 5.0, and 6.0 mm) are occasionally needed for treatment of large right coronary arteries or saphenous vein grafts. These once had to be obtained as special orders of the bulkier balloons used for peripheral vascular intervention, but large-size coronary balloons are now available in most coronary balloon lines. Quarter-sized balloons (e.g., 2.25, 2.75, and 3.25 mm) are also available, but that degree of precision probably exceeds the operator's ability to gauge vessel size, and stocking "quarter-sizes" tends unfavorably to increase the size of a laboratory's balloon inventory. The usual length of the inflatable balloon segment is 20 mm, but most balloons can be obtained with inflated segment lengths

that are shorter, such as a 10- or 15-mm length for a high-pressure dilation completely within a 15-mm stent, or longer, such as 30 or 40 mm for dilation of a diffusely diseased segment (12,13). Most lesions can be dilated effectively with balloon catheters from any of the several manufacturers, but the fact that there are still subtle differences in performance characteristics that can make the difference between success and failure makes it necessary for each interventional laboratory to stock a variety of balloon types. Although competition has brought the average balloon price down substantially (from its high of almost $700 to less than $300), continued pressure on catheterization laboratory budgets has raised the possibility of resterilization and reuse. At least one recent trial of this concept, however, showed an increase in procedure time and device failures for resterilized product (14).

PROCEDURE

In that catheters are introduced under local anesthesia, a coronary angioplasty procedure bears a superficial resemblance to diagnostic cardiac catheterization. However, because angioplasty involves superselective cannulation of diseased coronary arteries with guidewires and balloon catheters, temporary occlusion of antegrade coronary arterial flow, and an attempt to manipulate the offending atherosclerotic lesion by balloon inflation, the procedure is a great deal more complicated and entails roughly 10 times the risk (i.e., 1% vs. 0.1%) associated with diagnostic catheterization (15). These risks should be discussed in detail with the patient and family before the procedure. The potential use of new devices and any alteration in management related thereto (e.g., need for additional antiplatelet therapy including oral agents or intravenous platelet IIb/IIIa integrin receptor blockers) should also be discussed, along with the probability that a repeat intervention may be necessary if restenosis of the dilated segment occurs. Special problems, such as the risk of "no reflow" during vein graft intervention or loss of involved side branches, should be described, if relevant. These small but very real risks of major complications highlight why angioplasty should be attempted only by experienced personnel in a setting where full cardiac surgical and anesthetic support is available (16).

Although patients were once admitted the night before elective angioplasty, current cost-driven protocols delay admission until the morning of the procedure. Details of the patient evaluation, informed consent, and preprocedure laboratory work usually have been completed in a separate outpatient visit or are compressed into a very brief encounter immediately before the procedure. This is particularly true for patients who come to catheter-based intervention at the conclusion of what began as a diagnostic catheterization ("ad hoc angioplasty" or "catheterization with angioplasty standby"). The patient should have been prepared by proscription of oral intake after midnight on the evening before the procedure, and pretreatment with a calcium channel blocker (to prevent vessel spasm at the treatment site) and aspirin 325 mg/day to diminish platelet deposition on the disrupted endothelium (17). Other antiplatelet agents, including low-molecular-weight dextran and dipyridamole 200 mg/day, were once administered in conjunction with angioplasty but have now been abandoned due to lack of demonstrated efficacy, potential allergic or volume-overload side effects with dextran, and the availability of more potent antiplatelet agents. The increasing use of stents and the importance of oral platelet adenosine diphosphate–receptor antagonists (ticlopidine and clopidogrel) (18), as well as the important benefit of intravenous platelet IIb/IIIa integrin receptor blockers in preventing periprocedural infarction and emergency revascularization for vessel closure (19) (Table 23.1), has made either or both classes of agents commonplace additions to aspirin therapy. Controlled trials have yet to show that any type of antiplatelet therapy consistently decreases the incidence of subsequent restenosis. Because aspirin reduces late cardiac mortality in patients with coronary disease, it is generally continued indefinitely after the procedure. In the aspirin-allergic patient, use of these alternative antiplatelet agents (sometimes with the addition of oral sulfinpyrazone) is mandatory.

Angioplasty may be done by the brachial ap-

TABLE 23.1. *Factors associated with abrupt closure or increased mortality with coronary angioplasty*[a]

Factors associated with abrupt vessel closure	Factors associated with increased mortality
Preprocedure	**Clinical factors**
Clinical factors	Female gender
Female gender	Unstable angina
Unstable angina	Age >65 yr
Insulin-dependent diabetes mellitus	Congestive heart failure
Inadequate antiplatelet therapy	Chronic renal failure
Angiographic factors	**Angiographic factors**
Intracoronary thrombus	Left main coronary artery disease
Stenosis >90%	Three-vessel disease
Stenosis length >2 luminal diameters	Left ventricular ejection fraction <0.30
Stenosis at branch point	Myocardial jeopardy score
Stenosis on bend (>45°)	Proximal right coronary artery stenosis
Right coronary artery stenosis	Collaterals originate from dilated vessel
Postprocedure	
Intimal dissection >10 mm	
Residual stenosis >50%	
Transient in-lab closure	
Residual transstenotic gradient 20 mm Hg	

[a] Except for female gender and unstable angina, the risk factors associated with abrupt closure and increased mortality differ. Factors associated with abrupt closure are mostly *lesion-based* and are heavily influenced by a poor postprocedure result (i.e., residual stenosis or dissection). In contrast, factors associated with procedural mortality are mostly patient-based, reflecting poor left ventricular function and the extent of disease (see Ryan TJ, et al. Guidelines for percutaneous transluminal angioplasty; a report of the ACC/AHA task force. J Am Coll Cardiol 1993;22:2033).

proach, although more than 90% of current procedures are done from the femoral approach. Although most catheter-based interventions can be performed safely without right-sided heart catheterization, I still prefer to place a right heart catheter to allow potentially valuable measurement of baseline and intraprocedure filling pressures in patients with abnormal baseline left ventricular function or who are undergoing treatment of major vascular territories. The venous sheath also allows rapid initiation of ventricular pacing, although experience shows that placement of a prophylactic pacemaker is seldom needed in patients undergoing coronary angioplasty (20). After placement of the arterial sheath, intravenous heparin (70 units/kg, or 7,000 to 10,000 units) is administered. Because there is wide patient-to-patient variability in heparin binding and activity, the activated clotting time (ACT) should be measured, and additional heparin should be administered as needed to prolong the ACT to 275 to 300 seconds before any angioplasty devices are introduced and to maintain it at this level throughout the case. Lower levels of ACT (less than 250 seconds) are associated with a marked increase in the incidence of occlusive complications (21), although ACTs in the 275-second range are acceptable when adjunctive IIb/IIIa receptor blockade is used. Higher ACTs (greater than 300 to 350 seconds) tend to increase the risk of bleeding. In setting the target ACT, it is important to understand which machine is being used, because measurement with the Hemochron system (International Technidyne, Edison, NJ) tends to give values 30 to 50 seconds higher than those measured by the rival HemoTech machine (Medtronic Hemodynamics, Minneapolis, MN). Preliminary testing suggests that other direct thrombin inhibitors (e.g., low-molecular-weight heparin, hirudin, bivalirudin [Hirulog], argatroban) may find increasing use during angioplasty, based on more predictable dose-response characteristics than heparin and potentially greater efficacy against clot-bound thrombin (22–25). They may also be useful in patients with the heparin-induced thrombocytopenia or thrombosis syndrome (see Chapter 3).

Baseline angiograms are then obtained of one or both coronary arteries, using either standard diagnostic catheters or the angioplasty guiding catheter. When the guiding catheter is used for baseline angiography, it must be manipulated carefully, because its large diameter and nontapered tip increase the risk of ostial injury. Coronary injections should be repeated after the administration of 200 mg of intracoronary nitroglycerin to demonstrate that spasm is not a significant component of the target stenosis and to minimize the occurrence of coronary spasm during the subsequent angioplasty. My colleagues and I have seen cases where the intended target of a catheter-based intervention resolved with intracoronary nitroglycerin, and an unnecessary intervention was avoided! Baseline angiography also serves to evaluate any changes in angiographic appearance (interval development of total occlusion, thrombus formation) that have occurred since the diagnostic catheterization and to permit the selection of those angiographic views that allow optimal visualization of the stenoses and their surrounding branch vessels.

The appropriate guiding catheter is connected to the pressure manifold (see Chapter 11) by way of an extension tube and a rotating hemostatic valve (Tuohy-Borst valve) and positioned in the coronary ostium. The hemostatic valve contains an adjustable O ring that allows introduction and free movement of the angioplasty balloon while maintaining a sufficient seal around the balloon shaft to permit pressure measurement and contrast injection. The angioplasty guidewire is then steered across the target lesion, guided by puffs of contrast material through the guiding catheter, in a projection that shows the desired path free of foreshortening or overlapping side branches. Some operators advance the guidewire through a dilatation catheter that has been placed into the guiding catheter through the hemostatic valve so that it lies just inside the distal tip of the guiding catheter. Others (myself included) prefer a *bare-wire* technique, in which an exchange-length guidewire is placed into the hemostatic valve through a needle-like guidewire introducer. This permits free movement of the wire during advancement through the guiding catheter and down the involved coronary vessel, while preserving excellent contrast injections absent an obstructing balloon catheter. Once the position of the wire tip in the distal vasculature has been confirmed by contrast angiography, the introducer is removed from the hemostatic valve, and the desired angioplasty balloon or other device is selected.

Experience has shown that the best and safest angioplasty results are obtained with a balloon whose diameter closely approximates that of the presumably nondiseased "reference segment" adjacent to the site being treated (balloon/artery ratio, 0.9:1.1) (26,27). A slightly larger balloon (1.1 to 1.2 the reference lumen) may be used if an intravascular ultrasound study (see Chapter 19) shows that the outer vessel diameter in the reference segment (external elastic membrane diameter) is significantly larger than the reference lumen. On the other hand, a slightly smaller initial balloon may be chosen if it is difficult to estimate the correct reference size in a diffusely diseased or rapidly tapering vessel, or if great difficulty is anticipated in crossing the lesion. The selected balloon is prepared by flushing the central (guidewire) lumen with heparinized saline and filling the balloon inflation lumen with a dilute radiographic contrast material (Renografin-60, or Renografin-76 diluted to half strength). When balloon rupture was more frequent, contrast filling was accomplished by a "positive" preparation, in which the balloon was inflated with contrast material and then aspirated to remove any air. With more robust balloon materials, however, it is now more common to perform only a "negative" preparation, in which a contrast-filled syringe is used to pull air from the balloon lumen and then to let the balloon aspirate a small amount of contrast material when vacuum on the syringe is released. This method of preparing the balloon catheter avoids inflation of the balloon before it is across the target lesion and therefore helps to maintain the lowest possible deflated profile for crossing a severe stenosis. The prepared and flushed balloon catheter is then loaded onto the free end of the guidewire. The tip of this balloon is brought down to the O-ring, which is loosened to permit